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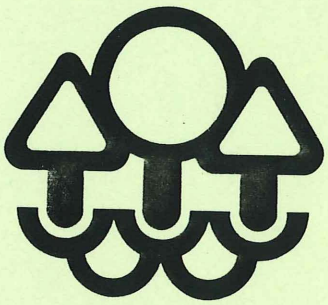
WINONA COUNTY RESOURCE RECOVERY FACILITY

WINONA, MINNESOTA



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MINNESOTA POLLUTION CONTROL AGENCY
OFFICE OF PLANNING AND REVIEW
CLIFFORD T. ANDERSON, DIRECTOR

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DRAFT ENVIRONMENTAL IMPACT STATEMENT

WINONA COUNTY RESOURCE RECOVERY FACILITY
WINONA, MINNESOTA

For further information contact:

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ABSTRACT: Winona County proposes to construct a 150 ton/day solid waste incinerator and electricity generator at one of two sites in the City of Winona. A draft environmental impact statement (DEIS) was prepared on the project by the Minnesota Pollution Control Agency. The DEIS includes an evaluation of health risk associated with air emissions from the facility as well as an evaluation of ash and other solid waste issues related to the facility. Alternatives to the proposal were evaluated regarding comparative financial costs and environmental impacts.

PUBLIC MEETING DATE: JUNE 20, 1988, 7:00 PM

WINONA AREA TECHNICAL INSTITUTE
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I. EXECUTIVE SUMMARY

1.1 DESCRIPTION OF THE PROJECT AND ENVIRONS

The City of Winona is located on an alluvial plain on the west bank of the Mississippi River in southeastern Minnesota approximately 115 miles southeast of the Twin Cities Metropolitan Area. The Mississippi at this point is broad in expanse and bordered by extensive bottomlands, backwaters, floodplain lakes, and side channels. This diverse range of conditions offer a wide range of habitat for fish and wildlife, a diverse population of which are found within the area. Much of the immediate area is included in the Upper Mississippi River Wildlife and Fish Refuge. The edges of the Mississippi Valley in the Winona area are characterised by high bluffs deeply cut by river and stream valleys. The maximum elevation of the upland areas in the Winona vicinity is nearly 600 feet above the Mississippi valley floor. Vegetation in the area is highly varied, depending upon water regime and human impacts. Lock and Dam 5A and Lock and Dam 6 are located upstream and downstream of Winona.

The population of Winona in 1986 was estimated at 24,675. The estimated 1986 population of the City of Goodview which lies immediatley to the west of Winona, was 2711. Winona County's population was estimated to be 46,795 in 1986. Winona is the County seat of Winona County. Its economy is based on diversified light industry, small heavy industry, retail trade, services, and academic institutions.

Two sites are being evaluated as possible locations for the incinerator facility. Both sites are within the City of Winona within areas zoned Heavy Industrial. The Riverview Drive site is located at the northern edge of Winona at a point roughly one-half mile north of the Highway 54 bridge. The Riverbend II site is located in the far southeast corner of Winona adjacent to the wastewater treatment plant.

The Riverview Drive site lies outside the flood levee at Winona and was filled with dredge spoils to an elevation above the 100-year base flood elevation in 1984. Surrounding land use includes Crooked Slough Channel of the Mississippi River and a barge fleeting site which are to the north, a small flood plain forest to the east, a railroad yard and auto roadway on the south, and grain elevators and coal storage piles on the west.

The Riverbend II site is located in the Riverbend II industrial park. The industrial park is within the flood levee. The site is a degraded wetland vegetated by reed canary grass with occasional river bullrush with interspersed clumps of willow and boxelder. Some of the area is wooded. Surrounding land use is varied. To the east of the site is similar wetland terrain and the Winona municipal wastewater treatment facility. To the north is the east end of Winona. Filling will be required at the site. Both the United States Fish & Wildlife Service and the U.S. Environmental Protection Agency have expressed concern over the continued filling of wetland areas within the Riverbend II industrial park.

The roughly 32,000 square foot resource recovery building will be a pre-engineered structural structural steel frame building on a concrete foundation supported on pilings. The building will enclose the tipping floor, process equipment area, ash load-out area, and support facilities.

The proposed facility will consist of two modular incinerator trains with a total capacity of 150 tons per day (TPD) of incoming mixed municipal solid waste. Each incinerator train will consist of a dual chamber incinerator, a

by-pass or dump stack, a heat recovery system, a turbine generator, the pollution control equipment, and the exhaust stack. The incinerators will be mass burn units. An auxiliary fuel system will provide natural gas to maintain temperatures at 1800 F. for at a residence time of at least one second.

Each incinerator will be provided with independent heat recovery equipment. Each heat recovery system will include a steam generator (boiler), economizer, trim, piping, and sootblowers. A single 1800 revolution per minute condensing turbine generator set will be provided to optimize the production of electrical power. The power generated will be sold to Northern States Power Company (NSP), with a portion being used for internal plant needs.

The air quality control system will consist of a dry scrubber and a fabric filter The incinerator will also be equipped with continuous monitoring equipment, which will aid the facility operator in maintaining proper combustion conditions. Two complete air pollution control systems will be employed to treat the flue gas from each incinerator and heat recovery system after it emerges from the economizer. The air pollution control equipment will be provided with one complete system for storage and preparation of lime additive. Each air quality control system will include one spray absorber, one fabric filter unit (bag house), and fly ash and spray absorber solids handling equipment. Particulate control will be achieved within the baghouse. Automatic ash handling system will be provided for bottom ash removal, fabric filter fly ash removal, and scrubber ash removal. A common 100 foot steel stack housing two-2 foot diameter flues will be provided for discharging exhaust from the air pollution control system.

The Rules of the Environmental Review Program (Minnesota Rules, Parts 4410.0200 through 4410.7800) promulgated by the Minnesota Environmental Quality Board, provide that an environmental assessment worksheet (EAW) be prepared on municipal solid waste (MSW) incinerators or resource recovery facilities (RRF's) of the size of the proposed facility. The purposes of the EAW is to determine whether the facility has the potential for significant environmental effects, and accordingly, whether an environmental impact statement (EIS) must be prepared to assess the extent of impacts, possible mitigation measures for those impacts, and alternatives to the project. The environmental review program rules also provide that a discretionary EIS may be prepared when the governmental unit responsible for EIS and the proposer agree that an EIS should be prepared. After discussions in the spring of 1987 yielded such an agreement between the County and the MPCA staff, the MPCA Citizens's Board authorized the preparation of a discretionary EIS.

1.2 ENVIRONMENTAL AND OTHER IMPACTS

During preparation of the Draft EIS, ambient air quality was assessed, contaminant emissions from the facility were projected, and the dispersion and deposition of these emissions were modeled. Results were used to conduct two separrate but complementary assessments of human health and ecological risks associated with air emissions from the facility. The upper bound health risk associated with the frequent consumption of locally-caught fish exceeded the Minnesota Department of Health guideline for additional cancer cases. This health risk was primarily a result of high levels of dioxins and furans predicted in fish tissue by the health risk model.

Issues relating to solid and hazardous waste were reviewed in the EIS. It was determined that the potential existed for very small quantities of household and non-household hazardous waste to be combusted in the proposed facility. The result of such combustion would be an unquantifiable increase in

concentrations of contaminants emitted from the facility or present in the incinerator ash. In the EIS, it was determined that the fly ash and bottom ash from the facility were subject to changing regulatory requirements which would likely result in increased costs of disposal. It was also concluded that industrial waste accepted at the facility would require careful review to prevent the entry of materials containing contaminants or their precursors.

It was determined that the local fire and emergency services would not require additional resources to respond to fire or medical emergencies at the facility. Impacts from the facility on water quality will be negligible, since the proposer has indicated that no discharge of industrial wastewater to the municipal treatment system or area surface water will occur.

Alternatives to the project were reviewed with respect to comparative costs and environmental impacts. Costs associated with the proposed incinerator were projected to be relatively high, although roughly within reach of other alternatives.

II. GOVERNMENTAL APPROVALS REQUIRED

2.1 The following permits and/or approvals are required for the proposed project by various levels of government.

Level of Government		Permit
Federal:		
	Federal Aviation Administration (FAA)	Stack Height Review
	U.S. Army Corps of Engineers	Section 404 Permit (Riverbend II site only)
State:		
	Minnesota Pollution Control Agency (MPCA)	Air Quality Emissions Facility Permit
	MPCA	Solid Waste Facility Permit (will be incorporated into the Air Quality Emissions Facility Permit)
	MPCA	Section 401 Certification of 404 Permit
	Minnesota Department of Health (MDH)	Plumbing Plans Review
	MDH	Public Water Supply Plan Review
	State Fire Marshall	Fire Safety Plan Review
Local:		
	City of Winona	Building Permit
	City of Winona	Plumbing Permit
	City of Winona	Gas Piping Permit
	City of Winona	Sewer Connection Permit
	City of Winona	Water Connection Permit
	City of Winona	Height Review

2.2 The following permits/approvals will require the preparation of a record of decision by the issuer of the permit in accordance with Minn. Rules Part 4410.2900

State:

Minnesota Pollution Control Agency (MPCA)	Air Quality Emissions Facility Permit
MPCA	Section 401 Certification of 404 Permit

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MPCA	Section 401 Certification of 404 Permit
Minnesota Department of Health (MDH)	Plumbing Plans Review
MDH	Public Water Supply Plan Review
State Fire Marshall	Fire Safety Plan Review
Local:	
City of Winona	Building Permit
City of Winona	Plumbing Permit
City of Winona	Gas Piping Permit
City of Winona	Sewer Connection Permit
City of Winona	Water Connection Permit
City of Winona	Height Review

III. PROJECT DESCRIPTION

3.1 DESCRIPTION OF THE GENERAL PROJECT AREA

The City of Winona is located on an alluvial plain on the west bank of the Mississippi River in southeastern Minnesota, on the Minnesota-Wisconsin border. Figure III-A shows the location of the city on a State map. The City is approximately 115 miles southeast of the Twin Cities Metropolitan Area. The City of Goodview, Minnesota is located adjacent to Winona on the west. The nearest large cities are Rochester, Minnesota, 50 miles to the west, and LaCrosse, Wisconsin, 30 miles to the southeast. Figure III-B is a copy of a United States Geological Survey 7.5 minute topographic map of the general area around Winona.

The valley of the Mississippi at Winona varies in width between two and one-half and three miles. In the immediate vicinity of Winona the orientation of the valley is from the west-northwest to east-southeast. The normal pool elevation of the Mississippi at Winona is 646 feet above mean sea level (MSL). In 1936, Lock and Dam 5A and Lock and Dam 6 were constructed upstream and downstream of Winona. Lock and Dam number 5A, operated by the US Army Corps of Engineers, is located about three miles northeast of the center of Winona, and Lock and Dam number 6 is located approximately 10 miles downstream from the City. The Mississippi at this point is broad in expanse and bordered by extensive bottomlands and backwater areas. Much of the immediate area, including several areas adjacent to the City, are part of the Upper Mississippi River Wildlife and Fish Refuge, which is administered by the United States Fish and Wildlife Service.

The elevation of Winona ranges between 667 and 647 feet above MSL. The City is built on generally level alluvial deposits of the Mississippi. The City is protected from flooding by a series of flood control levees. Immediately to the south of the City is Lake Winona, which lies between the City and the bluffs to the south.

The edges of the Mississippi Valley in the Winona area are characterised by high bluffs deeply cut by river and stream valleys. These valleys are highly branched and the terrain becomes very complex. Slopes of several of the valley walls exceed 45 degrees. The maximum elevation of the upland areas in the Winona vicinity is 1250 feet above MSL, or nearly 600 feet above the Mississippi valley floor.

Vegetation in the area is highly varied, depending upon water regime and human impacts. The original vegetation of pre-settlement days in the river bottomlands was a mixture of prairie, marsh meadows, wetlands, open water, and flood plain forests. This vegetation has been substantially impacted by human activity, including the logging of forests and the clearing of land and the filling of wetlands for agriculture and later for residential, commercial, and industrial land use. One result of the construction of the locks and dams was the stabilization of water levels in certain areas. Where the availability of water was increased, sensonally dry prairies were invaded by floodplain forests and marsh vegetation. The dominant vegetation in the flood plains forests consist of silver maple, with some ash, cottonwood, willow, and American elm. Understory vegetation is limited, and is dominated by wood nettles and poison ivy. Today, much of the riverbottoms are encompassed by the Upper Mississippi Wildlife and Fish Refuge and the Trempealeau National Wildlife Refuge. (Burwell and Haas, 1974, Faber, 1988, Hawkins 1988 Fremling, et. al., 1978)

Emergent wetland vegetation in the river bottoms near Winona is

predominantly river bulrush, with lesser numbers of arrowhead, cattail, and burreed. Reed canary grass is the most common wetland grass, occurring in margins between wetland and woodland. Fremling, et. al., (1978), described vegetation in Sam Gordy's slough, opposite Winona at River Mile 724-725.4, as dominated by white water lily and broad-leaf arrowhead, with lesser amounts of narrow-leaf arrowhead, burreed, soft-stem bulrush, and river bulrush. Submergent vegetation included predominantly coontail, with wild celery much less abundant. Other species occurring included curly-leaf pondweed, stargrass, and sago pondweed. The vegetation in the area of Sam Gordy's slough is highly diverse, and has resulted in a correspondingly diversity of wildlife and fish.

The river bottomlands include a wide variety of surface waters and wetlands. The main channel of the Mississippi River is characterized by a relatively strong current with little accretion of sediment and minimal emergent or submergent vegetation. The main channel is subject to disturbance by frequent barge traffic. Side channels and backwater channels, have slower currents and greater abundance of emergent and submergent vegetation. Sloughs and floodplain lakes in certain areas behave like eutrophic lakes and actually tend to stratify during summer months. Some areas are subject to winter kill due to oxygen depletion. There are also broad expanses of open water which are subject to wind aeration. This diverse range of surface water conditions offer a wide range of habitat for fish, and a diverse population of gamefish and rough fish are found within the area. The gamefish community in Sam Gordy's slough, for instance, is dominated by bluegill sunfish and crappies. Substantial populations of catfish, northern pike, bass, and walleye also exist in the area. Rough fish populations are dominated by carp and spotted suckers. Commercial setline fishing for catfish is successful in the area of Sam Gordy's slough. (Fremling, et. al., 1978)

The original upland vegetation was a mixture of bigwoods hardwood forest and oak openings and barrens. About 70% of the forest as it exists now is oak-hickory type forest, with those species comprising the dominant vegetation and other species being occaisional. On level uplands and valley floors, pre-existing plant communities have been cleared and planted with agricultural crops. The interspersion of agriculutral and forest land use has created excellent habitat for some species such as wild turkey and whitetail deer. The steep slopes have been logged in the past but are generally wooded, with oak, and hickory the dominant tree species. Dry prairies are common on some of the bluff areas, where southern exposure and excessive drainage combine to create "goat" prairies which contain little bluestem, side oats gramma, and indian grasses. (Burwell and Haas, 1974, (Golden, 1988)

The population of Winona in 1986 was estimated at 24,675 by the Office of the State Demographer. The estimated population of the City of Goodview in 1986 was 2711. Winona County's population was estimated to be 46,795 in 1986. (MSDO, 1988)

Winona is the County seat of Winona County. Its economy is based on diversified light industry, small heavy industry, retail trade, services, and academic institutions. (Wright, 1987d)

3.2 DESCRIPTIONS OF EXISTING CONDITIONS AT THE ALTERNATIVE SITES

Two sites are being evaluated as possible locations for the incinerator facility. Both sites are within the City of Winona, Winona County. The Riverview Drive site is located at the northern edge of Winona at a point roughly one-half mile north of the Highway 54 bridge. The Riverbend II site is

located in the far southeast corner of Winona adjacent to the wastewater treatment plant. The sites are described in detail below.

3.2.1 Riverview Drive Site

The Riverview Drive site is located in the SW 1/4 of the NW 1/4 of Section 22, Township 107 North, Range 7 West, in the City of Winona. The site is zoned M-2, Heavy Industrial, and the proposed resource recovery facility is a permitted use for such zoning. No conditional use permit or change in zoning is required. (MPCA, 1987)

The county is evaluating either of two 6-acre parcels near Riverview Drive which are currently vacant. Until 1984, the site was a wetland in the flood plain of the Mississippi River, when it was filled with dredge spoils as part of the Winona Waterfront development project. The site itself is presently vegetated by colonizing grasses and forbs. It cannot be considered a significant source of food or habitat for wildlife. The incinerator project will require six acres within the 16-acre site. Surrounding land use includes Crooked Slough Channel of the Mississippi River and a barge fleeting site which are to the north, a small flood plain forest to the east, a railroad yard and auto roadway on the south, and grain elevators and coal storage piles on the west. (MPCA, 1987) Across the Mississippi River from the site, at a distance of approximately 300 yards, is a unit of the Upper Mississippi River Wildlife and Fish Refuge which contains a heron rookery with approximately 300 breeding birds. (Faber, 1988)

A subsurface exploration program was conducted at the site in 1987. Seven soil borings at the site verified the presence of between 11 and 17 feet of dredged fill, consisting primarily of medium grained sand. Beneath the dredge fill are found organic clays and silts varying in thickness between one and nine feet. These deposits, which contain some wood and root material, confirm the previous existence of wetlands on the site. Beneath the organic deposits were very thick deposits of coarse alluvium. At some locations, the organic deposits were also overlain by coarse alluvium. All but two of the borings were completed to 40 feet of depth. One was completed to 62 feet and one to 81 feet. Bedrock was not encountered. The water table was encountered at 14 to 18 feet beneath the surface elevation. (Twin City Testing, 1987a)

The existing grade at the Riverview Drive site is approximately 663 feet above MSL. The finished grade will vary between 661 and 669 feet above MSL. An average of 2 feet of fill, a volume of approximately 13,000 cubic yards, will be placed on the site. The source for most of the fill will be the material excavated for the construction of the facility. All major building and equipment foundations will be supported on driven pilings. (Wright, 1987e)

The site lies outside the flood levee at Winona. The site has been filled with dredge spoils to an elevation above the 100-year base flood elevation. This fill project was evaluated in an EIS titled "Waterford Development" prepared by the City of Winona in 1976. The project was completed in 1984 and was permitted by the U.S. Army Corps of Engineers, the MPCA, and the Minnesota Department of Natural Resources. The site is no longer included in the Flood Plain. (MPCA, 1987)

3.2.2 Riverbend II Industrial Park Site

The Riverbend II site is located in the NE 1/4 of the NW 1/4 of the NW 1/4 of the NE 1/4 of Section 36, Township 107 North, Range 7 West, in the City of Winona. The site is zoned M-2, Heavy Industrial. The proposed facility is

a permitted use and no zoning change or conditional use permit is required. (MPCA, 1987)

The site is located on a six-acre parcel in the Riverbend II industrial park. the industrial park is within the flood levee. It was logged in 1973 and is presently a Type II wetland dominantly vegetated by reed canary grass with occasional river bullrush with interspersed clumps of willow and boxelder. Some of the area is wooded, with the dominant species in the wooded area being pole-sized cottonwood and silver maple, five to nine inches diameter at breast height. (MPCA, 1987, Golden, 1988)

Surrounding land use is varied. To the east of the site is similar wetland terrain, the flood protection levee, and the Winona municipal wastewater treatment facility. To the north, across railroad tracks and the outlet ditch of Lake Winona, is the east end of Winona, where several industries are located. The rest of the Riverbend II Industrial park, presently being developed by the City, is located to the west and south of the site.

The subsurface exploration of the Riverbend II site revealed the area to be underlain by very thick alluvial deposits similar to those found in the Riverview Drive site. Four borings were completed to a depth of roughly 50 feet, and three borings were completed to a depth of roughly 80 feet. Bedrock was not encountered, and the alluvial deposits extended from the bottom of each boring to just 4 to 9 feet below the surface. Organic clay and some peat deposits between 2.5 and 3.5 feet thick were found above the alluvial soil in five of the borings. Fine or mixed alluvial deposits, mostly clays and silts of 3 to 7 feet in thickness, were the topmost soils encountered. The water table was found at depths between 5 and 9 feet. (Twin City Testing, 1987b)

Filling will be required on all or part of the six-acre parcel, and filling permits will be required by the Corps of Engineers under Section 404 of the Clean Water Act. The MPCA will review the project pursuant to Section 401 of the Clean Water Act. The City of Winona will also require a filling permit. Existing elevation is 648 feet above MSL. The finished grade will vary between 654 and 662 feet above MSL. Approximately 70,000 cubic yards of fill we be obtained from an offsite borrow area, the location of which has not been identified at this time. (MPCA, 1987; Wright, 1987e)

In comments on previous Section 404 permits, the United States Fish & Wildlife Service has expressed opposition to the continued filling of wetland areas within the Riverbend II industrial park. In a letter dated October 15, 1987, to Colonel Joseph Briggs, District Engineers, U.S. Army Corps of Engineers, James L. Smith, Assistant Field Office Supervisor recommended against the issuance of a 404 permit for discharge of fill material. (Smith, 1987) That letter included a recommendation that a comprehensive plan be prepared to evaluate impacts of development on wetlands with the greater project area. The request for the preparation of such a plan was also made by the U.S. Environmental Protection Agency in its letter of October 29, 1987. (Bryson, 1987)

The Riverbend II site is located within a flood levee which provides protection against a flood with a recurrence frequency of 100 years. The levee is maintained by the City of Winona and inspected annually by the U.S. Army Corps of Engineers. This area is no longer designated as part of the 100-year flood plain as a result of revisions to the Federal Emergency Management Agency (FEMA) plan and flood plain map made on July 17, 1986. No Flood Plain Zoning amendments or other governmental approvals will be required. (Robert Bollant,

1988)

3.3 STRUCTURES AND ROADWAYS

Regardless of which site is eventually selected for the facility, the resource recovery building will be a pre-engineered structure consisting of a structural steel frame, and a concreted foundation supported on pilings. The building will be covered with insulated metal siding. The building will be constructed with a conventional sloped roof with an eave height of approximately thirty feet and a peak height of roughly forty feet. The approximate dimensions will be 195 feet by 165 feet, with a total area of roughly 32,000 square feet. The building will enclose the tipping floor, process equipment area, ash load-out area, maintenance shop, auxilliary equipment rooms, lavatory facilities, and administrative and plant services facilities. (Wright, 1987e)

If the Riverview Drive facility is used, the existing public roadway (Riverview Drive), located imediately south of the facility will be utilized to serve the facility. The roadway right of way will require some modification to allow access to the facility. The approximate costs of this modification is $5000, which will be born by the proposer and has been included in the project cost. (Wright, 1987e)

Should the Riverbend II site be selected, it will be served by a new public roadway (Frontenac Drive) which is in the planning stages. Frontenac Drive is intended to serve the entire Riverbend II Industrial Park, as well as the MSW incinerator. The cost of the construction of Frontenac Drive is being paid by the City of Winona. An additional roadway of 100 feet in length will be required to provide access to the facility. The cost of this construction will be paid by the proposer, and is estimated to be $20,000. This amount has been included in the project cost estimates provided by Black & Veatch. (Wright, 1987e)

3.4 UTILITY REQUIREMENTS

The utilities required for the facility include water, sanitary, storm sewer, natural gas and electricity. The extension of the water, sanitary, storm sewer, and electric power from existing utilities to the resource recovery facility will be the responsibility of the proposer. The extension of the natural gas utility to the facility will be by the local utility. The utility routing to the facility for each respective site is as follows:

3.4.1 Riverview Drive Site

Water - A potable water line will be installed from the existing city water supply system located approximately 1000 feet from the resource recovery building. An 8 inch water line is required for the facility, however the City has requested that the routing to the site be oversized to allow for future tie-ins. The additional cost for the oversized waterline will be payed by the City (approximately $2,000). The cost for installation of an 8 inch waterline has been included in the project cost estimate (approximately $18,000). (Wright, 1987e)

Sanitary - Sanitary sewer will be installed from the existing City sewer located approximately 1000 feet from the resource recovery building. A 6 inch sanitary sewer is required for the facility, however the City has requested that the routing to the site be oversized to allow for future tie-ins. The additional cost for the oversized sanitary sewer will be payed by the City

(approximately $1500). The cost for installation of a 6 inch sanitary sewer has been included in the project cost estimate (approximately $11,000). (Wright, 1987e)

Electrical - The project cost estimates provided by Black and Veatch include the cost of 1/2 mile of 15 KV, 3 phase line to connect to Northern States Power Company's 12.5 KV [SIC] distribution system. A list of interconnection equipment required by NSP has been obtained. The equipment includes metering and protective relaying equipment. The estimated cost for the connection to NSP is $47,500.00. (Wright, 1987e)

The electrical power required for the facility will not be greater than 750 KW (running horsepower). It is anticipated that the electrical requirement will be somewhat less, however actual numbers will not be available until the completion of design work and all electrical components have been specified. (Wright, 1987c)

Natural Gas - The natural gas supply for the facility will be routed and installed by the local utility. The gas pipeline will be installed to the building wall and will include a metering system. All installation costs will be payed by the local utility as part of their normal practice in providing service to a new customer. (Wright, 1987e)

The natural gas requirements will vary depending on the need to burn gas in the primary and/or secondary chambers of the incinerators. Under worst case conditions, when gas is being supplied to both chambers, the natural gas requirement of the facility will be 15,000 cubic feet per hour. This figure represents the maximum design flow of natural gas to the facility. It is not anticipated that this maximum value will ever be realized. (Wright, 1987c)

Storm Sewer - All surface runoff, including roof drainage, will be directed to an existing 24 inch storm sewer which runs below the proposed project site. The costs for routing to the storm sewer have been included in the project cost estimate (approximately $3,500). (Wright, 1987e)

3.4.2 Riverbend II Park Site

Water - A potable water line will be installed from the existing City water supply system. As part of the City's construction of Frontenac Drive (new access road to the proposed project site), the City will be routing a new 12 inch water line to a point approximately 350 feet from the resource recovery building. The installation cost to route the water line from the City's termination point to the facility has been included in the project cost estimate approximately $6,500). (Wright, 1987e)

Sanitary - A new 10 inch sanitary sewer will also be installed by the City to within approximately 350 feet of the resource recovery building as part of the Frontenac Drive construction. The installation cost to route the sanitary sewer from the City's termination point to the facility has been included in the project cost estimate (approximately $4,000). (Wright, 1987e)

Natural Gas - (same as indicated for Riverview Drive site.)

Electrical - (same as indicated for Riverview Drive site.)

Storm Sewer - A new 36 inch storm sewer will also be installed by the City to within approximately 350 feet of the facility property line as part of the Frontenac Drive construction to be performed by the City. All surface

runoff, including roof drainage will be directed to the City storm sewer. The costs for routing to the storm sewer have been included in the project cost estimate (approximately $8,000). (Wright, 1987e)

3.5 DESCRIPTION OF THE INCINERATOR FACILITY AND HEAT RECOVERY SYSTEM

The proposed facility will consist of two modular incinerator trains with a total capacity of 150 tons per day (TPD). Each incinerator train will consist of a dual chamber incinerator, a by-pass or dump stack, a heat recovery system, a turbine generator, the pollution control equipment, and the exhaust stack. The incinerators will be mass burn units, where incoming waste is not extensively processed to remove recyclable or noncombustible materials. The two incinerator units are intended to operate simultaneously, 24 hours per day, seven days per week, except for periods of scheduled and unscheduled maintenance. The proposers will be applying for a permitted volume of 150 TPD of incoming mixed municipal solid waste. (Wright, 1987a, 1987e)

3.5.1 Tipping Floor

The tipping floor will be enclosed in the incinerator building, with two truck doors to allow access. The dimensions of the tipping floor will be approximately 100' by 130', sufficient for storage of 600 tons of MSW, which is equivalent to four days of incoming waste. The floor will be concrete and sloped to a drain, which will be discharged to the ash quench pit. Tipping floor washdown wastewater will not be discharged to the municipal wastewater treatment system (Wright, 1987a, 1987e)

3.5.2 Incinerator

A front-end loader will move MSW deposited on the tipping floor into the incinerator feed hoppers. The feed hopper will be equipped with a hydraulically operated door. A hydraulic ram will discharge MSW from the feed hopper into the primary incinerator chamber. The feed hopper and primary chamber will be equipped with a refractory-lined fire door. During automatic operation, it will not be possible to open the feed hopper door and the fire door simultaneously. (Black and Veatch, 1987a)

Each primary chamber will be provided with a method to positively feed the MSW from the feeder system through the primary chamber. The primary chambers will also be provided with a mechanism for ejecting the bottom ash from the primary chamber and transporting it to a single bottom-ash collection point. The bottom ash ejection mechanism will also act as a seal for the chamber. An auxiliary fuel (natural gas) burner will be provided for start-up and shut-down purposes. Temperature control in the primary chamber will be accomplished by varying the combustion airflow in the secondary combustion chamber. This will be accomplished by hydraulically actuated dampers with position indication feedback to the facility control system. The hearth and sidewalls of the primary chamber will be lined with high-temperature firebrick. Overhead will be castable refractory. The service rating temperature of refractory will be 2800° F. (Black and Veatch, 1987a, 1987e)

The secondary combustion chamber is provided to assure complete combustion of the flue gas. The auxiliary fuel system will provide natural gas to the secondary chamber, as necessary, to maintain temperatures at 1800° F. for at a residence time of at least one second. Control of combustion temperatures in the secondary chambers will be accomplished by varying combustion airflow through hydraulically actuated dampers provided with similar feedback indication to the facility control system. The secondary chamber will

be provided with wool block lining having a minimum service temperature of 1900° F. and castable refractory with a minimum service temperature of 2800° F. (Black and Veatch, 1987a)

Both chambers will be provided with insulation and an outer metal protective lagging. Both chambers will also be provided with inspection ports and access doors. (Black and Veatch, 1987a)

Combustion air fans will be provided to supply the primary chamber underfire/overfire air and secondary chamber combustion air. All control air dampers will be provided with double acting cylinders, pneumatic drives and provisions for remote indication of damper position. Combustion air will be preheated by being drawn through shrouds which will surround the primary and secondary chambers. This air circulation will accomplish the cooling of the outer skin of the chambers and the preheating of combustion air simultaneously. (Wright, 1987a, 1987e)

3.5.3 By-pass Stack

The incinerators will incorporate a refractory-lined flue gas dump stack immediately following the secondary chamber for emergency use in by-passing the heat recovery system. There will be two dump stacks; one for each of the two incinerators. The dump stacks are provided solely for the protection of the boilers and may be used under emergency circumstances such as loss of facility power and/or boiler feedwater. Dump stack use may also be required if the incinerator experiences an upset and the two other methods of emergency temperature control (air flood and water spray) do not correct the situation. (Wright, 1987a, 1987e; Black and Veatch, 1987a)

The bypass stack will be provided with a control damper which will be held in the closed position by pneumatic or electrical means. In the event of loss of power, positive pressure within the incinerator, or boiler or induced draft fan failure, the damper will be designed to open automatically. Damper position will be indicated remotely on the main control panel. (Black and Veatch, 1987a)

Although detailed start up procedures have not yet been developed, the dump stacks may be open during the initial incinerator warming period while only natural gas is being used as fuel. The stacks may be used for this function as often as once per week per unit. (Wright, 1987e)

3.5.4 Heat Recovery System

Each incinerator will be provided with independent heat recovery equipment. Each heat recovery system will include a steam generator (boiler), economizer, trim, piping, and sootblowers. (Wright, 1987a, 1988c)

Saturated steam is collected in the steam drum and routed to the superheater, where it is raised to a temperature of 625 degrees. Each boiler will generate a maximum of 26,000 pounds per hour of 660 psig/625° F. steam from 274° F. feedwater. This steam will be delivered to the turbine generator. Flue gases will be cooled from 1800° F. measured in the secondary combustion chamber to 400° F. as measured at the economizer outlet. (Wright, 1987e; Korpela, 1987; Black and Veatch, 1987a)

The continuous tube economizer will be installed at the boiler flue gas outlet. Condensate from the turbine generator is routed through the economizer to be heated prior to its return to the boiler. A temperature probe will be

located at the economizer flue gas outlet to measure boiler exit gas temperature. (Black and Veatch, 1987a)

3.5.5 Automatic Soot Blowers

In the boiler and economizer, fly ash will tend to accumulate on the external tube surfaces. If this fly ash is not removed frequently, the efficiency of the heat exchange is reduced. The ash will be cleared from boiler and economizer tubes by automatic air soot blowers which will operate without requiring system shutdown. There will be no boiler ash hopper or economizer ash hopper, since the ash will be carried over to the dry scrubber/fabric filter, where it will be collected as fly ash. Final design of the fly ash collection system was not available at the time the draft EIS was in preparation. (Wright, 1988c)

3.5.6 Turbine Generator

A single 1800 revolution per minute condensing turbine generator set will be provided to optimize the production of electrical power. The power generated will be sold to Northern States Power Company (NSP), with a portion being used for internal plant needs. Steam will enter the turbine throttle at 650 psig/625° F. and exhaust 2.5" Hg. An uncontrolled extraction will be utilized for feedwater heating. The net power output from the 2400 V generator is estimated to be a nominal 2000 KW, based on the net total steam output from the heat recovery systems of both incinerators when burning 150 TPD of MSW. (Wright, 1987a)

3.5.7 Control System

The control devices include all process parameter measuring devices, flow controllers, pressure controllers, and any devices required for firing the auxiliary fuel. The control system will contain a local panel installed near the incinerator/boiler and a remote panel installed in a control room. Control of the equipment will be fully automated with either control panel being able to override the computer. (Wright, 1987a)

3.5.8 Ash Handling System

At the time the draft EIS was being prepared, final design of the ash handling system was not completed, pending the selection of the air quality control system vendor. Information of a general nature made available to the MPCA is as follows:

Automatic ash handling system will be provided for bottom ash removal, fabric filter fly ash removal, and scrubber ash removal. Bottom ash will be removed from the bottom of the primary combustion chamber by a drag chain conveyor which will discharge into the bottom ash quench tank. The quench water required for operation of the quench tank will be provided by various industrial wastewater streams generated in the incinerator facility, as well as non-contact cooling water blowdown water. Fly ash from each of the two spray reactors (dry scrubbers) will accumulate in the spray absorber hoppers. Fly ash which accumulates on the six fabric filters will be removed by an automatic pulse-jet system and fall to accumulate in the fabric filter hoppers. A system of screw conveyors will remove the fly ash from the respective hoppers. The fly ash will be deposited on the bottom ash conveyor at a controlled rate, prior to deposition in an ash storage bin. It is not clear at this time whether provisions will be made for mechanical mixing of the ash in the storage bin. (Wright, 1987a, 1988a, 1988c; Black and Veatch, 1987, 1988)

3.5.9 Stack

A common 100 foot steel stack housing two-2 foot diameter flues will be provided for discharging exhaust from the air pollution control system. The stack will include ladders, platforms, access doors, sampling ports, monitoring ports and drains. Aviation obstruction lighting will be provided, as necessary, in accordance with the latest advisory circulars of the Federal Aviation Administration. (Wright, 1987a)

3.6. Description of Air Pollution Control Equipment

The types and concentrations of contaminants in the flue gases of resource recovery facilities depend on incinerator design, refuse composition, and combustion conditions. Flue gas contaminants are typically categorized as follows:

- Particulate Matter
- Acid Gases
- Metallic Compounds
- Products of incomplete combustion

Particulate matter consists primarily of entrained non-combustible matter in the flue gas, as well as products of incomplete combustion that exist in solid or aerosol form. Particle size distribution is an important consideration in evaluating emission control systems, especially in light of the recent emphasis on the health impacts of fine particulates in the atmosphere. Particle size distribution appears to vary widely among incinerators, and fluctuates over time at a single facility. (Wright, 1987a)

Acid gases are the flue gas constituents which form acids when combined with water or water vapor. They include nitric oxide, nitgrogen dioxide, sulfur dioxide, sulfur trioxide, hydrogen chloride and hydrogen fluoride. Nitric oxide (NO) and nitrogen dioxide (NO_2) formation in a waste-to-energy facility is a function of combustion temperature, flue gas residence time in high temperature regions, combustion air rate, and nitrogen content of the refuse. Sulfur dioxide (SO_2) and sulfur trioxide (SO_3) are formed by the oxidation of sulfur in the refuse, and therefore vary with refuse composition. Hydrogen chloride (HCl) is formed by the reaction of chlorine in the refuse with hydrogen ions from the combustion of hydrocarbons. Since chlorine content of refuse varies considerably, as does the fraction which reacts with hydrogen ions, the concentration of HCl in flue gas has been found to range over a broad spectrum. (Wright, 1987a)

Metallic compounds in flue gas consist primarily of oxides and chlorides of metals contained in the refuse. Most of the metallic compounds are in the vapor phase within the furnace section of an incinerator, since these compounds boil or sublime at temperatures around 1800° F. The metallic compounds tend to condense as the flue gas passes through the boiler and these compounds are adsorbed onto particulate matter. A portion of the metallic compounds may still be present in the vapor phase as the gas exits the heat recovery section, but this proportion is unknown. More than a dozen metals have been detected in incinerator emissions. Lead, zinc, cadmium, chromium, mercury, nickel, arsenic, tin, and others have been measured in concentrations sufficiently large to necessitate their removal by appropriate emission controls. Emissions of metal compounds will depend in large part on the composition of the incinerator feed stock. (Wright, 1987a; Idzorek, 1988)

Products of incomplete combustions include carbon monoxide (CO), high

molecular weight hydrocarbons, and chlorinated aromatic hydrocarbons. The later category includes chlorobenzenes, chlorophenols, dibenzo-dioxins and dibenzo-furans. (Wright, 1987a; Idzorek, 1988)

The air quality control system which will be utilized by the proposed project to control these emissions is a combination of dry scrubbing, fabric filters, incinerator design, and combustion conditions. (Wright 1987a) The incinerator will also be equipped with continuous monitoring equipment, which will aid the facility operator in maintaining proper combustion conditions. (Wright 1987c) Natural gas will be used in the secondary combustion chamber to maintain temperatures of 1,800° F. Flue gas will be exposed for a least one second residence time in the secondary combustion chamber before passing on to the economizer. (Black and Veatch 1987) Two complete air quality control systems will be employed to treat the flue gas from each incinerator and heat recovery system after it emerges from the economizer. The air pollution control equipment will be provided with one complete system for storage and preparation of lime additive. Induced draft fans will draw the flue gas from each incinerator and heat recovery system through the air quality control systems. Each air quality control system will include one spray absorber, one fabric filter unit (bag house), and fly ash and spray absorber solids handling equipment. Each unit will then discharge to a 24 inch diameter stack which is 100 feet high. (Black and Veatch, 1988)

The dry scrubbing/fabric filter process utilizes two discrete major pieces of equipment; a quench reactor and a fabric filter baghouse. The dry scrubbing process will take place within the quench reactor. Particulate control will be achieved within the baghouse. (Wright, 1987a)

Flue gases from the heat recovery system will enter the quench reactor where they will be directed through a fine spray of atomized calcuim-based alkaline slurry droplets. Gaseous emissions will be absorbed by and react with the atomized reagent. The heat of the flue gas will evaporate the water from the slurry. The slurry would also act as an extinguisher if sparks are present in the flue gas stream. The flue gas stream will become humidified, but not saturated in this process. The dry scrubbing process will result in a dry product which will be collected from both the quench reactor and the fabric filter baghouse. (Wright, 1987a)

The flue gases will pass directly from the quench reactor to the baghouse. The baghouse will contain Teflon-coated fiberglass fabric filters, or bags. Particulate will be collected on the bags and create a filter cake. Additional acid gas removal will occur as the flue gas passes through the filter cake. As the pressure drop across the bags increases (due to the filter cake buildup) to a predetermined point, the bags will be automatically cleaned by a pulse jet system. The filter cake (fly ash) will be collected in hoppers at the bottom of the baghouses. (Wright 1987a)

On June 30, 1987, the U.S. Environmental Protection Agency issued a notice of intent to proposed regulations to control emissions from municipal solid waste incinerators. In a first step towards controlling emissions, EPA sent guidance to its regional offices and states, directing the permitting authorities to consider a dry scrubber and a fabric filter or electrostatic percipitator (ESP) as best available control technology for sulfur dioxide and particulate matter. (Wright, 1987a)

3.7 Facility Wastewater

The proposed Winona County Resource Recovery Facility is expected to

generate stormwater runoff, sanitary wastewater, industrial wastewater and non-contact cooling water. (Wright, 1987c)

Stormwater runoff will not be collected from the facility, but rather will be allowed to follow existing drainage pathways, seeping directly to the groundwater. All exposed impermeable surfaces within the site will be kept clean of refuse, and therefore it is not expected that stormwater runoff leaving the facility will be different from any ordinary parking lot. The tipping floor and msw storage areas will be enclosed, so stormwater will not be generated from this area. It is not expected that any type of treatment will be required for the stormwater runoff. (Wright, 1987c)

Sanitary wastewater will be generated at the facility in small quantities, perhaps 35 gallons per hour maximum. This will be discharged directly to the wastewater collection system of the City of Winona. The small volume and typical quality of the sanitary wastewater discharge will not present any problems for the Winona Municipal Treatment Facility. (Wright, 1987c)

Industrial wastewater sources at the facility will include boiler blowdown water, tipping floor wash down wastewater, ash pit quench water, air pollution control equipment slurry wastewater, regenerant waste water from the boiler feedwater treatment system, and housekeeping wash down wastewater. (Wright, 1987c)

The applicant's consulting engineers have indicated that all of the above industrial wastewater streams generated at the facility will be discharged into the ash quench pit. These industrial wastewater streams are not expected to exceed 10 gallons per minute (gpm) during normal operation. No industrial wastewater will be discharged to the Winona municipal wastewater collection system. (Wright, 1987c)

Cooling needs at the facility will be provided by a cooling tower system which will be supplied by water from the Winona municipal water distribution system. This water will be further treated at the incinerator facility. Blowdown water from the cooling tower will be discharged to the ash quench pit as much as possible. The remainder will be discharged into the Winona municipal wastewater system. A permit will be required from the City for this discharge, and that permit will stipulate allowable levels of contaminants. Cooling water will not be discharged to the Mississippi River, so there will not be a thermal pollution. (Wright, 1987c, 1988a)

3.8 Facility Operations

3.8.1 General Operations

Traffic signals will be normally used to control incoming commercial truck traffic. During periods of high traffic volumes, vehicles will be required to maintain their position along the two lane access road. A traffic signal located near the entrance gate will turn green when the scale is clear of the previous vehicle weighed. Weighing time for vehicles will normally involve less than three minutes and unloading time will normally last less than five minutes. The vehicles will thus be in the facility for less than ten minutes on the average. There will be no parking or storage of refuse trucks on site. Following visual inspection by the scale master, commercial trucks carrying processable waste (usable for incinerator) will be routed to one of the two truck doors in the tipping floor area. Trucks hauling non-combustible material such as demolition waste, etc., will be directed to the bypass

landfill. A further discussion of handling proceedures for unacceptable waste follows this section. Private vehicles will be directed to the transfer/recycle station located adjacent to the resource recovery buidling. (Wright, 1987a, 1987b)

The transfer/recycle station will consist of 3 roll-off containers. One of the containers will be designated for non-combustible/recyclable materials. The roll-off containers containing combustible materials will be weighed and trucked to the tipping floor when thay are full. Non-combustible/recyclable materials will be weighed then trucked to recycling centers or landfilled as required. The transfer/recycle station containers will be emptied at a minimum of once per day. (Wright 1987b)

Waste at the facility will be burned on a first-in, first-out basis. During the facility's scheduled "downtime", all waste delivered in excess of tipping floor storage capacity will be diverted to the ash/bypass landfill. The facility is designed for a maximum of four days storage, but it is not anticipated that waste will actually be stored for more than two days. In the event of scheduled or unscheduled "downtime" of more than four days, waste will be transferred to the landfill. (Wright, 1987b)

3.8.2 Operating Procedures for Handling of Unacceptable Waste

The policy of Winona County is that unacceptable wastes will not be processed at the waste to energy facility. Unacceptable wastes are defined below. This definition is taken from the Solid Waste Management Agreements between Winona and Houston and Wabasha Counties which governs procedures to deliver waste to the Winona waste to energy facility. (Mossing 1988)

Unacceptable wastes include animal waste used as fertilizer, "white goods", demolition debris, earthen fill, construction operations [SIC], sewage, silt, and any material defined as "Hazardous Wastes" under Minnesota Statutes, Section 116.06 as may be amended from time to time, or the Occupational Safety and Health Act of 1970 (Public Law 91-596), Section 10.6 or any other similar law. (Mossing 1988)

Solid waste haulers, through each Counties' licensing, permitting, designation contracting and designation ordinance authorities will be prohibited from delivering unacceptable wastes or any load which contains unacceptable wastes to the waste to energy facility. If quantities of unacceptable wastes are mixed with the solid waste haulers' load, that load may be rejected by the waste to energy facility plant operator. If the load is rejected before being tipped on the facility floor, the waste hauler will be charged the waste to energy facility's tipping fee, then redirected to the bypass landfill and charged a bypass landfill disposal charge. If the load is rejected after being tipped on the facility floor, the waste hauler will be charged the waste to energy facility tipping fee, plus costs to Winona County for collection, transport and disposal at the bypass landfill. Hazardous wastes will be transported to a regional household hazardous waste collection facility (further discussed below). Tires, white goods, or other large metal materials will be turned over to recyclers or to scrap dealers. All other unacceptable wastes which are not hazardous or recyclable will be transported to the bypass landfill for disposal. (Mossing 1988)

During the early weeks of operation of the waste to energy facility, the plant operator or designated individual will be available on the tipping floor in an effort to determine, through discussions with the waste hauler if the load contains unacceptable wastes. Upon weigh-in and delivery to the tipping

floor, each hauler will be encouraged to ask questions if there is any uncertainty concerning unacceptable wastes on the load. (Mossing 1988)

3.9 FACILITY CONSTRUCTION AND ASSOCIATED IMPACTS

Initial construction activities will be somewhat different at each of the respective sites. A brief discussion of these initial activities is provided below:

3.9.1 Riverview Drive Site

The Riverview Drive site consists of 6 acres of a 17 acre industrial development constructed by the Winona Port Authority. Existing grade elevation is above the 100 year flood level. Existing soil conditions will require the use of piling to support the major equipment and building foundations. The first construction activities will involve securing the site (fencing) and the routing of necessary utilities to the property limits for construction use. Excavation will then commence to the depth required for the deepest foundations (basement level). All excavated material will be stockpiled for later use as structural backfill and/or site fill material. Once the piling is driven, the concrete foundation will be formed and poured. The foundations are then backfilled to existing grade. Construction activities will be similar at either site from this point on. (Wright 1987e)

3.9.2 Riverbend II Industrial Park Site

The Riverbend Park site is part of an existing designated wetlands area. Access to the proposed site will be provided by the construction of a new public roadway (Frontenac Drive) to be completed by the City. An Army Corps of Engineers permit must be obtained prior to the commencement of any construction activity on the incinerator site. The first construction activities will involve clearing the site, (trees, vegetation, unusable soil (muck) etc). Site security and the routing of utilities to the site for construction purposes will also be initiated at this time. Sufficient fill material will be placed in order to allow for construction vehicle access and maneuvering. Excavation will then commence to the depth required for the deepest foundations. A dewatering system will be installed, as necessary, until the piling is driven and the foundations are formed, poured and backfilled. Construction activities will then continue with further site filling, foundation work, etc., similar to the Riverview Drive site. (Wright 1987e)

At each respective site, intensive sediment control measures will to be taken by way of hay bales, mulching and other acceptable methods to ensure surface water runoff will not carry excess sediment from the site. (Wright 1987e)

Detailed specifications and drawings will be given to the contractor to delineate the soil erosion/sediment control measures. The measures will be further enforced by a resident engineer at the contruction site who will be overseeing the construction activities. (Wright 1987e)

REFERENCE LIST WINONA COUNTY RESOURCE RECOVERY FACILITY PROJECT DESCRIPTION

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and #4800 87-263, April 14, 1987, Rochester, Minnesota.

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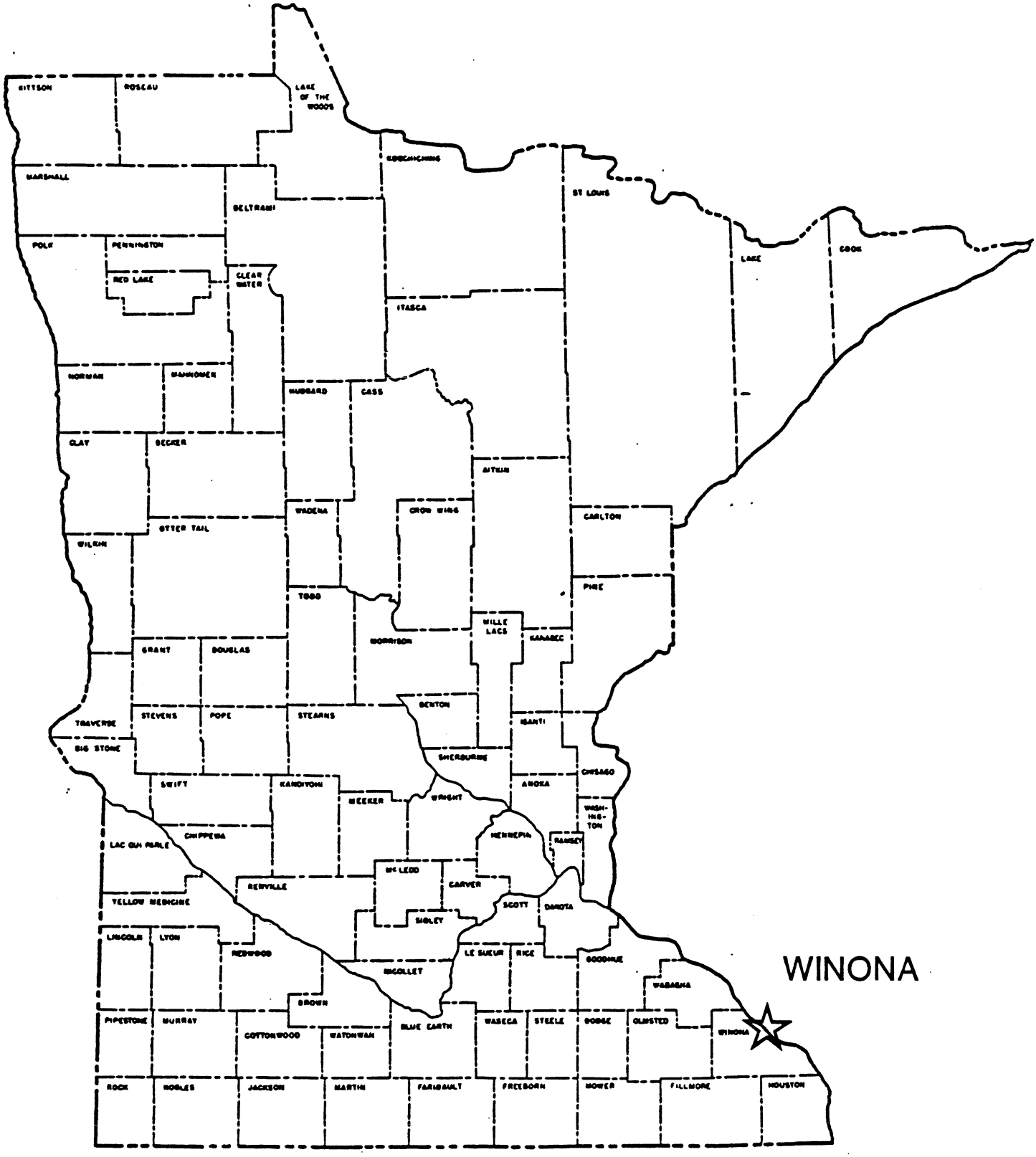

FIGURE IIIA PROJECT LOCATION

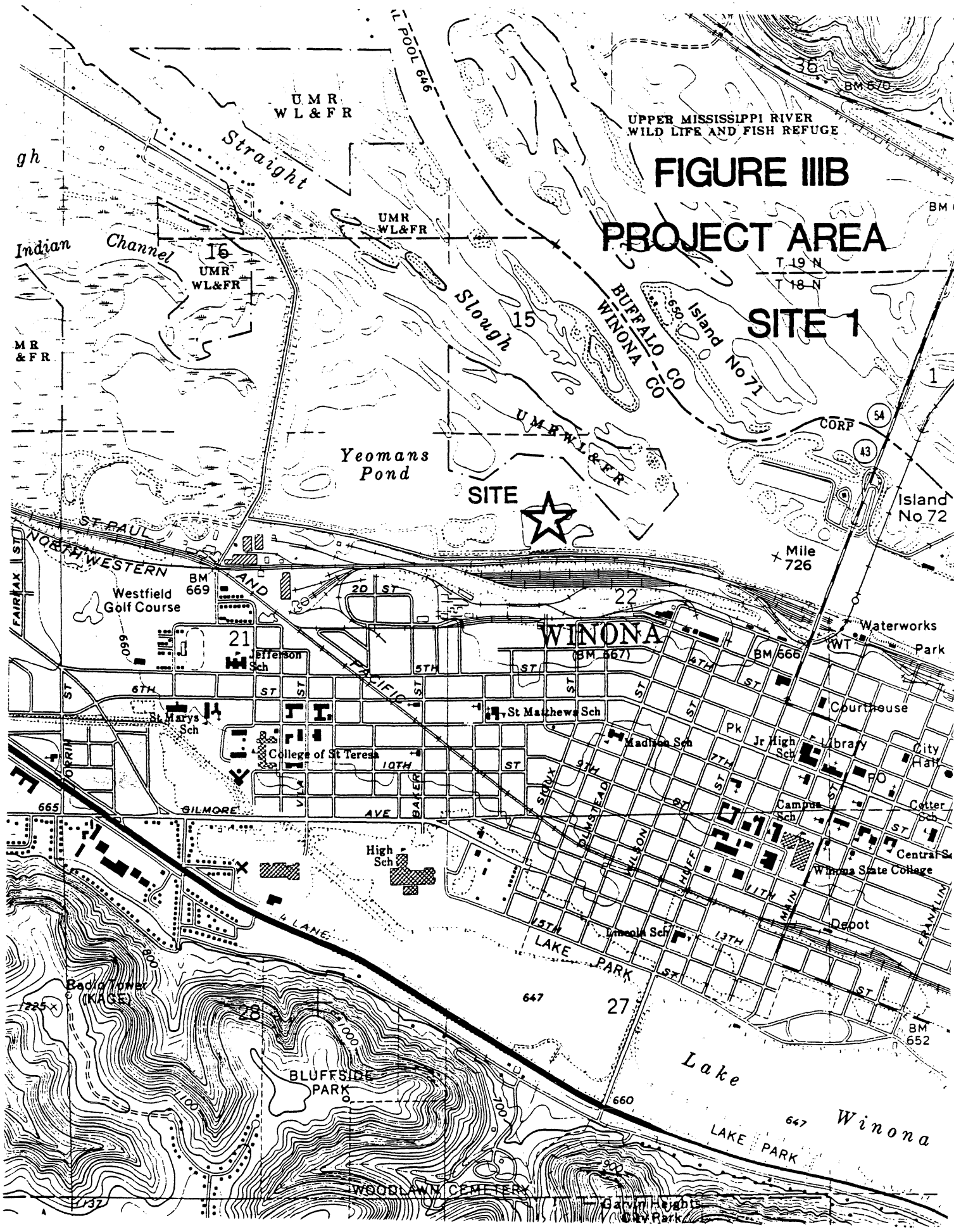
FIGURE IIIB
PROJECT AREA
SITE 1
SITE
UPPER MISSISSIPPI RIVER
WILD LIFE AND FISH REFUGE
UMR
WL&FR
Straight
Indian
Channel
Slough
Yeomans
Pond
Island
No 71
Island
No 72
BUFFALO CO
WINONA CO
T 19 N
T 18 N
CORP
ST PAUL
NORTHWESTERN
AND
Mile
726
WINONA
(BM 667)
Westfield
Golf Course
BM
669
BM 666
Waterworks
Park
Courthouse
Library
City
Hall
PO
Jefferson
Sch
St Marys
Sch
College of St Teresa
St Matthews Sch
Madison Sch
Jr High
Sch
Campus
Sch
Cotter
Sch
Central
Sch
Winona State College
High
Sch
Lincoln Sch
Depot
Pk
WT
4 LANE
LAKE
PARK
BLUFFSIDE
PARK
WOODLAWN CEMETERY
Garvin Heights
City Park
Lake
Winona
Badger Mine
(RAGE)
5TH
6TH
7TH
9TH
10TH
11TH
13TH
20 ST
ST
AVE
GILMORE
PACIFIC
MAIN
FRANKLIN
SIOUX
DUMSTEAD
WILSON
HUFF
BAKER
ORRIN
FAIRFAX
22
21
28
27
15
16
1
36
BM 670
BM
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647
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665
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1100
1225
1137

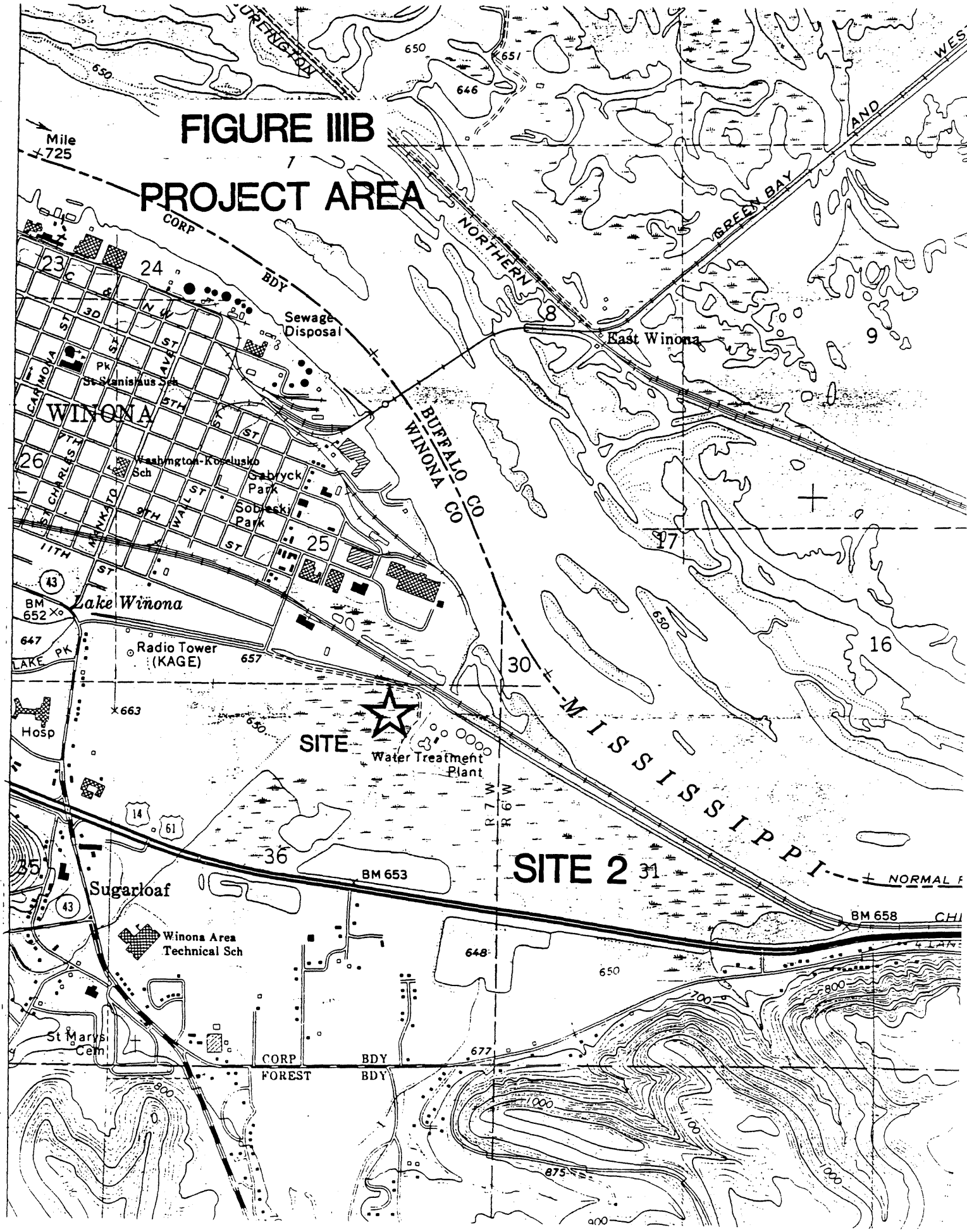
FIGURE IIIB
PROJECT AREA
Mile 725
CORP BDY
NORTHERN
GREEN BAY AND WEST
Sewage Disposal
East Winona
WINONA
St Stanislaus Sch
Washington-Kosciusko Sch
Sabryck Park
Sobieski Park
Lake Winona
BM 652
Radio Tower (KAGE)
Hosp
SITE
Water Treatment Plant
BUFFALO CO
WINONA CO
MISSISSIPPI
NORMAL
R 7 W
R 6 W
SITE 2
BM 653
BM 658
Sugarloaf
Winona Area Technical Sch
St Marys Cem
CORP BDY
FOREST BDY
650
651
646
647
648
657
663
677
700
800
875
900
1000
1100

IV. ENVIRONMENTAL IMPACTS

4.1 AIR QUALITY IMPACTS

4.1.1 AIR EMISSIONS FROM THE FACILITY TIPPING FLOOR

The incinerator facility itself will be fully enclosed, and vehicles will drive into the facility to deposit refuse on the tipping floor. Combustion air for the incinerator will be drawn from the building, so that the tipping floor will experience a slight net negative pressure. This will assure that odors are drawn into the incinerator where they will be destroyed. Since incoming waste will tend to be somewhate moist, it is not expected that particulate emissions will be significant

The facility will not have capacity to store accumulated municipal solid waste (MSW) for more than 4 days. It is expected that the longest period of storage will not exceed 2 days. During scheduled downtime, waste delivered in excess of the tipping floor capacity will be diverted to the ash/bypass landfill. (Wright, 1987a, 1987b, 1987c)

4.1.2 EXISTING AIR QUALITY

Information on existing air quality in Winona County can provide background against which incremental impacts of the proposed facility can be compared. Date pertaining to ambient air quality in the city of Winona is not abundant, and the available information is limited to routinely sampled chemical parameters. The Minnesota Pollution Control Agency (MPCA) provided ICF-Clement with several summaries of chemical air concentrations measured in Winona between 1973 and 1985. Monitored parameters included cadmium, lead, nickel, sulfur dioxide, and particulate matter (MPCA 1987a, b, c). Particulate matter was most recently tested in 1985, sulfur oxides in 1979, and the remainder in 1975, therefore current air levels of these chemicals may vary somewhat from the older measurements. Table IV-1 summarizes air concentration data for the selected chemicals of concern. Concentrations of compounds for which Winona monitoring results exist are presented as monthly averages, the highest and lowest values defining the range. The annual average concentration, when available, is also shown.

For those chemicals with no available Winona concentration data ICF-Clement relied on USEPA reports (USEPA 1984, 1985, 1986) to provide data which might shed some light on approximate backround air concentrations likely to be measured in Winona. It cannot be presumed, however, that these concentrations would be representative of Winona. For each compound a range of background air concentrations is given in Table IV-1. USEPA data availability varied among chemicals. Arsenic, beryllium, chromium, nitrogen oxides, and polycyclic aromatic hydrocarbons (PAHs, expressed as benzo(a)pyrene) had been measured in various Minnesota cities including in all cases Duluth and Minneapolis, and in some cases St. Paul, St. Louis Park, Virginia, and assorted sites in Sherburne and Wright Counties. For these five chemical categories concentrations are reported as quarterly averages. No Minnesota test data was available for aldehydes, antimony, chlorobenzenes, chlorophenols, hydrogen chloride, mercury, polychlorinated biphenyls (PCBs), and PCDDs/PCDFs. To avoid presenting artificially high background values in Table V-1, source-related monitoring data (e.g. data collected in the vicinity of a known pollutant source) were excluded from consideration, as were any data with a USEPA quality rating greater than 2.00, i.e., of low reliability. (ICF, 1987b)

TABLE 4.1A

BACKROUND AMBIENT AIR CONCENTRATIONS FOR THE WINONA COUNTY RESOURCE RECOVERY PROJECT

Backround Ambient Air Concentrations Data		
Compound	Concentration Range (ug/m3)	Site(s) of Measurement
Aldehydes	NA	
Antimony and compounds	NA	
Arsenic and compounds	0.0029 - 0.0041	3 stations in MN (a)
Beryllium and compounds	0.13 - 0.24	4 stations in MN (b)
Cadmium and compounds	0.001 - 0.002	1 station in Winona (c)
Chlorobenzenes	NA	
Chlorophenols	NA	
Chromium and compounds	0.52 - 0.64	2 stations in MN (e)
Hydrogen Chloride	NA	
Lead and compounds	0.11 - 0.58	1 station in Winona (f)
Mercury and compounds	NA	
Nitrogen Oxides	8 - 39	9 stations in MN (g)
Nickel and compounds	0.001 - 0.017	1 station in Winona (h)
Particulate Matter	20 - 100	1 station in Winona (i)
Polychlorinated Biphenyls (PCBs) PCDDs/PCDFs	NA	
Polycyclic Aromatic Hydrocarbons (Expressed as benzo(a)pyrene)	0.13 - 0.93	4 stations in MN (b)
Sulfur Oxides (expressed as SO2)	2.62 - 23.6	1 station in Winona (j,k)

(a) USEPA (1984). Quarterly averages from Duluth, St. Paul and Minneapolis.
(b) USEPA (1985). Quarterly averages from Duluth, Minneapolis, St. Louis Park, Virginia (city).
(c) MPCA Data Report (1987a). 1975 monthly averages. Annual arithmetic mean = 0.001 ug/m3.
(d) Only 1st and 2nd highest 1- and 8-hour average values available (USEPA 1986b).
(e) USEPA (1985). Quarterly averages from Duluth and Minneapolis.
(f) MPCA Data Report (1987a). 1975 monthly averages. Annual arithmetic mean = 0.33 ug/m3.
(g) USEPA (1986b). 1985 annual mean of 24-hour averages for stations in Minneapolis, Roseville, Sherburne Co. (5) and Wright Co. (2).
(h) MPCA Data Report (1987a). 1975 monthly averages. Annual arithmetic mean = 0.006 ug/m3.
(i) MPCA Data Report (1987c). 1985 monthly averages. Annual arithmetic mean = 54 ug/m3.
(j) MPCA Data Report (1987b). 1979 monthly averages. Annual arithmetic mean = 7.87 ug/m3. (see note "l")
(k) Values for SO2 converted from parts per million to ug/m3 assuming ambient temperature and pressure. NA = Not Available.

Source: ICF,(1987b)

4.1.3 RELEVENT CLIMATOLOGY

Climatological conditions in Winona will greatly affect both the

dispersion of chemical emissions and the deposition of particles emitted from the proposed incinerator. Unfortunately, no comprehensive climatological data for the Winona area are available. The nearest city for which records exist is La Crosse, Wisconsin, located approximately twenty-five miles east-southeast from Winona. The data from La Crosse is considered to provide an adequate basis for an estimate of Winona conditions because of their relative proximity and similarity in position within the valley of the Mississippi River. However, because of the influence by the river valley, predominant wind directions at La Crosse and Winona may not be strictly comparable. At La Crosse the Mississippi River turns from a northwest-southeast to a north-south alignment. At Winona the valley runs roughly northwest-southeast. Therefore, when prevailing winds are from the north, local wind patterns at Winona are likely to be similar to those at La Crosse, but when winds are southerly, local wind directions at Winona patterns are expected shift in direction by approximately minus 45 degrees (counter-clockwise) from wind directions observed in La Crosse. The relevence of climatological data to dispersion of particles emitted from the Winona facility will be discussed in further detail in Section 4.1.5.4. The wind in La Crosse is primarily from the south with a mean speed of approximately nine miles per hour. Based upon averages for the 1951-1980 period (NOAA 1987), monthly mean temperatures in La Crosse range from 14° F. in January to 73° F. in July. The minimum daily temperature is not below 32° F. for approximately 215 days per year. The mean annual precipitation (converting snow to equivalent liquid amounts) is 30.25 inches, falling primarily (approximately 80%) in the Spring and Summer months. (ICF, 1987b)

4.1.4 ESTIMATED EMISSIONS

In modeling health risk from a facility which emits air pollutants, normal procedure requires an estimate of the type and quantities of air emissions expected from the facility. The air emissions become input to the dispersion and deposition modeling to estimate the deposition rates of the contaminants on the water or ground surface. These rates become the basis for calculating the environmental fate of the contaminants and the exposures to humans and wildlife which are critical to the Health Risk and Ecological Risk Assessments.

Air emission factors for the Winona County Resource Recovery Facility were initially developed by ICF/Clement Associates (a discussion of this development is included in the technical work paper prepared by ICF/Clement). These factors were based on a review of available test data for mass burn municipal solid waste incinerators using various types of air pollution control (APC) equipment. The emission facts reported by ICF/Clement were the arithmetic mean of the included test results. (Idzorek, 1988)

MPCA staff review of the ICF/Clement emission factors suggested that an alternate analysis of the data may produce more appropriate values. The review and development of the alternate emission factors was completed as follows (Idzorek, 1988):

1. Test results included in the ICF/Clement database were reviewed and checked for accuracy against other data sources.

2. The data was separated into two groups for further analysis; one group consisted of all data and the second group consisted of only those facilities where cooling of the stack gases would be effected (these systems generally use a dry scrubber). This grouping was selected because review of available data suggests that the gas cooling effects enhanced removal of metals and organics due in part

to condensation. Most of the APC systems which effect gas cooling also are adding lime for acid gas control; this provides additional particulate surface area for adsorption of condensing gases and metals.

3. The data was then analyzed by using the t-statistic to establish the 95 percent confidence interval in which the true mean is expected to reside. The upper limits of the intervals for each data group were then compared and the lower of the two limits was selected as the recommended emission factor. The reason for this selection was that the limited data for dry scrubbers and the scatter of the data in some cases resulted in the upper limit for the entire data set being the lower value. This selection is justified because of the better control provided by the dry scrubber.

The results of this analysis are presented in the following table. Mass emission rates were calculated based on the estimated stack gas flowrate of 563.2 cubic meters per minute at standard conditions. The revised emission rates are compared to those originally developed by ICF in Table 4.1C. (Idzorek, 1988)

TABLE 4.1B

ESTIMATED EMISSION RATES FOR PROPOSED WINONA CO. INCINERATOR HEALTH RISK ASSESSMENT

	AVG. CONC. (ng/nm^3)		RECOMMENDED EMISSION VALUES[3]	
Pollutant	DS Data[1]	All Data[2]	ng/nm^3	g/SEC
Arsenic	135	14,600	472	4.43×10^{-6}
Beryllium	0.88	40.4	5.64	0.05×10^{-6}
Cadmium	66,900	131,000	232,000	2.18×10^{-3}
Chromium	134,000	145,000	302,000	2.83×10^{-3}
Lead[4]	429,000	3,610,000	1,450,000	13.61×10^{-3}
Mercury5	197,000	466,000	977,000	9.17×10^{-3}
Nickel	123,000	102,000	223,000	2.09×10^{-3}
Antimony	257	83,100	NO RECOMMENDATION	
PCBs	8.31	833	12.88	0.12×10^{-6}
CL Benzenes	160	1,400	1,120	10.51×10^{-6}
CL Phenols	299	3,330	940	8.82×10^{-6}
PAHs	-	675,000	2,190,000	20.53×10^{-3}
Aldehydes	-	2,270,000	10,800,000	101.38×10^{-3}
PCDD/PCDF[6]	2.63	3.31	5.21	48.90×10^{-9}

Notes:

1. Average concentration calculated from Data for systems using dry scrubbers.
2. Average concentration calculated from all available data, (most systems use only ESP for control.)
3. Recommended emission value, based on the lesser of the upper 95% condifence limit for Ds or all Data.
4. PERMIT LIMIT FOR HERC is 0.007 LB/TON (860,300 NG/NM^3).
5. PERMIT LIMIT FOR HERC is 0.002 LB/TON (245,800 NG/NM^3).
6. PERMIT LIMIT FOR HERC is 3 NG/NM^3.

TABLE 4.1C

COMPARISON OF MPCA'S AND CLEMENT'S
CHEMICAL SPECIFIC EMISSION ESTIMATES

CHEMICAL	MPCA EMISSION RATE (g/sec)	CLEMENT EMISSION RATE (g/sec)	MPCA/CLEMENT RATIO
Aldehydes (treated as formaldehyde)	1.01E-01	1.70E-02	6.0
Antimony and compounds	6.24E-04	6.24E-04	1
Arsenic and compounds	4.43E-06	1.10E-04	0.04
Beryllium and compounds	5.00E-08	3.03E-07	0.2
Cadmium and compounds	2.18E-03	9.83E-04	2.2
Chlorobenzenes (treated as 1,4-dichlorobenzene)	1.05E-05	1.06E-05	1.0
Chlorophenols (treated as 2,4,6-trichlorophenol)	8.82E-06	2.50E-05	0.4
Chromium (VI) and compounds	2.83E-03	1.09E-03	2.6
Lead and compounds	1.36E-02	2.71E-02	0.5
Mercury (inorganic) and compounds	9.17E-03	3.50E-03	2.6
Nickel and compounds	2.09E-03	7.68E-04	2.7
Polychlorinated Biphenyls	1.20E-07	6.26E-06	0.02
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	4.89E-08	2.48E-08	2.0
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	2.05E-02	5.07E-03	4.0

4.1.5 AIR MODELING OF CONTAMINANT DISPERSION

4.1.5.1 Methodology Utilized in Air Modeling

Because the dispersion and distribution of air emissions of a facility cannot be measured until such a facility is actually operating, it is necessary to model the dispersion of those emissions in an effort to predict their fate in the environment. The results of dispersion modeling are used in the assessment of health risk associated with emissions from the facility. Modeling is normally performed with the aid of computer programs and standard procedures developed by the U.S. Environmental Protection Agency which have been accepted by atmospheric scientists.

The air modeling effort in this draft EIS was performed by Winona County's engineering consultant, Black and Veatch, Engineers-Architects. (Black and Veatch, 1987a) The air modeling methodology utilized by Black and Veatch was reviewed and accepted by the staff of the Minnesota Pollution

Control Agency (MPCA), subsequent to the submission of additional data, (Black and Veatch, 1987b), and later reviewed by ICF-Clement Associates for the MPCA.

Determination of the appropriate air model needed to calculate dispersion from a source is critically dependent on the nature of the local terrain. Because of the complex terrain surrounding the proposed Winona facility, the USEPA guideline models (UNAMAP6) are somewhat inadequate for modeling in the area. For this reason Black and Veatch was requested by the MPCA to utilize a variety of air models to characterize the dispersion of emissions from the proposed Winona facility. The following discussion describes and evaluates the air models and methodology utilized. (ICF, 1987a)

The USEPA Guideline on Air Quality Models (1986a) recommends simple terrain air models for calculating ground-level concentrations in areas with elevations less than the stack height. These air models are subdivided according to the level of sophistication used in estimating air quality. The first level of models is the screening models, which provide conservative estimates of air quality. The second level consists of the refined models, which require detailed input data and provide a more sophisticated treatment of atmospheric processes. Refined models executed with the appropriate input data will provide a more realistic estimation of air quality impacts than a screening model executed for the same source. (ICF,1987a)

The Industrial Source Complex (ISC) model, a refined air quality model for use in simple terrain, has received USEPA (1986a) preferred status for modeling the dispersion of emissions from complicated sources such as the proposed Winona facility. Depending upon the particular application, one of two ISC model programs can be used. The ISCST (ICS, short term) model calculates concentrations for time periods ranging from one hour to one year. The ISCLT (ICS, long term) program calculates only seasonal (quarterly) or annual average concentrations. The County's consultant used both ISC programs to model the potential air quality impacts from the Winona facility. Both ISC programs were executed using the rural dispersion coefficients appropriate for the Winona area. The ISC programs contain a number of model options for characterizing the modeled source. The model options selected by Black and Veatch were generally appropriate and consistent with USEPA (1977, 1981) guidelines. Table IV-3 lists several of the model options used by Black and Veatch in the ISC programs. (Pratt, 1988)

The input data in terms of stack parameters are important in determining the modeling results. The stack parameters used by Black and Veatch are appropriate with one possible caveat. The incinerator design calls for two flues in a common 100 foot chimney, each flue with a two foot diameter and an air flow of 15,914 cubic feet per minute. Black and Veatch modeled only one flue with the interpretation that the impacts should be doubled to account for the two flues. This approach is acceptable in the sense that the predicted impacts will be greater than if both flues were modeled. The more precise approach of modeling both plumes will result in greater dispersion due to bouyancy effects. Hence, the Black and Veatch approach will results in higher predicted air quality impacts and higher health risks than the more precise approach of modeling both plumes. The magnitude of the difference between the two approaches cannot be determined precisely, without remodeling, but it is estimated to be on the order of 10 percent. (Pratt, 1988)

Both ISC programs require extensive meteorological input data. To calculate annual average concentrations the ISCST requires one year of sequential hourly meteorological data. The ISCLT program requires a statistical summary of the meteorological conditions. According to the USEPA

(1986a) the variability of model estimates due to meteorological data input is adequately captured if a five year record of meteorological input is used. Black and Veatch used ISCST to calculate annual average concentrations from meteorological data compiled for La Crosse, Wisconsin for the five year period from 1975 to 1979. The ISCLT annual average concentrations were calculated using data collected at the same site but for the five year period from 1967 to 1971. Both ISC programs were executed using the same input data and model options except with respect to the meteorological data. However, The ISCST program located the maximum annual ground-level concentration southeast of the proposed facility, whereas the ISCLT program located the annual maximum north of the facility with a value about 25 percent less than that predicted by ISCST. The difference between the model outputs could be due to the meteorological data or to slight differences in the individual program algorithms. The variations between the calculated ISCLT and ISCST annual averages could be minimized by using input meteorological data collected for the same period in both models. Comparisons between model outputs would then have more validity. (ICF,1987a)

For modeling purposes, terrain with elevations greater than the height of the stack top are defined as complex terrain. USEPA guidelines (1986a) currently include no recommended refined air models for calculating ground-level concentrations in complex terrain. Instead, there are two recommended levels of screening models. The initial screening technique uses the Valley dispersion model to determine the 24-hour average concentrations in complex terrain. Inputs to the Valley model include source data and receptor elevations. Worst-case meteorological conditions in terms of pollutant dispersion are assumed for atmospheric stability, wind speed and hours of occurrence. The conservative and simple nature of this screening technique is designed to provide an evaluation of the maximum expected ground-level concentrations in complex terrain. (ICF,1987a)

The second level of screening techniques requires hourly meteorological data inputs and represents a more refined methodology than the first level screening. For application in the rural areas surrounding the proposed Winona facility the Complex 1 model used by Black and Veatch is consistent with USEPA guidelines (1986a). The Complex 1 model was developed by incorporating the plume impaction algorithm of the Valley model into a refined, simple terrain model called MPTER. The resulting Complex 1 model has been shown to overestimate the peak ground-level concentrations when compared to actual measurements of buoyant plume impaction in complex terrain. The USEPA recommends using on-site meteorological data when applying a second level screening model in complex terrain. Complex 1 can be used to calculate concentrations for averaging periods ranging from one hour to one year. Black and Veatch used the Complex 1 model to make predictions using the off-site LaCrosse meteorological data. Like the ISC model, the Complex 1 model contains several model options to describe the source characteristics. The model options selected by Black and Veatch were generally consistent with USEPA guidelines. As shown in Table IV-3 the options used in the Complex 1 model match the options used in the ISC modeling described earlier. (ICF, 1987b)

Black and Veatch used both the simple and complex terrain models to calculate concentrations at receptors with elevations above the stack height. This procedure yields results that are consistent with USEPA guidelines (1986a). For risk assessment purposes the following numbers are considered most appropriate. For acute effects the most appropriate normalized values (up to 24 hour averages) are given in Tables 1 and 2 of the Supplemental Information for the Technical Work Paper on Air Emissions Modeling (Black and Veatch, 1987b). For subchronic effects or for deposition during the growing

season, the seasonal normalized values given in Tables 3 and 4 of the Supplemental Information are best, and for long term effects the annual normalized values in Tables 3 and 4 of the Supplemental Information are most appropriate. (Pratt,1988)

The Valley model described earlier can be used to calculate annual averages in complex terrain. However, using the Valley model to predict annual averages is not part of the screening procedures recommended in the USEPA guidelines (1986a). Black and Veatch used the Valley model to calculate annual averages for complex terrain. The input meteorological data was the same record used in the ISCLT model. Model options and input source parameters were consistent with all previous models except for the stack downwash option which is not available in the Valley model. (ICF,1987a)

In conclusion, the modeling protocol selected by Black and Veatch was generally consistent with USEPA guidelines (1977, 1981, 1986a) for air quality modeling except for the application of the Valley model. Black and Veatch used a combination of simple and complex terrain models preferred (USEPA 1986a) for modeling a site like the proposed Winona facility. Thus, the modeling protocol utilized by Black and Veatch represents recommended air modeling, lacking site-specific meteorological data. For the sake of consistency, however, use of the same meteorological record for all model runs would have been preferable. (ICF, 1987b)

It should be noted that the model results are highly dependent on the representativeness of the meteorological data, discussed in a later section. Given the limitations of the National Weather Service meteorological database and the extensive meteorological input requirements of the air models, Black and Veatch used the most appropriate records available. The receptor grid network was of sufficient density to locate the maximum ground-level concentrations for each model. In the following sections alternative modeling and the problems with the input meteorological data are discussed in greater detail. Ground-level concentrations calculated by the air models and used in the health risk assessment are presented in Section 4.1.5.5. (ICF, 1987)

4.1.5.2 Deposition Modeling

In order to assess the potential human health risk from the indirect exposure pathways, such as ingestion of soil and homegrown produce, deposition modeling was conducted. The purpose of the deposition modeling was to estimate the rate at which fly ash particles emitted from the proposed Winona facility would be deposited to the surface. (ICF, 1987a)

The ISC models used to estimate annual average ground-level air concentrations can also be used to estimate dry deposition rates (but not wet deposition rates). However, the deposition methodology used in the ISC models was considered inappropriate for modeling the deposition of the small fly ash particles typical in resource recovery plant emissions. The ISCLT model calculates deposition rates based on gravitational settling velocities and reflection coefficients. Reflection coefficients refer to the fraction of particles that are reflected back into the atmosphere after impaction on the surface. The remainder of the particles are deposited and retained at the surface. The guidance provided by the ISC User's Manual (USEPA 1986b) for determining the reflection coefficient is based on a field study where the results may be applicable only to the specific surface and large particle size diameters used in the study. (ICF, 1987a)

In addition, the gravitational settling velocity describes the particle

velocity due only to gravitational effects. For particles with a diameter of 10 um or more, the gravitational settling velocity is sufficient to predict the deposition rate. For smaller particles the effects of molecular and turbulent diffusion as well as gravitational settling must be considered. Based on emissions tests from the Munich North resource recovery facility in West Germany and others, over 70% of the particles emitted from the proposed Winona facility will likely have diameters less than 10 um. Thus gravitational settling velocity alone will not accurately predict the deposition rate of fly ash particles emitted from the facility. Furthermore, the suggested ISC reflection coefficients (USEPA 1986b) may be inappropriate for these small particle sizes. (ICF, 1987a)

Due to the limitations of the ISC deposition modeling option, deposition rates for this risk assessment were estimated from a model developed by Sehmel and Hodgson (1979). The model is based on wind tunnel studies of particle deposition velocities for a range of particle diameters and deposition surface types. The predicted deposition velocities account for the effects of molecular and turbulent diffusion as well as gravitational settling. (ICF, 1987a)

The Sehmel and Hodgson (1979) methodology has been proposed by the California Air Resources Board (CARB 1987) as a refined method to estimate deposition rates for resource recovery facility emissions. The Sehmel and Hodgson (1979) model can be used to estimate deposition velocities as a function of particle size, surface roughness length, and friction velocity, and when multiplied by the predicted annual average air concentrations, the deposition velocities provide the basis for estimating deposition rates (i.e., particulate flux in g/m2-yr). (ICF, 1987a)

The particle size distribution for the North Munich facility was used to derive the deposition velocities for the proposed Winona facility emissions. The particles were separated into discrete ranges based on particle diameter and the mass fraction of particles in each diameter range was determined (ICF, 1987). The deposition velocity for each size range was determined from the Sehmel and Hodgson (1979) model, and then the overall deposition velocity for particle emissions was based on a mass-weighted average of the deposition velocities for each size range. Since the deposition velocity is a function of surface roughness characteristics, deposition velocities were calculated separately for the Mississippi river assuming a surface roughness length of 0.1 cm and a friction velocity of 20 cm/sec, for seasonal crop land areas assuming a surface roughness length of 10 cm and a friction velocity of 40 cm/sec and for the combined overland Winona area assuming a surface roughness length of 54.4 cm and a friction velocity of 100 cm/s. Surface roughness values were chosen from values or methods suggested by the National Oceanic and Atmospheric Administration (NOAA 1983). The estimated weighted-average deposition velocity for the Mississippi river was calculated to be 0.7 cm/sec, for seasonal crop land areas 1.0 cm/sec and for the combined overland Winona area 2.4 cm/s. These calculations are summarized in Appendix B of the Health Risk Assessment for the Proposed Resource Recovery Facility in Winona, Minnesota. (ICF, 1987)

In calculating deposition rates, it was assumed that plume depletion would not have an effect on the amount deposited at any receptor. In effect, the annual average air concentrations were modeled assuming no deposition of particles from the plume, and these concentrations were then used to calculate deposition rates without any adjustment for prior particle loss due to deposition. This assumption results in overestimates of both the deposition rates and the suspended ambient air concentrations with increasing distance from the proposed facility stack. (ICF, 1987a)

It should be recognized that the deposition modeling based on Sehmel and Hodgson (1979) considered only dry deposition processes. The occasional periods of wet deposition due to precipitation scavenging were not included due to the variability and complexity of this process. Reliable information on the scavenging and wet deposition of toxic substances from stack emissions is not available. (Pratt, 1988) Also, no attempt was made to model possible physical or chemical transformations of particle-bound pollutants that could occur in the atmosphere. Modeling only the dry deposition processes according to the method outlined above will likely overestimate dry deposition, but may slightly underestimate total deposition due to the omission of wet deposition. (ICF, 1987a)

4.1.5.3 Alternative Air Models

The protocol used by Black and Veatch to model dispersion of emissions has been described and evaluated in section 4.1.5.1. The protocol was in most cases consistent with the USEPA (1986a) guidelines for air quality modeling and appropriate for the Winona facility. However, the use of the following alternative model may have improved the overall model protocol. (ICF, 1897a)

The proposed Winona facility is located in an area where stack emissions may impact on the surrounding elevated terrain. During the periods in which plume impaction occurs, ground-level concentrations can increase significantly. Complex terrain models are designed to identify the ground-level concentrations associated with plume impactions. Black and Veatch used a USEPA-approved (1986a) complex terrain model in evaluating the concentrations in complex terrain. However, the model required an extensive meteorological input in order to calculate concentrations in the hills surrounding the proposed Winona facility. As discussed in the next section, this meteorological data is not truly representative of the wind flow patterns expected in the Winona area. Since the accuracy of the dispersion models is critically dependent on the input meteorological data, the location and possibly the magnitude of the maximum ground-level concentration for the complex terrain may be inaccurate. (ICF, 1987)

Atmospheric scientists generally agree that if the 24-hour air quality standard is not exceeded due to emissions from a single continuous source, then standards with longer averaging time periods are also not likely to be exceeded. The Valley model is recommended in USEPA (1986a) guidelines for predicting conservative values of the maximum ground-level concentration for 24-hour averages. The Valley model was designed to model the dispersion of buoyant plumes in rural complex terrain over a 24-hour period where the emissions were from a single facility with an elevated point source. Further, the Valley model was developed to model plume dispersion in a stable atmosphere with low wind speeds, considered representative of the worst dispersion conditions in complex terrain. The USEPA recommended technique involves using the Valley model with a set of worst-case meteorological conditions as determined by meteorologists with considerable experience in this area. For plume dispersion in a rural area they recommend a wind speed of 2.5 m/s and a Pasquill-Gifford stability class of F. The wind is set to blow in one direction for a period of six hours. From these conditions a worst-case concentration for receptors along the direction of the wind can be calculated. Use of this method removes the question of the representativeness of input meteorological data and provides an upper bound estimate on the peak 24-hour concentrations in elevated terrain. Black and Veatch could have used this methodology to calculate values that could be compared to the concentrations calculated using more refined models with questionable meteorological input. (ICF, 1987a)

Other models are available for calculating concentrations in rural complex terrain, but as of yet they have not received the USEPA (1986a) recommended status of the Complex 1 and Valley models. Therefore, application of these models would require appropriate evaluation before they could be substituted for the recommended models. It should also be noted that most of the other complex terrain models require an extensive meteorological input representative of the site in order to produce accurate concentration estimates. Since that type of data is not presently available for the proposed Winona site, application of complex terrain models should not be considered until such data are available. However, such site-specific meteorological data utilized in the USEPA (1986a) recommended complex terrain models described earlier would adequately describe dispersion of the emissions from the proposed Winona facility in the surrounding complex terrain. (ICF, 1987a)

4.1.5.4 Representativeness and Effect of Input Meteorological Data

The accuracy of the air dispersion models used by Black and Veatch depends in large part on how well the input meteorological data characterizes the conditions at the proposed Winona facility. The USEPA Guideline on Air Quality Models (1986a) provides specific guidance on the selection of meteorological data to be used in air models. They state that "meteorological data used as input to a dispersion model should be selected on the basis of spatial and climatological (temporal) as well as the ability of the individual parameters selected to characterize the transport and dispersion conditions in the area of concern." The representativeness of the meteorological data is dependent on the proximity of the meteorological monitoring site to area under consideration. Black and Veatch used meteorological data collected in La Crosse, WI to model dispersion in the Winona area. La Crosse is located approximately 50 miles south of Winona. Like Winona, La Crosse is situated along the Mississippi river in a valley with bluffs rising 450 to 500 feet above the valley floor. Since both sites are located in a large river valley with only 50 miles of separation between them, the La Crosse data satisfy the requirements of representativeness with respect to the weather conditions expected in Winona. (ICF, 1987a)

Wind flow in both La Crosse and Winona is channeled by the steep hills rising up from the river valley. The La Crosse Municipal Airport is the official monitoring station for meteorological records from the La Crosse area. The airport is located on the north end of French Island. South of the airport the river valley runs in a southerly direction. However, north of the airport, the river valley bends toward the northwest. The effect of the river valley is to channel wind flows, resulting in northwesterly prevailing winds from January through April and southerly winds for the remainder of the year. The river valley in Winona does not bend as sharply to the south as the valley in La Crosse, but it does have the same strong northwesterly bearing north of the city. Thus, the winds in Winona should show the same channeling from the northwest, but will not show the same southerly component. For Winona the predominate wind directions will be northwesterly and southeasterly. For a large portion of the year (from May through December) the predominant wind direction in Winona will be from the southeast, differing from observations in La Crosse by approximately 45 degrees. (ICF, 1987b)

Using the La Crosse meteorological data to describe dispersion in Winona will make the model location of the maximum ground-level concentration suspect. Because both valleys have a strong northwest bearing above the city, the locations of the ground-level concentrations calculated to the south of the facility could be accurate. However, the valley in La Crosse is about twice as wide as the Winona valley. This could result in higher wind speeds in the

Winona valley due to lateral compression of the air flow. The higher wind speeds would likely result in a lower ground-level concentration. Calculated maximum concentrations due north of the proposed facility would probably shift to the northeast if site-specific meteorological data were used. (ICF,1987a)

The La Crosse data should be representative of atmospheric stabilities in Winona since both are subject to the same synoptic weather conditions and both have the same type of terrain features. The rise of a buoyant plume is dependent on the atmospheric stability and the wind speed. The degree of plume rise affects the value of the maximum ground-level concentration and how far from the stack it will occur. In complex terrain the atmospheric stability will be a major determinant in whether the plume centerline will impact on the valley walls or rise up over. Because of the similarity in stability conditions between La Crosse and Winona, plume rise calculations based on the La Crosse data would likely be similar if done using site-specific meteorological data. This means that the values for the ground-level concentrations in complex terrain should be accurate, especially to the south of the facility since the frequency of wind in that direction should be similar for both La Crosse and Winona. (ICF,1987a)

Site-specific meteorological data is the preferred input to air models when the quality and length of the data is adequate. However, USEPA (1986a) guidelines require at least one year of data and possibly longer in order to identify the worst-case meteorological conditions. Since the major difference between the La Crosse and Winona meteorological conditions involve the wind direction and possibly the speed, it should be safe to use the La Crosse data in the Winona model provided the location of the maximum ground-level concentration is not needed. In the absence of site-specific meteorological data, the most conservative method of utilizing the model output would be to pick the maximum concentration within each radial distance and apply that value to all receptors along that ring regardless of the individual azimuth. (ICF, 1987a)

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4.2. HEALTH RISK AND ENVIRONMENTAL RISK ASSESSMENT

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4.2.1 INTRODUCTION

4.2.1.1 SUMMARY

This Health Risk Assessment of the Draft EIS presents an environmental health risk assessment for Winona County's proposed resource recovery facility in Winona, Minnesota. This draft EIS is based on findings of the technical work paper prepared by ICF/Clement Associates, Inc. The proposed facility comprises one segment of the integrated solid waste management program developed by Winona County. The proposed resource recovery facility would be able to convert 150 TPD of wastes into energy, reduce the volume of raw refuse by 80%, and would be equipped with state-of-the-art air pollution control devices--scrubbers for the removal of acid gases and fabric filter baghouses for the removal of particulate matter. Like all modern technology, the proposed Winona County resource recovery facility has the potential to have an impact on the environment. Due to this, a discretionary EIS is being prepared by the MPCA pursuant to Minnesota Rules Part 4410.2000, Subpart 3B. MPCA retained the services of ICF-Clement Associates, Inc. in association with E.A. Hickok and Associates to prepare a Technical Work Paper evaluating the potential for adverse human health effects and ecological impacts from emissions released from the proposed facility. This risk assessment was conducted using conservative assumptions according to the general guidelines outlined by USEPA (1986a,b). The draft EIS presents a conservative analysis intended to indicate the upper bounds of the potential for health risks to be present. When applying the broad principles of risk assessment to site-specific risk assessments for resource recovery facilities, areas important for evaluation include the specifications of the facility and its associated control devices, the methods of calculating exposure including fate and transport models and local population considerations, and the methods of dealing with toxicity. An evaluation of 16 risk assessments conducted for resource recovery facilities indicates there are not only significant differences in the types of facilities evaluated, but also in the risk assessment methods used. Most of the differences are in the areas of exposure assessment, especially with regard to the number of pathways selected and the approaches used to evaluate these pathways.

4.2.1.2 THE NATURE OF HUMAN HEALTH RISK ASSESSMENT

Many everyday activities undertaken by people involve risk in some form. Associated with everyday life are activities which involve risk which could potentially result in accidents, illness, or death. Individuals often make decisions about the acceptability of risk before undertaking the activity. The basic purpose of a risk assessment is to provide sufficient information about the possible environmental health effects of a product or process so that a decision as to its acceptability may be made.

In a human health risk assessment of exposures to potentially toxic chemicals, the probability of an occurrence and the resulting outcome of that occurrence are important in evaluating the potential impact on human health. The probability of harm associated with exposure to a contaminant is related to the strength and duration of exposure as well as to the potentially harmful properties of the chemical, which most often must be predicted for humans from observations in test studies using laboratory animals. In practical situations, the critical factor in determining risk is not the toxicity of a substance but the risk associated with exposure to the substance. For example, an extremely toxic chemical poses little or no risk if there is little or no exposure to the chemical. Risk is the probability that harm will be caused under specified exposure conditions. Even extremely toxic

substances may not result in risk provided one can control the degree of exposure using proper engineering controls.

Risk assessments generally contain the following four steps (NAS 1983) which may be specifically applied to the proposed Winona facility as:

1. Hazard identification: Determination of the nature and amount of toxic chemicals potentially released from the Winona plant and selection of those chemicals of greatest potential concern to human health. Identification of health effects potentially associated with these chemicals.

2. Dose-response quantification: Determination of relationships between exposure to the toxic substances emitted and the expected human health effects.

3. Exposure assessment: Mathematical modeling to determine the amounts of toxic substances emitted to which individuals are exposed. Exposures may occur through several pathways, including inhalation, accidental ingestion of outdoor soil, and ingestion of locally-produced dairy products.

4. Risk characterization: Calculation of the probability that an adverse health effect may result from exposure to the selected toxic chemicals emitted.

This risk assessment was conducted using conservative assumptions according to the general guidelines outlined by USEPA (1986a,b).

4.2.1.3 CHEMICALS OF CONCERN

Another important difference between risk assessments is that different sets of chemicals are often evaluated for different facilities. When attention was first focused on the potential health risks from resource recovery plants, only criteria pollutants and 2,3,7,8-TCDD were evaluated. Since that time, research results have shown that numerous other materials including metals, organic chemicals, and acid gases may be emitted from combustion facilities. If the results of two or more risk assessments for various facilities are being compared to each other, it is important to keep in mind that this comparison is valid only if the same chemicals are being evaluated. A matrix showing the chemicals evaluated in the risk assessments which were analyzed for this report in shown in Table 4.2.1A.

TABLE 4.2.1A

MATRIX OF CHEMCIALS INVESTIGATED IN RISK ASSESSMENTS

CHEMICAL	FACILITY*													
	W	BA	BL[a]	GP	B1	PN	O	SP	B2[b]	H	N	YC	ST[c]	SA[d]
CRITERIA POLLUTANTS[e]														
Nitrogen Oxides	--	--	--	--	--	--	--	x	--	--	--	--	--	x
Sulfur Oxides	--	--	--	--	--	--	--	x	--	--	--	--	--	--
Carbon Monoxide	--	--	--	--	--	--	--	x	--	--	--	--	--	x
Lead & Compounds	x	x	x	--	--	--	--	x	x	--	--	x	x	x
Total Particulate Matter	--	--	--	--	--	--	--	x	--	--	--	--	--	x
METALS														
Antimony	--	--	x	x	--	--	--	--	x	--	--	--	x	x
Arsenic	x	x	x	x	--	--	--	x	x	x	--	x	x	x
Beryllium	x	x	x	--	--	--	--	x	x	x	--	x	x	x
Cadmium	x	x	x	x	--	--	--	x	x	x	--	x	x	x
Chromium	x	x	x	x	--	--	--	x	x	x	--	x	x	x
Cobalt	--	--	x	x	--	--	--	--	x	--	--	--	--	--
Copper	--	x	x	x	--	--	--	--	--	x	--	--	x	x
Manganese	--	x	x	x	--	--	--	--	--	x	--	x	x	x
Mercury	x	x	--	--	--	--	--	x	--	x	--	x	x	x
Molybdenum	--	--	x	x	--	--	--	--	--	--	--	--	--	x
Nickel	x	x	x	x	--	--	--	x	x	x	--	--	x	x
Selenium	--	x	--	x	--	--	--	--	x	--	--	x	x	x
Tin	--	x	x	x	--	--	--	--	--	--	--	--	x	x
Vanadium	--	x	x	x	--	--	--	--	--	x	--	x	x	x
Zinc	--	x	x	x	--	--	--	--	x	x	--	x	x	x
ORGANICS														
Aldehydes	x	--	--	--	--	--	--	--	x	x	x	--	--	x
Chlorobenzenes	--	--	--	--	--	--	--	--	--	--	--	--	--	x
Chlorophenols	--	--	--	--	--	--	--	--	--	--	--	--	--	x
PAHs	x	x	--	x	--	--	--	x	x	x	--	x	x	x
PCBs	--	x	--	x	--	--	--	--	x	x	--	x	x	x
PCDDs/PCDFs	x	x	x	x	x	x	x	x	x	x	x	x	x	x
ACID GASES														
Hydrogen chloride	x	--	--	x	--	--	--	x	--	--	x	--	--	--

[a] Also Thorium.
[b] Also Scandium.
[c] Also Hydrogen Fluoride.
[d] Also Alumium, Barium, Calcium, Iron, Magnesium, Silicon, Sodium, Titanium, Acetic Acid, Chlorine, Formic acid, Hydrogen fluoride, nonmethane HC, and Vinyl chloride.
[e] These chemicals are often addressed separately in PSD Permit Applications.

TABLE 4.2.1A (Continued)

* Facility Key:

W = Webster
BA = BAARP
BL = Bloomington
GP = Greater Portland
B1 = Brooklyn I
PN = Philadelphia NW
O = Oneida
SP = South Phildelphia
B2 = Brooklyn II
H = Hempstead
N = Norfolk
YC = York County
ST = Stanislaus
SA = SANDER

4.2.1.4 EXPOSURE ASSESSMENTS

There are numerous areas where differences both in exposure assessment methodology and in site-specific exposure conditions can result in large differences in risk when two plants are being compared. Four of the most important factors are air dispersion modeling, source of emissions data, number of exposure pathways evaluated, and assumptions of values for exposure parameters.

Several different air dispersion models have been used in conducting risk assessments at resource recovery plants. Since the results of these models are often used for new source permitting in addition to risk assessment work, they usually follow state or USEPA protocols (USEPA 1986d). The most common model used is USEPA's Industrial Source Complex Long-Term (ISCLT) model often preceeded by a screening model such as PTPLU or PTMAX. Other models such as VALLEY and COMPLEX I have been used in complex terrain situations. In order to evaluate indirect pathways of exposure, deposition modeling is also required. Typically, emission factors are based on measured data from operating facilities, although there have been some cases in which thermodynamic have been used. USEPA (1987) calculates emissions by assuming a certain emission control efficiency for a given technology. For example, in evaluating projected efficiencies for new municipal waste combustion, USEPA assumed a generic control efficiency of 20% for organics and 99% for particulate matter and metals. This may or may not reflect conditions at an operating plant. The most accurate site-specific risk assessments use emissions data from operating facilities with similar characteristics to the facility under study. In the optimum, these characteristics should include waste feed capacity, degree of source separation, combustion temperature and detention time, type of stoker and grate, flue gas temperature, gas exit velocity and types of emission control devices in use. There is a fairly large literature base for numerou chemicals, including PCDDs/PCDFs (Siebert et al. 1987, MRI 1987). The data base for many potential trace contaminants, however, such as formaldehyde, polycyclic aromatic hydrocarbons, and antimony is severely limited, making comparisons with similar operating facilities extremely difficult.

The first risk assessments of resource recovery plants were concerned only with the direct inhalation pathway. Since then, most risk assessments have included more exposure pathways based on site-specific information. Pathways frequently analyzed include inadvertant consumption of soil, consumption of homegrown produce, consumption of fish from water bodies in the area of deposition, consumption of human-breast milk, consumption of agricultural produce, and dermal absorption from contacted from soil. The risks for the indirect pathways are often greater than the inhalation risks due to the tendency for some emitted materials (PCDDs/PCDFs and cadmium for example) to move through and bioconcentrate in the food chain.

The last area in which exposure assessments in site-specific risk assessments differ relates to the values assumed for a wide variety of exposure parameters. These can range from the amount of air breathed per unit time to the degradation rate constant for 2,3,7,8-T_4CDD in water. Many exposure parameters are population dependent. For example, inadvertant ingestion of soil is probably of greatest concern for children (Lagoy 1987), thus, facilities proposed for areas where there is a large young population would have to be evaluated using different parameters than those in areas with a very old population. Other exposure parameters are dependent on the environment. For example, rate constants for the photodegradation of 2,3,7,8-T_4CDD depend, in part, on latitude and season (Podoll et al. 1986). Dioxin emissions from a plant located in the northern United States may photolyze at a considerably different rate than those from a plant in the south. The key to site-specificity for exposure parameters is a careful survey of the area where the plant is to be located and the application of best scientific judgement to match up available values for parameters with those directly relevant to the site.

4.2.1.5. TOXICITY CONSIDERATIONS

In most recent risk assessments, the calculation of cancer risk is performed by means of what is termed a cancer potency factor. The lifetime cancer risk for a exposure to a chemical is calculated by multiplying thecancer potency factor [in $(mg/kg/day)^{-1}$] by the estimated chronic chemical intake (in mg/kg/day). The cancer potency factors are typically numbers generated by USEPA on the basis of extrapolation from animal experiments or from situations such as the workplace where human beings are exposed to high levels of chemicals.

Many of the chemicals potentially emitted from resource recovery plants occur in large families of compounds such as PCDDs/PCDFs (a family of 210 related compounds), PAHs, PCBs, and aldehydes. Often individual chemicals are chosen as surrogates for a class of compounds, for example, formaldehyde as a surrogate for the class of aldehydes or 2,3,6-trichlorophenol for the class of chlorinated phenols. Another approach is to assign a cancer potency factor to the total class as is done with polychlorinated biphenyls (Aroclor 1254). Still another method is to characterize members of the class by their toxicities relative to that of one compound in the class, as is typically done for the family of PCDDs/PCDFs. The most commonly used are those developed by the USEPA (1986e), Eadon et al. (1982), and the State of California (1983). The USEPA toxicity equivalency scheme was used for PCDDs/PCDFs.

4.2.1.6. REPORT ORGANIZATION

This report is divided into several sections, each of which addresses the issues relevant to evaluating the potential human health risk associated with the proposed Winona County resource recovery facility.

Section 4.2.2. presents a population and activity analysis for the proposed facility area. The section describes the demography of the surrounding community, identifies potentially sensitive receptor locations and population subgroups, and discusses the potential for abnormal epidemiological patterns among Winona residents. Important activity patterns that may influence the potential likelihood and extent of individual exposures are also discussed, including existing dairy or beef farms or produce farms in nearby areas, and fishing patterns among Winona residents.

In Section 4.2.3., Hazard Identification, the chemicals of greatest potential concern to human health that may be emitted from the facility are selected for detailed evaluation. The methodology used to describe the toxicity of these chemicals and the range of their toxic effects are also reviewed.

In Section 4.2.4., Exposure Assessment, the most important pathways through which individuals may be exposed to chemicals emitted from the proposed facility are selected for detailed evaluation. An exposure assessment is then presented which estimates the quantities of chemicals individuals may be exposed to through the selected exposure pathways.

In Section 4.2.5., Risk Characterization, the risks from exposure to the chemicals of concern are estimated for both carcinogenic and noncarcinogenic effects. In addition, the estimated chemical concentrations in the ambient air are compared with air toxics standards and guidelines for Minnesota and other states as well as for several other cuntries. A discussion of the interpretation of risks is also presented and the estimated risks are compared with risks associated with everyday activities. The uncertainties and limitations of this risk assessment are also discussed.

An ecological risk assessment is presented in Section 4.2.6. In this section, potential ecological receptors of importance are identified and toxicity data for the chemicals and receptors of concern are identified. An exposure assessment is then presented for ecological receptors and the potential ecological risks are estimated.

4.2.2. POPULATION AND ACTIVITY ANALYSIS

4.2.2.1. OBJECTIVE

The objective of this section is to more accurately identify potential receptors affected by the proposed resource recovery facility. General information concerning the Winona population is addressed. Demographic trends and population distributions are discussed, sensitive sub-populations are identified, and available epidemiological data are reported. Finally, general activity is categorically analyzed and quantitative information, when available, is presented.

4.2.2.2. DEMOGRAPHIC ANALYSIS

In order to perform a site-specific demographic analysis, ICF-Clement commissioned a study (CACI 1987) outlining the demographics of the Winona area. Based on the 1987 demographic update the population within a five-mile radius of the proposed Riverview Drive site was reported at 28,659. An additional 9,373 people live within five and ten miles of the site. The most recent estimate for the entire population of Winona County, reported for 1985, was 46,984 (Minnesota Center for Health Statistics 1987a). The five-mile radius population resides in an estimated 10,154 households, resulting in an average household size of 2.48. Within the five-to-ten mile range there are an additional 3,117 households.

On average, the Winona area has a relatively stable population size. The 0-11, 22-29, and 65+ age groups all contain roughly 15% of the population. There is only a six year difference (35.4 and 29.3) between the mean (average) and median (point at which 50% of population is younger, 50% older) age.

Homes in the area surrounding Winona are situated primarily in the numerous valleys extending perpendicularly away from the Mississippi. Due to the area's topography there are few plots (approximately ten) near Winona on which large scale farming (greater than two hundred acres) can occur. Although there are numerous smaller farms and livestock herds within a five mile radius of Winona, larger commercial operations are restricted to more distant, and relatively more level, countryside.

4.2.2.3. IDENTIFICATION OF SENSITIVE RECEPTOR LOCATIONS AND SENSITIVE POPULATION SUBGROUPS

Sensitive population subgroups are those identified as more likely to be adversely affected by emissions from the proposed Winona facility than the general public. In practice, this chiefly includes three categories: 1) children, whom by age are more likely to sustain longer exposures than adults, and whom by stage of development are at risk of more profound exposure effects than a mature individual; 2) the elderly, whose natural immune systems are weaker than those of the general population; and 3) the infirm, many of whom are also elderly, whose immune systems are weakened by illness. People suffering from respiratory or cardiovascular diseases, such as asthma, arteriosclerosis, emphysema, and bronchitis, may represent an even more sensitive subset of the infirm population. All these groups are represented in the Winona community. According to the CACI (1987) report, within a five mile radius of Winona in 1987 there were 2,009 children under the age of 5, an additional 2,334 children ages 5 to 11, and 4,286 individuals age 65 years or older. There also are sensitive ecological receptors in the Winona area, such as the Upper Mississippi River Wildlife and Fish Refuge, just upstream from Winona. These will be further discussed in the section titled Ecological Risk

Assessment.

Several receptor locations can be associated with these sensitive population subgroups. Young children are chiefly located in schools and playgrounds. Public school facilities in Winona include seven grade schools and one junior and one senior high schools. Additionally there are five parochial and two private grade schools, two parochial junior high schools, and one parochial senior high school. The nearest school to the Riverview Drive site is St. Matthews School, positioned approximately one half-mile to the south. Washington-Kosclusko School is the school closest to the Riverbend II site, located approximately one mile to the northwest. Four child care centers are also known to exist in Winona (Northwestern Bell 1987). There also are a number of public recreation areas in Winona, including Lake Park, West Lake Park, Mankato Field, Sobieski Park, and Gabrych Park (Winona Convention and Visitor's Bureau 1987). All of these sites are within the City of Winona, and therefore proximal to both sites proposed for the resource recovery facility.

Hospitals and nursing homes are the prime locations of the elderly and infirm. Winona has one hospital, Community Memorial Hospital, located approximately three fourths of a mile due west of the Riverbend II site along Mankato Avenue. There are three known nursing homes in Winona--St. Anne Hospice, Sauer Memorial Home, and the Watkins Home.

In addition to the three principal categories mentioned above, two other smaller sensitive sub-groups exist. First is the pica child, i.e., the child who habitually ingests non-food substances (Glickman et al. 1981). Habitual pica occurs in only a small percentage of the population and for only a small period of time. Mehaffy and Annest (1985) note that habitual pica is most prevalent in children six months to three years old. Due to the occasional exhibition of pica behavior in the general population, a small number of Winona children may exhibit habitual pica behavior, and therefore may be at greater risk due to their increased consumption of outdoor soil. Also at some elevated risk from the general Winona population are those people who regularly eat local game. Although accurate statistics on game consumption are unavailable, those individuals who regularly consume local game, as opposed to commercial meat or poultry (which is not locally produced), may be exposed to higher levels of chemicals emitted from the Winona facility.

4.2.2.4. ACTIVITY ANALYSIS

The residents of Winona engage in many activities that will influence the extent and magnitude of potential exposures to chemicals emitted from the proposed facility. Water-related activities such as fishing, boating and swimming are common, as are outdoor activities in general, such as bird watching, gardening, and park recreation. Additionally, numerous small farms exist within five miles of Winona at which animals (cows, pigs, chickens, horses) are raised and crops (hay, corn, oats, alfalfa, vegetables) are grown.

The Mississippi River with its many man-made pools is a natural attraction for fishing, boating, swimming, waterfowl hunting, and many other water-related sports. Over 500,000 man-hours were spent recreationally at the Pool 5 area, approximately five miles upstream of Winona, in 1978, over half of which were spent sport fishing (MDNR 1979). Activity at Pools 5a (approximately one mile upstream of Winona) and Pool 6 (extending from Winona until Lock and Dam Number 6, approximately eight miles downstream), are expected to be similar to Pool 5, for which accurate data have been collected. Although activity is much higher during the summer months, recreational use continues all year round. Boating, fishing and swimming are also popular at Lake Winona, and trout fishing occurs

in many of the local valley streams descending to the Mississippi.

Less than one mile north of Winona begins the Upper Mississippi River Wildlife and Fish Refuge. Besides being a sanctuary for numerous bird, animal and plant species, it is also a popular spot for bird watching, canoeing, and general nature observation. The Refuge and other nearby wetlands are considered great resources, prized for both their natural beauty and as tourist attractions.

The extensive wilderness areas around Winona are also favored locations for hunting and trapping. Winona County resident license sales in 1986 (MDNR 1987) included 1,850 small game hunting licenses, over 6,000 deer hunting licenses, and almost 200 trapping licenses. More than 1,000 migratory waterfowl and 450 pheasant licensing stamps were sold, as were numerous non-resident licenses in the various hunting categories. These figures demonstrate that a large proportion of Winona County citizens, roughly ten to twenty percent, may be regular outdoor sportsmen.

As mentioned previously, there is a wide variety of agricultural activity in Winona. Cows (beef and dairy), horses, pigs, and chickens are all raised on local farms. Although topography is the prime factor limiting farm size, numerous small farms with a few animals exist in the area, as do a smaller number of larger plots. Harvests are rich, and farmers are able to grow suitable crops to feed their own herds. Feed is primarily corn and hay, occasionally mixed with smaller amounts of oats, alfalfa, and nutritional supplements. An accurate numerical estimate of the degree of farming in the Winona area is difficult to obtain due to the large number of households with large backyard gardens as well as a few animals.

4.2.3. HAZARD IDENTIFICATION

The objective of this section is to present the analytical framework for evaluation of the potential human health effects associated with the proposed Winona resource recovery facility. The first subsection discusses the contaminants that were selected for detailed evaluation in this risk assessment. The second subsection presents a summary of health effects associated with exposure to these chemicals, and the health criteria for these chemicals that will be used to estimate potential human health risks.

4.2.3.1. SELECTION OF CONTAMINANTS OF CONCERN

The chemicals selected for detailed evaluation in this risk assessment were identified based on: (1) a list of chemicals of concern provided to ICF/Clement by the Minnesota Pollution Control Agency (MPCA); (2) chemicals frequently associated with combustion that are considered to pose unusually high health risks; and (3) chemicals that accounted for the preponderance of risks estimated in risk assessments of other resource recovery facilities.

The MPCA identified 16 chemicals or families of chemicals to be evaluated in the risk assessment. These chemicals included five criteria pollutants that are currently regulated under the U.S. Clean Air Act: nitrogen oxides, sulfur oxides, particulate matter, carbon monoxide, and lead. Also included in MPCA's list were 11 non-criteria pollutants. Among these were several inorganic compounds that would likely be emitted from the proposed facility: arsenic, beryllium, cadmium, chromium, mercury, and nickel. The remaining non-criteria pollutants were organic compounds that are generally present as mixtures. These include the families of: polychlorinoated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurnas (PCDFs) often referred to as dioxins and referred to in this report as PCDDs/PCDFs, polycyclic aromatic hydrocarbons (PAHs), chlorobenzenes, chlorophenols, and polychlorinated biphenyls (PCBs). In addition, MPCA subsequently requested that antimony be added to their list of chemicals of concern.

ICF/Clement's experience analyzing the emissions at other resource recovery facilities led to the addition of aldehydes and hydrogen chloride to the organic constituents list. These compounds have been found in the emissions from many combustion sources (MRI 1980).

The final list of chemicals selected for detailed evaluation in this risk assessment is shown in Table 4.2.3a.

4.2.3.2. CLASSIFICATION OF HEALTH EFFECTS

In the following subsection, a methodology for classifying health effects from exposure to chemicals is presented. This methodology has been recommended by USEPA (1986h,i) and, where necessary, adapted by ICF/Clement Associates to reflect the particular chemicals and types of exposures of concern in this risk assessment.

For the purposes of this risk assessment, long-term average chemical intake estimates (averaged over periods of a few years to a lifetime) for all of the important pathways of exposure will be compared with health criteria for chronic exposures.

For risk assessment purposes, individual pollutants are separated into two categories of chemical toxicity depending on whether they exhibit

TABLE 4.2.3.a
CHEMICALS SELECTED FOR EVALUATION IN THE
HEALTH RISK ASSESSMENT FOR THE PROPOSED RESOURCE
RECOVERY FACILITY IN WINONA, MINNESOTA

Aldehydes (treated as formaldehyde)
Antimony and compounds
Arsenic and compounds
Beryllium and compounds
Cadmium and compounds
Carbon monoxide
Chlorobenzenes (treated as 1,4-dichlorobenzene)
Chlorophenols (treated as 2,4,6-trichlorophenol)
Chromium and compounds (hexavalent)
Hydrogen chloride
Lead and compounds
Mercury and compounds (inorganic)
Nickel and compounds
Nitrogen oxides
Particulate matter
PCDDs and PCDFs (expressed as 2,3,7,8-T_4CDD equivalents)
Polychlorinated biphenyls (PCBs)
Polycyclic aromatic hydrocarbons (treated as benzo[a]pyrene)
Sulfur oxides (treated as sulfur dioxide)

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noncarcinogenic or carcinogenic (cancer-causing) effects. Classification into these categories is related to fundamentally different views of the biological mechanisms of action involved in the two cases. In the case of chemicals exhibiting noncarcinogenic effects, most authorities consider organisms to have repair and detoxification capabilities that must be exceeded by some critical concentration (threshold) before the toxic end point is mainfested. For example, an organ can have a large number of cells performing the same or similar functions that must be significantly depleted before the effect on the organ is seen. This threshold view holds that a range of exposures from just above zero to some finite value can be tolerated by the organism without an appreciable risk of adverse effects. For chemicals that exhibit carcinogenic effects, most authorities recognize that one or more molecular events can evoke changes in a single cell or a small number of cells that can lead to tumor formation. This is the nonthreshold theory of carcinogenesis which purports that any level of exposure to a carcinogen can result in some finite possibility of generating the disease. As a result, risks for exposure to potential carcinogens are usually expressed as probabilities.

Health Criteria for Noncarcinogenic End Points of Toxicity. Health criteria for chemicals exhibiting noncarcinogenic effects are generally developed using risk reference doses (RfDs) promulgated by the USEPA. These RfDs are usually derived either from human studies involving workplace exposures or from animal studies, and are adjusted using uncertainty factors. The RfD is an estimate of the daily exposure to a chemical which appears to present low risk of adverse effects during a lifetime of exposure by a person. The purpose of the RfD is to provide a benchmark to which other chemical intakes into the body (e.g., those projected from human exposure to various environmental conditions) might be compared. Intakes that are less than the RfD are not likely to be of concern. Intakes that are significantly greater than the RfD may indicate that an inadequate margin of safety could exist for

exposure to that substance. The RfD is an approximate number, and although chemical intakes higher than the RfD have a greater chance of producing an adverse effect, one should not infer that such intakes are, by definition, "unacceptable."

Where RfDs for specific compounds have not been developed by USEPA, the health effects criteria used in this risk assessment were derived from other sources including USEPA national ambient air quality standards (NAAQS), USEPA Health effects assessments, or threshold limit values (TLVs) proposed by the American Conference of Governmental Industrial Hygienists (ACGIH 1986).

The USEPA has not developed a comprehensive data base describing inhalation RfDs for the general population, and the TLVs were chosen as the basis of inhalation criteria for this report. The TLVs are based on exposure of a healthy worker for a 5-day work week, 8 hours per day. It is necessary to adjust the TLVs for application to the general population, and this involves the application of "uncertainty factors" to the TLV.

The uncertainty factor for chronic inhalation criteria is based on two components: a factor of 100 to account for the fact that workers are a relatively healthy (insensitive) subgroup of the population and are exposed only during their adult life and a factor of 2.8 to account for workers inhaling 10 m^3 in an 8-hour workday for 6 days per week while the average person breathes 20 m^3 of air in a 24-hour day for 7 days per week.

Health criteria are presented in two sets of units. For comparison with ambient airborne concentrations, criteria such as NAAQS and TLVs are presented in units of ug/m^3. For comparison with rates of intake into the body (either by ingestion or by absorption through the lungs or skin), health criteria are presented in units of mg/kg body weight/day. Conversion from one measure of exposure to another requires assumptions about average breathing rates, average rates of food consumption, and chemical-specific absorption factors. Development of inhalation criteria based on TLVs assumed that an average human adult weighs 70 kg and breathes 20 m^3/day.

For acute inhalation criteria, a safety factor of 10 is applied to the TLV to compensate for the healthy worker effect (formaldehyde and hydrogen chloride). A safety factor of 100 is applied to the ceiling TLV to compensate both for the healthy worker effect and for the fact that the ceiling value is possibly an effect level rather than an acceptable exposure level.

In response to MPCA's request, ICF/Clement Associates developed a Table (4.2.3.b) which summarizes the toxic endpoints associated with exposures to each of the selected chemicals of concern. The toxic endpoint used as the basis for the RfD is noted in this table (the most sensitive endpoint. Because the RfDs are based on the most sensitive endpoint, and because the maximum hazard indices for all the chemicals of concern, exposure pathways and scenarios were less than one, ICF,s conclusions were that potential effects as a result of the concentrations associated with facility emissions, would not be expected. Table 4.2.3.c lists the available effects levels for the most sensitive endpoint of eah chemical.

In addition, ICF at the request of MPCA and the Minnesota Depart of Health, developed a table presenting the rating of confidece in the RfDs contained in the Integrated Risk Information System (IRIS), Table 4.2.3.d. It should be noted that in a few cases, the most up-to-date RfD available in November 1987 for use in the Winona TWP has been updated by a newer RfD in IRIS.

Health Criteria for Carcinogenic End Points of Toxicity. For chemicals exhibiting carcinogenic effects, USEPA's Carcinogen Assessment Group (CAG) has estimated the excess lifetime cancer risks associated with various levels of exposure to potential human carcinogens by developing a value called a cancer potency factor (q_1^*). The cancer potency factor is a number which, when multiplied by the lifetime average dose of a potential carcinogen, yields the upper-bound lifetime individual cancer risk associated with exposure at that dose. An individual risk level of one in a million ($1x10^{-6}$), for example, represents an upper bound probability of 0.0001% that an individual will develop cancer over his or her lifetime as a result of lifetime exposure to a potential carcinogen.

In practice, cancer potency factors are derived from the results of human epidemiological studies or chronic animal bioassays. The animal studies are usually conducted using relatively high doses in order to observe possible adverse effects. Since humans are expected to be exposed at lower doses, the data are adjusted by using a mathematical model. The data from animal studies are fitted to the linearized multistage model and a dose-response curve is obtained. The low-dose slope of the dose-response curve is subjected to various adjustments and an interspecies scaling factor is applied to derive the cancer potency factor for humans. Dose-response data derived from human epidemiological studies are fitted to dose-time-response curves on an individual basis. These models provide rough, but plausible, estimates of the upper limits on lifetime risk. Although the actual risk is unlikely to be higher than the estimated risk, it could be considerably lower.

In addition, there are varying degrees of confidence in the weight of evidence for carcinogenicity of a given chemical. USEPA (1986a) has proposed a system for characterizing the overall weight of evidence for a chemical's carcinogenicity based on the availability of animal, human, and other supportive data. The weight-of-evidence classification is an attempt to determine the likelihood that an agent is a human carcinogen and thus affects the estimation of potential health risks. Three major steps are followed in characterizing the overall weight of evidence for carcinogenicity: (1) the quality of the evidence from human studies and (2) the quality of evidence from animal studies which are combined into a characterization of the overall weight of evidence for human carcinogenicity, and then (3) other supportive information which is assessed to determine whether the overall weight of evidence should be modified. USEPA's final classification of the overall evidence has five categories:

Group A--Human Carcinogen

This category indicates that there is sufficient evidence from epidemiological studies to support a causal association between an agent and cancer.

Group B--Probable Human Carcinogen

This category generally indicates that there is at least limited evidence from epidemiological studies of carcinogenicity to humans (Group B1) or that, in the absence of adequate data on humans, there is sufficient evidence of carcinogenicity in animals (Group B2).

Group C--Possible Human Carcinogen

This category indicates that there is limited evidence of carcinogenicity

in animals in the absence of data on humans.

Group D--Not Classified

This category indicates that the evidence for carcinogenicity in animals is inadequate.

Group E--No Evidence of Carcinogenicity to Humans

This category indicates that there is no evidence for carcinogenicity in at least two adequate animal tests in different species or in both epidemiological and animal studies.

The cancer risks developed in this report are all accompanied by this weight-of-evidence classification. The reader should note the qualitative differences between chemicals which have been demonstrated to be human carcinogens and those chemicals for which the evidence is limited.

Development of Criteria for Chemicals Exhibiting Route-Specific End Points of Toxicity. Among the slected contaminants of concern are several chemicals that exhibit route- specific end points of toxicity. Inhalation of beryllium, cadmium, chromium VI, and nickel is associated with increased incidences of pulmonary cancer in animals and/or humans. There is no adequate evidence, however, of systemic carcinogenic effects following oral exposure to these compounds. The USEPA utilizes separate methodologies for estimating the risks from oral or inhalation exposure to these compounds. This report follows the USEPA guidance for developing health criteria. The criteria for chronic inhalation exposure to these compounds were developed utilizing USEPA's cancer potency factors and the criteria for ingestion were developed utilizing RfDs intended for evaluation of oral exposures.

In addition, certain chemicals associated with increased incidences of cancer exhibit route-specific cancer end points and/or potencies. For example, B[a]P and arsenic have exposure route-specific cancer potency factors published by USEPA.

4.2.3.3. HEALTH EFFECTS SUMMARIES FOR THE SELECTED CHEMICALS OF CONCERN

This section includes a brief summary of the critical human health effects associated with the contaminants of concern at the proposed Winona facility.

4.2.3.3.1. ANTIMONY

4.2.3.3.1.1. Systemic Toxicity

Acute Toxicity. Acute antimony (Sb) toxicity in humans is characterized by nausea, vomiting, impaired liver function, pneumoconiosis, cardiovascular changes, skin rashes, and hemolytic anemia (USEPA 1980a).

Subchronic and Chronic Toxicity. Cardiovascular changes have been associated with chronic exposure to antimony. Exposure to either trivalent or pentavalent. Antimonial compounds can produce electrocardiogram (ECG) changes in humans. Histopathological evidence of cardiac edema, myocardial fibrosis, and other signs of myocardial structural damage indicate that antimony may produce even more severe, possibly permanent, myocardial damage in humans (USEPA 1980a). Parallel findings of functional changes in ECG patterns and of histopathological evidence of myocardial structural damage have also been obtained in animal toxicity studies. Pneumoconiosis in response to inhalation

exposure and dermatitis in response to skin exposure have also been observed in humans and experimental animals chronically exposed to antimony or its compounds (USEPA 1980a). Liver, kidney, spleen, and adrenal effects have been reported in experimental animals following subchronic or chronic exposure to antimony or its compounds (USEPA 1980a).

4.2.3.3.1.2. Teratogenicity and Reproductive Effects

A single report (Balyaeva 1967) indicated an increase in late occurring spontaneous abortions, premature births, and gynecological problems in female workers exposed to mixed dusts of antimony metal, Sb_2O_3, and Sb_2O_5, when compared with a control population. Studies reported by the same author, in which rats were administered 50 mg/kg antimony intraperitoneally or exposed to 250 mg/m^3 Sb_2O_3 dust 4 hr/day from day 1 to day 16 of gestation reported reduced litter sizes and abnormalities of the uterus and ovaries which were believed to complicate ovulation and implantation (Balyaeva 1967). Casals (1972) observed no fetal abnormalities when 125 or 250 mg/kg antimony dextran glycoside was administered to pregnant rats, although other reports indicate reduced rate of growth when rats were fed a diet containing 2% Sb_2O_3 (Gross et al. 1955), and a shortented life span in rats provided 5 ppm potassium antimony tartrate in drinking water (Schroeder et al. 1970).

4.2.3.3.1.3. Immunotoxicity

No information concerning the immunotoxicity of antimony was found in the available literature.

4.2.3.3.1.4. Mutagenicity

Antimony trioxide, antimony trichloride, and antimony pentachloride were reported to be positive in the rec-assay procedure in Bacillus subtilis (USEPA 1980a).

4.2.3.3.1.5. Carcinogenesis

The carcinogenic potential of antimony has been examined in experimental animals with negative results (Kanisawa and Schroeder 1969, Schroeder et al. 1970). Occupational exposure to antimony dust has been suggested to be associated with lung cancer deaths among antimony workers (Davies 1973).

4.2.3.3.1.6. Standards and Criteria

The ACGIH threshold limit value for antimony is 0.5 mg/m^3 (ACGIH 1986). Accounting for the fact that workers are a relatively healthy (insensitive) subgroup of the population, are exposed only during their adult lifetime, and are exposed for 8 hours/day, 5 days/week, and RfD of $3x10^{-5}$ mg/kg/day can be derived.

USEPA (1987a) calculated an RfD for oral exposure to antimony of 0.4 ug/kg/day based on a chronic oral bioassay in rats conducted by Schroeder et al. (1970). In this study, rats administered 0.35 mg/kg/day antimony in their drinking water exhibited decreased longevity and altered blood glucose and cholesterol levels.

4.2.3.3.2. ARSENIC

4.2.3.3.2.1. Systemic Toxicity

Acute Toxicity. Occupational exposure to high concentrations of arsenic trioxide is associated with severe irritation of the nasal mucosa, larynx, and bronchi (USEPA 1984a). In humans and experimental animals, acute oral exposure to arsenic results in gastroenteritis, diarrhea, and cardiovascular effects (USEPA 1984a).

Subchronic and Chronic Toxicity. Subchronic and chronic exposure to arsenic in humans and experimental animals has characteristic effects on the peripheral nervous system and, in children, on the central nervous system (USEPA 1984a). Biopsy and autopsy studies have provided histopathological evidence of peripheral neuropathic effects that are associated with sensory and motor nerve sensitivity or weakness (USEPA 1984a). Rats exposed to an arsenic trioxide aerosol resulting in an arsenic concentration of 46 ug/m^3 exhibited central nervous system functional deficits and structural damage (Rozenshtein 1970). In humans, anemia, cardiovascular effects, hepatic damage, possible renal damage, and nasal septum perforation have also been reported following chronic exposure to arsenic (USEPA 1984a).

4.2.3.3.2.2. Teratogenicity and Reproductive Effects

The number of spontaneous abortions was reported to be increased among women working in or near a smelter in Sweden (Nordstrom et al. 1978a,b). However, the number of agents involved with smelter work make it difficult to determine the specific effects that can be attributed to arsenic.

Arsenic has been found to be embryotoxic, fetotoxic, and teratogenic in several animal species. Prenatal intravenous or intraperitoneal injection of 15-41 mg/kg body weight sodium arsenate is associated with morphological and skeletal malformations in hamsters (Ferm and Carpenter 1968), mice (Hood and Bishop 1972), and rats (Beaudoin 1974). Oral exposure to 120 mg sodium arsenate/kg body weight in mice resulted in only minimal teratogenic and reproductive effects (Hood et al. 1977).

4.2.3.3.2.3. Immunotoxicity

Arsenic has been used therapeutically in the treatment of steroid-responding disorders and as a lymphocytostatic agent; certain effects associated with immune system disorders (e.g., herpes simplex and chronic pulmonary infections) have been observed with arsenic exposure (NAS 1977).

4.2.3.3.2.4. Mutagenicity

Arsenic is clastogenic and induced sister chromatid exchanges in a number of different cell types in vitro. Arsenic may also affect DNA by inhibition of DNA repair processes (USEPA 1984a).

4.2.3.3.2.5. Carcinogenicity

It is well established that inhalation of certain arsenic compounds can cause cancer in humans. Several studies of workers in smelters and plants manufacturing arsenical pesticides have shown significiant excess rates of death from lung cancer (USEPA 1984a). The carcinogenic effects of inhaled arsenic have only recently been demonstrated in experimental animals, using a model that enhanced retention of arsenic in the lung (Ishinishi et al. 1983, Pershagen et al. 1983).

Ingestion of arsenic-contaminated drinking water is associated with bladder, lung, and liver tumors (Chen et al. 1986). Tseng et al. (1968) and

Astolfi et al. (1981) reported an association between the ingestion of arsenic in drinking water and skin cancer (at concentrations around 1 ppm).

USEPA (1984a) has classified arsenic in Group A--Human Carcinogen. This category applies to chemical agents for which there is sufficient evidence of carcinogenicity from animal and human studies.

4.2.3.3.2.6. Standards and Criteria

USEPA (1984a) based it quantitative risk assessment for inhaled arsenic on five studies of three exposed worker populations (Higgins et al. 1982, Lee-Feldstein 1983, Brown and Chu 1982, 1983a,b, Enterline and Marsh 1980, Ott et al. 1974). All five studies showed excess risks of lung cancer that were related to the intensity and duration of exposure and the duration of follow-up (latency). The estimates of unit risk obtained from the five studies were in reasonably good agreement, ranging from 1.25×10^{-3} to 1.36×10^{-2} $(ug/m^3)^{-1}$. USEPA omitted the highest value, derived from the study of Ott et al. (1974) which was considered least reliable, and calculated the geometric mean for each of the two remaining populations and then an overall geometric mean to obtain a best estimate of 4.3×10^{-3} $(ug/m^3)^{-1}$ for the unit risk. Based on an assumed 20 m^3/day inhalation rate and a 30 percent absorption rate, the cancer potency factor is 50.1 $(mg/kg/day)^{-1}$.

USEPA (1984a) based its quantitative assessment for ingested arsenic on the study by Tseng et al. (1968). A cancer potency factor of 15 $(mg/kg/day)^{-1}$ was estimated. USEPA (1986a) is currently reevaluating the oral cancer potency of arsenic and may lower the quantitative estimate of carcinogenic potency (USEPA 1987i).

4.2.3.3.3. BERYLLIUM

4.2.3.3.3.1. Systemic Toxicity

Acute Toxicity. Acute respiratory effects associated with beryllium exposure include rhinitis, pharyngitis, tracheobronchitis, and acute pneumonitis. Dermal exposure to soluble beryllium compounds can cause contact dermatitis. Ocular effects include inflammation of the conjunctiva from splash burns or in association with contact dermatitis (USEPA 1986b).

Subchronic and Chronic Toxicity. The most common clinical symptoms caused by subchronic and chronic beryllium exposure are granulomatous lung inflammation with accompanying cough, chest pain, and general weakness. Chronic beryllium disease is generally manifested as lung effects without systemic toxicity (USEPA 1986b).

4.2.3.3.3.2. Teratogenicity and Reproductive Effects

Poor performance in behavior tests of offspring has been reported in mice exposed to beryllium sulfate during pregnancy (Tsujii and Hoshishima 1979). However, current data are not sufficient to determine if beryllium compounds have the potential to cause teratogenicity or reproductive toxicity (USEPA 1986b).

4.2.3.3.3.3. Immunotoxicity

No information concerning the immunotoxicity of beryllium was found in the available literature.

4.2.3.3.3.4. Mutagenicity

Beryllium sulfate and beryllium chloride were found to be nonmutagenic in all bacterial and yeast mutation assays. Beryllium was reported to be positive in some gene mutation assays and chromosomal aberration and sister-chromatid exchange studies and weakly positive in a DNA damage and repair assay (USEPA 1986b).

4.2.3.3.3.5. Carcinogenicity

A number of studies have shown that beryllium and several beryllium compounds are carcinogenic in experimental animals when delivered intravenously, intratracheally, or by inhalation, and that occupational exposure to beryllium may result in an increased lung cancer risk (USEPA 1986b). No neoplasms were observed following beryllium exposure by the oral or percutaneous routes (USEPA 1986b).

Applying the USEPA weight-of-evidence criteria for evaluating carcinogenic potential, beryllium is most appropriately classified in Group B1--Probable Human Carcinogen following inhalation exposure because of limited evidence from human studies and sufficient evidence from animal studies (USEPA 1986b).

4.2.3.3.3.6. Standards and Criteria

USEPA (1987a) established an RfD of 0.005 mg/kg/day for beryllium based on a study by Schroeder and Mitchner (1975) in which rats and mice were given 5 mg/liter beryllium in drinking water for lifetime; there were no differences in body weight or lifespan between exposed animals and controls.

The USEPA Carcinogen Assessment Group calculated a cancer potency factor (q_1^*) of 8.4 $(mg/kg/day)^{-1}$ for inhalation exposure to beryllium based on an epidemiological study (Wagoner et al. 1980) and industrial hygiene reviews (NIOSH 1972, Eisenbud and Lisson 1983).

4.2.3.3.4. CADMIUM

4.2.3.3.4.1. Systemic Toxicity

Acute Toxicity. In humans, symptoms from acute inhalation of cadmium oxide fumes include cough, dypsnea, chest pain, and shortness of breath; pulmonary edema may occur within 24 hours, often followed by bronchopneumonia (USEPA 1980b, 1985a). The first symptoms occurring after acute ingestion of cadmium are nausea, vomitting, diarrhea, muscular cramps, and salivation; other reported symptoms include abdominal pain, sensory disturbances, liver injury, and convulsions (USEPA 1985a).

Subchronic and Chronic Toxicity. Subchronic and chronic exposure to cadmium has been associated with a number of noncarcinogenic toxic effects in humans and experimental animals including hypertension, endocrine activities, osteomalacia, osteoperosis, anemia, emphysema, bronchitis, pulmonary function changes, and renal toxicity (USEPA 1985a,b). Renal toxcity (proteinurea) has been observed in humans and rats chronically exposed to cadmium (Friberg et al. 1974, Decker et al. 1958) and is considered the most significant noncarcinogenic end point of cadmium toxicity (USEPA 1985a,b). Renal toxicity occurs when cadmium reaches a concentration in the kidney of about 200 ug/g, a level that has been referred to as the critical concentration of cadmium (Goyer 1986). This value is based on the comprehensive literature review by Friberg et al. (1974), who examined data from animal studies and autopsy data from

cadmium-exposed workers. Friberg et al. (1974) estimated that a daily cadmium intake of 0.352 mg/day for 50 years would result in a renal cortex concentration of 200 ug/g.

4.2.3.3.4.2. Teratogenicity and Reproductive Effects

Exposure of female rats to 100 mg/liter cadmium in drinking water during gestation resulted in fetal growth retardation; developmental effects were not observed in rats exposed to 0.1 or 10 mg/liter cadmium (Ahokas et al. 1980). Schroeder and Mitchener (1971) reported decreased fertility, runting, young deaths, and sharp angulation of the distal third of the tail in a three-generation study in rodents. Sutou et al. (1980) exposed male and female mice to 0.0, 0.1, 1.0, or 10.0 mg/kg/day cadmium as cadmium chloride for six weeks prior to mating, during mating, and to pregnant females during the gestation period. Significant decreases in the number of implants and live fetus, increased resorption, decreased fetal body weight, and delayed ossification of the sternebrae and caudal vertebrae were observed at 10 mg/kg/day cadmium.

4.2.3.3.4.3. Immunotoxicity

Effects suggestive of immunosuppression have been reported in rabbits and mice exposed to cadmium in drinking water (USEPA 1985a). These effects have been noted in mice exposed to cadmium concentrations as low as 3 mg/liter (0.5 mg/kg/day) (Koller et al. 1975). Adverse effects include reduced serum antibody titers, reduced numbers of plaque-forming cells in the spleen, reduced ability of bone marrow lymphocytes to form rosettes, and decreased memory T-cell and B-cell activities (USEPA 1985a).

4.2.3.3.4.4. Mutagenicity

Results of mutagenicity studies are generally mixed and conflicting. Marginally positive responses were reported in gene mutation studies in mammalian cell cultures, mouse lymphoma cells and Chinese hamster lung and ovary cells (USEPA 1985a,b). Cadmium was also reported to be weakly positive in the rec-assay in Bacillus subtilis, positive in the dominant lethal test in Drosphila, and to cause chromosomal aberrations in several in vitro studies (USEPA 1985a,b).

4.2.3.3.4.5. Carcinogencity

Cancers of the prostrate and lung have been observed in cadmium smelter workers in an epidemiological study (Lemen et al. 1976). Thun et al. (1985) updated the Lemen et al. (1976) study and ascertained that the increased lung cancer risk was associated with cadmium inhalation exposure. In experimental animals, the incidence of lung carcinomas was significantly increased over controls in male and female Wistar rats exposed to a continuous (23 hours/day) airborne concentration of 12.5, 25, or 50 mg/m^3 cadmium for 18 months (Takenaka et al. 1983). No evidence of cadmium carcinogenicity has been found in chronic oral animal bioassays.

Cadmium has been classified by USEPA (1985a) on the basis of inhalation data in Group B1--Probable Human Carcinogen. This category applies to agents for which there is limited evidence of carcinogenicity from human studies and sufficient evidence from animal studies.

4.2.3.3.4.6. Standards and Criteria

USEPA (1987b) derived an oral RfD of $5x10^{-4}$ mg/kg/day for cadmium based on the Friberg et al. (1974) study in which exposure to 0.352 mg/day cadmium (0.005 mg/kg/day) for a 70-kg adult is associated with renal dysfunction. The RfD was used to develop a lifetime health advisory.

USEPA determined an inhalation cancer potency factor (q_1^*) of 6.1 $(mg/kg/day)^{-1}$ for cadmium based on the epidemiological data from the Thun et al. (1985) study (USEPA 1984b).

4.2.3.3.5 CARBON MONOXIDE

Inhalation of carbon monoxide from ambient air can increase the carbon monoxide level in the blood where it combines with hemoglobin to form carboxyhemoglobin. The presence of carboxyhemoglobin (COHb) in blood reduces the availability of oxygen to the tissues and inhibits the dissociation of the oxygen from hemoglobin (USEPA 1979).

4.2.3.3.5.1. Systemic Toxicity

Acute Toxicity. The primary health effects associated with acute (less than 8-hour) exposure to carbon monoxide in air are adverse cardiovascular and nervous system effects.

Adverse cardiovascular effects in experimental animals have been demonstrated following exposure to 58 mg/m^3 (50 ppm) carbon monoxide, resulting in COHb levels of 4-7% (USEPA 1979). Becker and Haak (1979) exposed 11 adult mongrel dogs to increasing concentrations of carbon monoxide 1 hour after coronary artery ligation. These exposures produced step-wise increases in the COHb level from 4.9% to 17%. The investigators reported an increase in myocardial ischemia with increasing carbon monoxide exposure.

In humans, 8-hour exposure to concentrations of carbon monoxide as low as 29 mg/m^3 (25 ppm), which resulted in a COHb level of 4%, was reported to affect behavior and the central nervous system (USEPA 1979). Numerous studies have demonstrated decrements in vigilance (alertness over time), visual perception, manual dexterity, learning ability, and performance of complex motor tasks (Beard and Wertheim 1967, USEPA 1984c). Groll-Knapp et al. (1982) reported marginal increases in deep sleep and decreases in rapid-eye-movement sleep folloing exposure to 100 ppm (115 mg/m^3) carbon monoxide for 8 hours, which resulted in a COHb level of 8%. Adverse effects on the central nervous system in experimental animals have been reported at airborne carbon monoxide concentrations as low as 115 mg/m^3 (100 ppm), which resulted in a COHb level of 12% (USEPA 1979). Several authors have reported adverse effects on general activity, sleep, and motor performance following carbon monoxide exposure in animals (USEPA 1979).

Subchronic and Chronic Toxicity. Subchronic exposure to carbon monoxide is associated with fibrinolytic effects in blood in humans and experimental animals. El-Attar and Sairo (1968) found an accelerated clot lysis time in workers chronically exposed to high levels of carbon monoxide (approximately 100 ppm, with occasional levels of 200-400 ppm); in 15 control subjects, no fibrinolytic activity was detected. Kalmaz et al. (1977) studied fibrinolytic activity in rabbits exposed for 8 weeks to an ambient concentration of 50 ppm (57 mg/m^3) carbon monoxide. The COHb levels reached 30.9% by the end of the exposure period. The investigators found increases in whole blood clotting time, serum fibrinogen degradation products, and an acceleration of whole blood clot lysis.

4.2.3.3.5.2. Teratogenicity and Reproductive Effects

Certain data suggest that perinatal effects, such as reduced birth weight and slowed development result from maternal exposure to carbon monoxide (USEPA 1979). Longo and Hill (1977) found that in sheep exposed to 58 mg/m^3 (50 ppm) carbon monoxide, the fetal COHb level was substantially higher (11.3%) than the maternal level (7.2%). These data suggest that the fetus is at special risk because of higher COHb levels than the mother. In human studies, reduced birth weight and slowed development have been reported in children of mothers who smoked cigarettes during pregnancy (USEPA 1984c).

4.2.3.3.5.3. Immunotoxicity

No information concerning the immunotoxicity of carbon monoxide was located in the available literature.

4.2.3.3.5.4. Mutagenicity

No information concerning the mutagenicity of carbon monoxide was located in the available literature.

4.2.3.3.5.5. Carcinogenicity

No information concerning the carcinogenicity of carbon monoxide was located in the available literature.

4.2.3.3.5.6. Standards and Criteria

USEPA (1980c) has developed national ambient air quality standards (NAAQS) for carbon monoxide. USEPA standards were set at 9 ppm, 8-hour average and 25 ppm, 1-hour average, originally based on the study by Beard and Wertheim (1967) in which inhalation exposure to 100 ppm for less than 1-hour affected performance in a behavioral test in rats.

4.2.3.3.6. CHROMIUM

Although both trivalent (CrIII) and hexavalent (CrVI) chromium are expected to be released in incinerator emissions chromium VI is a human carcinogen via the inhalation route of exposure and is therefore considered to be of greater concern in this risk assessment.

4.2.3.3.6.1. Systemic Toxicity

Acute Toxicity. Acute oral exposure to chromium VI compounds generally produces nausea, repeated vomitting, and diarrhea (USEPA 1985c).

Subchronic and Chronic Toxicity. Occupational inhalation exposure to hexavalent chromium is reported to be associated with nasal tissue damage (i.e., nasal septal perforation, ulcerated septum, inflamed mucosa) and pneumonia (USEPA 1984d). Subchronic and chronic dermal exposure to chromium VI as chromic acid is associated with contact dermatitis and ulceration of the skin (Burrows 1978). Epithelial necrosis, marked hyperplasia and atrophy of the pulmonary bronchi, emphysema-like changes, and atrophy of the spleen and liver were reported in mice exposed to an aerosol of 10 mg/m^3 calcium chromate (4.33 mg/m^3 chromium VI) 5 hours/day, 5 days/week for lifetime (Nettesheim et al. 1971).

One-year oral exposure of male and female rats to 0 to 25 mg/liter

chromium VI in drinking water did not result in significant differences in weight gain, appearance or pathological changes in the blood or other tissues (Mackenzie et al. 1958). Other studies reported no adverse health effects in animals chronically exposed to lower doses of chromium VI in drinking water (USEPA 1987c).

4.2.3.3.6.2. Teratogenicity and Reproductive Effects

No information concerning the teratogenicity or reproductive toxicity of chromium VI was found in the available literature.

4.2.3.3.6.3. Immunotoxicity

No information concerning the immunotoxicity of chromium VI was found in the available literature.

4.2.3.3.6.4. Mutagenicity

Chromium VI has been reported to be mutagenic in bacterial systems without metabolic activation and nonmutagenic with metabolic activation. Chromium VI has also been reported to be mutagenic in eukaryotic test systems and clastogenic in cultured mammalian cells (USEPA 1984d).

4.2.3.3.6.5. Carcinogenicity

In humans, epidemiological studies of occupational exposure to chromium compounds in the chromate, chrome-plating, and chrome pigment industries have revealed an association between occupational exposure to chromium and lung cancer. Mancusco and Hueper (1951) and Mancusco (1975) reported that lung cancer deaths were associated with chromium exposure in chromate workers. Inhalation bioassays in experimental animals have been negative. There is no evidence that chromium is carcinogenic in humans when ingested, and USEPA (1987c) does not consider chromium to be carcinogenic by the oral route of exposure.

USEPA has classified chromium VI in Group A--Human Carcinogen following inhalation exposure (USEPA 1987c). This category is for agents for which there is sufficient evidence to support the causal association between exposure to the agents and cancer.

4.2.3.3.6.6. Standards and Criteria

USEPA (1987a) developed an RfD of 5×10^{-3} mg/kg/day for noncarcinogenic effects of chromium VI based on the Mackenzie et al. (1958) chronic oral study in rats.

USEPA (1984e) derived a cancer potency factor (q_1^*) for exposure to chromium by inhalation based on the epidemiological study by Mancusco (1975). Expressed in terms of total intake, the cancer potency factor is 41 $(mg/kg/day)^{-1}$.

4.2.3.3.7. 1,4-DICHLOROBENZENE

Chlorobenzenes expected to be released in incinerator emissions occur in mixtures. 1,4-Dichlorobenzene (1,4-DCB) is a carcinogenic chlorobenzene; therefore, it is considered in this hazard assessment as a surrogate for all chlorobenzenes expected to be released in incinerator emissions.

4.2.3.3.7.1. Systemic Toxicity

Acute Toxicity. Short-term inhalation exposure to dichlorobenzenes is associated with headache, nausea, and throat irritation in humans. 1,4-Dichlorobenzene (1,4-DBC) is also an eye and skin irritant (USEPA 1987d). Acute oral or parenteral administration of dichlorobenzenes produces sedation and anesthesia in experimental animals (USEPA 1987d).

Subchronic and Chronic Toxicity. Liver and kidney effects have been reported in experimental animals exposed to 1,4-dichlorobenzene. Hollingsworth et al. (1956) exposed rats, guinea pigs, and rabbits to 4,800 mg/m^3 1,4-DCB vapor 7 hours/ days for 5 days/week. After 69 exposures, the observed effects included tremors, eye irritation, weight loss, liver degeneration and necrosis, cloudy swelling of renal tubular epithelium (rats), lung congestion, and emphysema (rabbits). Battelle-Columbus Laboratories (1979a,b) reported histopathological alterations in the liver, spleen, and thymus in mice and rats following 13-week exposures by gavage 5 days/week to doses of 675 and 800 mg/kg/day 1,4-DCB in the mouse and 300 and 600 mg/kg/day in the rat; hematopoietic hypolasia of the bone marrow was also found in mice and rats exposed to 1,500 mg/kg/day 1,4-DCB.

4.2.3.3.7.2. Teratogenicity and Reproductive Effects

Maternal inhalation exposure of up to 500 ppm in rabbits and rats did not result in any embryotoxic, fetotoxic, or teratogenic effects in offspring (Hodge et al. 1977, Hayes et al. 1985).

4.2.3.3.7.3 Immunotoxicity

No information concerning the immunotoxicity of 1,4-DCB was found in the available literature.

4.2.3.3.7.4 Mutagenicity

Exposure of the plant Allium to 1,4-DCB vapor for 4 hours resulted in polyploidy (abnormal chromosome number), lagging chromosomes, and abnormally shaped nuclei. Chromosomal breakage and mitotic anomalies have been reported in some plant test systems (USEPA 1980d).

4.2.3.3.7.5. Carcinogenicity

In an NTP bioassay (NTP 1987a), male and female F344 rats and B6C3F1 mice were exposed to 1,4-DCB in the diet for 2 years. 1,4-DCB produced a dose-related increase in the incidence of renal tubular cell adenocarcinomas in male rats and increased the incidences of hepatocellular carcinomas and hepatocellular adenomas in male and female mice.

USEPA (1987d) classified 1,4-DCB in Group C--Potential Human Carcinogen based on limited evidence of carcinogenicity in experimental animals (USEPA 1987d). However, the clear evidence of the carcinogenicity of 1,4-DCB in male rats and male and female mice reported in the NTP (1987a) bioassay suggests that 1,4-DCB may be more appropriately classified in Group B2--Probable Human Carcinogen.

4.2.3.3.7.6. Standards and Criteria

The USEPA Office of Drinking Water (USEPA 1987e) determined an RfD of 0.1 mg/kg/day based on the 13-week treatment study in rats by Battelle-Columbus

(1979b). This RfD was used to develop a lifetime health advisory.

USEPA (1986c) derived a provisional cancer potency factor (q_1*) for oral exposure to 1,4-DCB based on a draft of the NTP (1987) bioassay. The q_1* for 1,4-DCB is 2×10^{-2} $(mg/kg/day)^{-1}$ (USEPA 1986c).

4.2.3.3.8. FORMALDEHYDE

Aldehydes occur in emissions as complex mixtures. Formaldehyde is a representative aldehyde that undergoes chemical reactions typical of other aldehydes and its toxicity has been more adequately characterized.

4.2.3.3.8.1. Systemic Toxicity

Acute Toxicity. Observed effects in humans following acute inhalation exposure to formaldehyde include neurophysiological changes, eye irritation, nose and throat irritation, lower airway irritation, pulmonary lesions, and death (CIR 1984). Inhalation exposure of experimental animals to formaldehyde vapor results in similar health effects (CIR 1984). In humans, dermal exposure to formaldehyde can cause skin irritation, skin sensitization, and contact urticaria. Ocular exposure to aqueous formaldehyde solutions can cause conjunctival erythema and edema, corneal opacity, and loss of vision (CIR 1984).

Subchronic and Chronic Toxicity. Data on the chronic effects of formaldehyde in humans are derived largely from epidemiological and occupational exposure studies. Observed effects in humans following chronic inhalation exposure to formaldehyde include eye, nose, and throat irritation, bronchoconstriction, altered pulmonary function, headache, and fatigue (USEPA 1985d).

The chronic effects of formaldehyde have been studied in a variety of laboratory animals. Other observed effects in laboratory animals include inflammation of the lungs, reduced liver weights, and lesions of the kidney, liver, cerebral cortex, and respiratory tract (USEPA 1985d). Rats and dogs administered formaldehyde for 90 days in the drinking water or diet at oral doses of 150 mg/kg/day (rats) or 100 mg/kg/day (dogs) showed reduced weight gain (CIR 1984). Mice and rats exposed by inhalation to 5.6 or 14.3 ppm formaldehyde vapor for 6 hours/day, 5 days/week for 24 months developed a variety of nasal cavity lesions, including dysplasia and squamous metaplasia of respiratory epithelium, and purulent or seropurulent rhinitis (CIR 1984).

4.2.3.3.8.2. Teratogenicity and Reproductive Effects

No teratogenic effects have been observed in experimental animals following inhalation, oral, or dermal exposure to formaldehyde. The number of resorption sites was increased and mean litter size decreased in pregnant CD-1 mice exposed to oral doses of 185 mg/kg but not to 148 mg/kg formaldehyde on days 6-15 of gestation (Marks et al. 1980). Several reports also have presented evidence suggesting an increase in menstrual disorders (most commonly, dysmenorrhea) among women occupationally exposed to formaldehyde (USEPA 1985d).

4.2.3.3.8.3. Immunotoxicity

No information concerning the immunotoxicity of formaldehyde was found in the available literature.

4.2.3.3.8.4. Mutagenicity

Results of mutagenicity studies with formaldehyde are mixed. Formaldehyde has been shown to be mutagenic in yeast, fungi, Drosophila, grasshopper, and several species of bacteria (Escherichia coli, Pseudomonas fluorescens, Staphylococcus aureus). Both positive and negative results have been reported in the Ames Salmonella reversion-mutation assay (CIR 1984, USEPA 1985d).

In in vitro studies with mammalian systems, formaldehyde induced transformation in mouse BALB/c 3T3 cells, unscheduled DNA synthesis in HeLa cells, DNA-protein crosslinks in mouse L1210 cells and Chinese hamster V79 cells, and sister-chromatid exchanges in cultured human lymphocytes and Chinese hamster ovary cells. Formaldehyde was also mutagenic in the L5178Y mouse lymphoma assay but not in Chinese hamster ovary (CHO) cells or C3H/10TI/2 mouse embryo cells. No evidence of increased sister-chromatid exchanges, DNA damage, or chromosomal aberrations have been reported in rat in vivo assays following inhalation exposure (CIR 1984, USEPA 1985d).

4.2.3.3.8.5. Carcinogenicity

Evidence of the carcinogenicity of formaldehyde in humans is inconclusive (IARC 1982). Although some epidemiological studies suggest an association between exposure to formaldehyde and an excess risk of cancer, many studies show no increase in the cancer risk in exposed groups compared with control populations. Other studies lack sufficient numbers of cancer deaths or do not implicate formaldehyde clearly enough to demonstrate a conclusive carcinogenic effect.

Increased incidences of nasal carcinoma have been observed in rats exposed by inhalation to formaldehyde (Kerns et al. 1983a,b). In a study conducted by Battelle's Columbus Laboratories, groups of Fischer 344 rats and B6C3F1 mice were exposed by inhalation to 0, 2.5, 6.9, or 17.6 mg/m^3 formaldehyde for 6 hours/day, 5 days/week for up to 24 months. Two of 120 male mice exposed at 17.6 mg/m^3 developed squamous cell carcinoma of the nasal turbinates compared with 0/240 in the controls. No nasal carcinomas were found in any of the other dose groups. Although this tumor incidence is not statistically significant ($p>0.05$), the incidence of spontaneous nasal cancers in mice is very low. This has led USEPA (1984f) to regard these results as indicative of a carcinogenic effect. The incidences of squamous cell carcinoma of the nasal cavity in male and female rats were significantly higher than those reported for controls. The incidence of nasal carcinoma among rats exposed to 6.9 mg/m^3 formaldehyde were 1/119 and 1/116 for males and females, respectively. Although the incidence of nasal carcinoma in this dose group is not significantly increased above controls, these results suggest a dose-related trend, since spontaneous nasal carcinomas are also very rare in rats. No nasal carcinomas were observed in the 2.5-mg/m^3 exposure group (Kerns 1983a,b).

Applying the criteria described in USEPA's Guidelines for the Assessment of Carcinogenic Risk (USEPA 1986h), formaldehyde may be classified in Group B2--Probable Human Carcinogen. This category applies to agents for which there is inadequate evidence of carcinogenicity from human studies and sufficient evidence of carcinogenicity from animal studies.

4.2.3.3.8.6. Standards and Criteria

Brown (1985) estimated the lifetime cancer risk for formaldehyde based on the Battelle inhalation bioassay in rats (Kerns et al. 1983a,b) in which the incidence of squamous cell carcinoma of the nasal cavity was significantly

increased in high-dose males and females. Brown fitted the multistage model to the incidences of tumors occurring in male and female rats surviving at least 6 months and determined an upper 95th percentile cancer potency for inhalation of 1.7×10^{-3} $(mg/m^3)^{-1}$. Brown (1985) subsequently calculated cancer risks without adjustments for dfferences in the laboratory exposure regimen and expected environmental exposure in humans. After adjusting the exposure of 6 hours/day and 5 days/week in the experimental animals to continuous exposure, the upper 95th percentile cancer potency is 9.5×10^{-3} $(mg/m^3)^{-1}$; this corresponds to a cancer potency factor (q_1*) of 3.3×10^{-2} $(mg/kg/day)^{-1}$.

4.2.3.3.9. HYDROGEN CHLORIDE

4.2.3.3.9.1. Systemic Toxicity

Acute Toxicity. Hydrogen chloride gas is a strong irritant to the eyes, mucous membranes, and skin. High concentrations of the gas can cause eye irritation and may cause prolonged or permanent visual impairment and possibly total vision loss. When inhaled, it immediately causes irritation of the upper respiratory tract accompanied by burning sensation, choking, and coughing. Delayed effects may include laryngitis, bronchitis, and pulmonary edema. Ulceration of the nose and the throat may also occur (ITTI 1976, NIOSH 1981). When swallowed, hydrogen chloride causes mucous membrane burns, circumoral burns, hematemesis, respiratory distress (secondary to epiglottal edema), and renal failure. Residual sequelae may occur with lesions of the esophagus and gastrointestinal tract that may progress to scarring and strictures (Klaassen et al. 1986). Exposure of the skin to hydrogen chloride or hydrochloric acid will cause burns (NIOSH 1981).

Human, male volunteers exposed to hydrogen chloride found concentrations of 50 to 100 ppm barely tolerable for 1 hour; 35 ppm caused irritation of the throat after brief exposures; and 10 ppm was considered the maximum concentration acceptable for prolonged exposures (Henderson and Haggard 1943). Elkins (1959) reported that hydrogen chloride was immediately irritating to humans when inhaled at concentrations of 5 ppm or higher, and concentrations below 5 ppm resulted in no lasting effects.

Subchronic and Chronic Toxicity. Long-term inhalation exposure is primarily associated with irritation. Exposure of rabbits, guinea pigs, and pigeons to 100 ppm hydrogen chloride 6 hours/day for 50 days caused only slight unrest and irritation of the eyes and nose (Jones 1972). Inhalation of 33 ppm hydrogen chloride 6 hours/day for 20 exposures did not harm a monkey or smaller animals. Repeated exposures at higher concentrations resulted in weight loss that paralleled the severity of the exposure (Machle et al. 1942).

4.2.3.3.9.2. Teratogenicity and Reproductive Effects

No information concerning the teratogenicity or reproductive toxicity of hydrogen chloride was found in the available literature.

4.2.3.3.9.3. Immunotoxicity

No information concerning the immunotoxicity of hydrogen chloride was found in the available literature.

4.2.3.3.9.4. Mutagenicity

No information concerning the mutagenicity of hydrogen chloride was found in the available literature.

4.2.3.3.9.5. Carcinogenicity

No information concerning the carcinogenicity of hydrogen chloride was found in the available literature.

4.2.3.3.9.6. Standards and Criteria

The current OSHA standard for hydrogen chloride is a ceiling of 5 ppm (7 mg/m^3) (CFR 1979).

4.2.3.3.10. LEAD

4.2.3.3.10.1. Systemic Toxicity

Lead has diverse biological effects in humans and animals. Its effects are seen at the subcellular level of structures and processes as well as at the overall level of general functioning of the body. Considerable data exist on the effects of lead exposure in humans, but the effects are generally related to blood lead levels rather than to estimated exposure.

Acute Toxicity. Information concerning the toxicity of lead following acute inhalation exposure in humans and experimental animals or acute oral exposure in humans and experimental animals was not found in the available literature.

Subchronic and Chronic Toxicity. In humans, the major toxic effects caused by exposure to lead are alterations in the hematopoietic and nervous systems. Anemia caused by lead exposure results from the inhibition of heme synthesis. Impaired heme synthesis has been reported in adults at levels of less than 30 ug/dl lead in the blood (USEPA 1984g).

Subchronic and chronic exposure in humans to low doses of lead have been associated with alterations in heme biosynthesis (10 mg/dl), renal system effects and neurological alterations (15 mg/dl), IQ deficits in children (10-15 ug/l), and electroencephalogram readings (USEPA 1986g). A threshold has not yet been found for effects such as elevated levels of aminolevuluric acid (a potential neurotoxin) or stature effects (USEPA 1986g). Neurotoxic effects are associated with exposure to lead. Levels of lead in the blood (PbB) of over 80 ug/dl in children and over 100 ug/dl in sensitive adults can cause severe, irreversible brain damage, encephalopathy, and possibly death. Persons with these high levels may be asymptomatic or show only slight signs of intoxication, but rapid deterioration can occur. In children, permanent learning disabilities are seen at these levels, even if there are no overt signs of lead poisioning (USEPA 1984g).

In experimental animals, administration of 5 mg/liter lead acetate in drinking water to rats for 20 months produced depression of conduction tissue excitability and effects on systolic blood pressure and cardiac ATP concentrations (USEPA 1984g). Effects on kidneys were reported in rats at higher levels of lead arsenate in drinking water (USEPA 1984g).

4.2.3.3.10.2. Teratogenicity and Reproductive Effects

In humans, occupational and inhalation exposure to lead compounds during pregnancy is associated with elevated miscarriage rate, increases in spontaneous abortions, premature delivery, and early membrane rupture (USEPA 1984g).

No information on the potential teratogencity of lead or lead compounds in experimental animals was found in the available literature although postanatal development delays were reported in pups from rats that received 50-250 mg/liter lead in drinking water throughout gestation (Kimmel et al. 1976, Reiter et al. 1975). A number of studies have reported decreased fertility and fetotoxic effects in animals exposed to oral doses of lead greater than 250 mg/liter (USEPA 1984g). Schroeder and Mitchener (1971) exposed rats and mice of both sexes to 25 ppm lead in drinking water from weaning and allowed the animals to produce litters through 6 months (mice) or 9 months (rats) of age. Randomly selected pairs from F_1 litters were allowed to produce an F_2 generation and randomly selected pairs from F_2 generation were allowed to produce an F_3 generation; F_1 and F_2 pairs received the same treatments that their parents received. Delay in birth of the first litter of the original parents, runting and excessive mortality among the offspring before weaning, a decrease in the male/female ratio of the F_1 generation, and a decrease in pregnancies and litter size by the third generation were reported in rats; in mice, the effects were similar but more severe.

4.2.3.3.10.3. Immunotoxicity

No information concerning the immunotoxicity of lead or lead compounds was located in the available literature.

4.2.3.3.10.4. Mutagenicity

No information concerning the mutagencity of lead or lead compounds was located in the available literature.

4.2.3.3.10.5. Carcinogenicity

Oral ingestion of certain lead salts (lead acetate, lead phosphate, lead subacetate) has been associated with increased renal tumor frequency in rats (USEPA 1985e).

USEPA (1985e) noted that the available data provide an insufficient basis on which to regulate lead acetate, lead phosphate, and lead subacetate as human carcinogens. However, these lead salts have been classified by USEPA (1984g) in Group B2--Probable Human Carcinogen. This category applies to agents for which there is inadequate evidence of carcinogenicity from human studies and sufficient evidence from animal studies.

4.2.3.3.10.6 Standards and Criteria

The Clean Air Act national ambient air quality standard for lead is 1.5 ug/m^3 (USEPA 1984h). Assuming that a 70-kg adult breathes 20 m^3 air each day, an Rfd of $4.3x10^{-4}$ mg/kg/day can be derived.

The USEPA Office of Drinking Water established a maximum contaminant level goal (MCLG) of 20 ug/liter for lead based on the health effects of lead in infants and pregnant women as a sensitive subpopulation (USEPA 1985h). An oral RfD of $6x10^{-4}$ mg/kg/day can be derived based on the ODW analysis.

4.2.3.3.11. MERCURY

4.2.3.3.11.1. Systemic Toxicity

Mercury has long been recognized as one of the more toxic metals. The toxicity of mercury depends to some extent on its form; it can be part of both

inorganic and organic compounds.

Acute Toxicity. In humans, inhalation exposure to concentrations of 1 mg/m^3 inorganic mercury is associated with damage to lung tissue and acute mercurial pneumonitis; neurological effects have been reported following inhalation exposure at lower concentrations (USEPA 1984i). Ingestion of a single dose of inorganic mercury as mercuric chloride was reported to produce gastrointestinal distress (USEPA 1984i).

Subchronic and Chronic Toxicity. Classical symptoms of inorganic mercury vapor intoxication are mental disturbances, objective tremors, and gingivitis, which have been observed following chronic occupational exposure to average air concentrations of 0.1-0.2 mg/m^3 mercury (USEPA 1984i). The central nervous system appears to be the primary target of organic mercury intoxication. Primary lesions include destruction of cortical cerebral neurons and damage to the Purkinje cells and granular layer of the cerebellum. Clinical symptoms suggest damage to peripheral nerves, but histopathological documentation is lacking. Clinical symptoms following chronic oral exposure to organic mercury include paresthesia, loss of sensation in the extremities, ataxia, construction of the visual field, and hearing impairment (WHO 1976).

In animal studies, oral and subcutaneous injection exposure to mercury salts is associated with morphological kidney changes. Fitzhugh et al. (1950) fed male and female rats mercury as mercury acetate in food for up to 2 years and reported lesions in the proximal convoluted tubules of kidneys in animals exposed to 40 ppm but not to 10 ppm mercury acetate. Druet et al. (1978) exposed rats to mercuric chloride by subcutaneous injection three times a week for 12 weeks. Rats that received doses of 0.1 mg/kg or higher developed renal disease characterized by antiglomerular basement membrane antibodies and deposits in the glomerular tufts and small renal arteries; treatment-related changes were not reported in rats exposed to 0.05 mg/kg/injection mercuric chloride.

4.2.3.3.11.2. Teratogenicity and Reproductive Effects

Several investigators have reported embryotoxic and teratogenic effects in experimental animals exposed orally to organic mercury. The most common findings are neurological effects, but an increased incidence of cleft palate in mice has been reported (USEPA 1984i). Reduced birth rates and possible neurological damage have been reported at a dose of 0.1 mg/kg/day mercury (Fujita 1969). Brain damage, but not anatomical defects, has been reported in humans exposed prenatally to organic mercury; these epidemiological studies may not have been sensitive enough to detect possible teratogenic effects of organic mercury in human populations (USEPA 1984i).

4.2.3.3.11.3. Immunotoxicity

No information concerning the immunotoxicity of inorganic or organic mercury was found in the available litrature.

4.2.3.3.11.4. Mutagenicity

There is no evidence currently available to indicate that inorganic mercury and mercuric salts are mutagenic. Exposure to methyl mercury is associated with increased frequency of chromosome breaks in humans, blocked mitosis in human leukocytes in vivo and human cells in tissue culture, and point mutations in Drosophila (USEPA 1980e).

4.2.3.3.11.5. Carcinogenicity

No data are available regarding the carcinogenic potential of mercury in humans and animals.

4.2.3.3.11.6. Standards and Criteria

USEPA (1984i) determined an acceptable chronic intake of 3,6 ug/day for exposure to inorganic mercury based on available data that suggest that 0.005 mg/m^3 should be protective for subchronic inhalation exposures to inorganic mercury and mercury vapor. An RfD of $5x10^{-5}$ mg/kg/day can be derived for a 70 kg adult.

The USEPA Office of Drinking Water (USEPA 1987f) determined an oral RfD for inorganic mercury of $1.6x10^{-4}$ mg/kg/day based on the Druet et al. (1978) 12-week treatment study in rats. This RfD was used to develop the lifetime health advisory.

4.2.3.3.12. NICKEL

4.2.3.3.12.1. Systemic Toxicity

Acute Toxicity. Exposure to the acutely toxic organic nickel compound nickel carbonyl is associated with pathological pulmonary lesions in humans and experimental animals. Nephrotoxic effects (renal edema) have been also reported in humans and experimental animals following exposure to nickel salts (USEPA 1986d).

Subchronic and Chronic Toxicity. Nickel salts are known to produce allergic contact dermatitis, which most frequently develops after several years of continued low exposure (Klaassen and Amdur 1986). Once acquired, nickel sensitivity tends to persist. Chronic exposure to nickel is also associated with respiratory effects, including asthma, nasal septal perforations, and chronic rhinitis and sinusitis in humans and effects on alveolar macrophage activity in animals; endocrine effects in animals (decreased release of prolactin and decreased uptake of iodine); and cardiovascular effects in humans and animals (vasoconstriction, myocardial depression, and subcellular injury) (USEPA 1986d).

4.2.3.3.12.2. Teratogenicity/Reproductive Effects

Exposure to nickel and nickel compounds is associated with developmental and reproductive effects in experimental animals but not in humans. Sunderman et al. (1980) reported that inhalation exposure of hamsters to 0.06 mg/liter nickel carbonyl for 15 minutes on gestation day 4 or 5 resulted in decreased fetal viability and increased numbers of litters and fetuses with malformations (exencephaly and cystic lungs). Injection exposure to nickel has also been associated with delayed embryonic development and increased resorptions (USEPA 1986d).

In male rats, exposure to nickel is associated with degenerative changes in the testis, epididymis, and spermatozoa; in female mice, nickel may affect the ability of embryos to implant and the viability of recently implanted embryos (USEPA 1986d).

4.2.3.3.12.3. Immunotoxicity

Nickel chloride has been shown to significantly suppress natural killer

cell activity and decrease the percentage of T-lymphocytes in the spleens of mice following single intramuscular injection of 18.3 mg/kg (Smialowicz et al. 1984).

4.2.3.3.12.4. Mutagenicity

Nickel has been found to induce gene mutations in bacteria and cultured mammalian cells and to induce chromosomal aberrations in cultured mammalian cells and sister-chromatid exchanges in cultured mammalian cells and human lymphocytes (USEPA 1986d).

4.2.3.3.12.5. Carcinogenicity

The occurrence of statistically significant excesses of nasal cavity and lung cancers in nickel refinery workers has been conclusively demonstrated in a number of epidemiological studies. It is generally believed that these excess risks are primarily due to inhalation of metallic nickel, nickel subsulfide, nickel oxide, and nickel carbonyl. These conclusions have been supported by observations in experimental animals. However, because simultaneous exposure to several nickel compounds usually occurs in the workplace, it has been difficult to determine which specific compounds are carcinogenic under these conditions. There is no evidence that nickel is carcinogenic in humans when ingested, and USEPA (1987g) does not consider nickel to be carcinogenic by ingestion.

USEPA has classified inhaled nickel and certain nickel compounds in Group A--Human Carcinogen (USEPA 1987g). This category applies to agents for which there is sufficient evidence to support the causal association between human exposure to the agents and cancer.

4.2.3.3.12.6. Standards and Criteria

The USEPA Office of Drinking Water (USEPA 1987g) derived an RfD of 0.01 mg/kg/day for nickel based on a 2-year rat feeding study by Ambrose et al. (1976) in which no significant effects were reported in mice exposed to 100 ppm (5 mg/kg/day) nickel sulfate. The RfD was used to derive a lifetime health advisory for nickel.

The USEPA Carcinogen Assessment Group estimated unit risks for development of cancer associated with inhalation exposure to nickel based on the results of several epidemiological studies of nickel refinery workers. Using both the additive and multiplicative excess risk models and four data sets, a range of incremental unit risks from 1.1×10^{-5} $(ug/m^3)^{-1}$ to 4.6×10^{-4} $(ug/m^3)^{-1}$ was calculated with a midpoint of 2.4×10^{-4} $(ug/m^3)^{-1}$. The cancer potency factor for inhaled nickel based on the midpoint is 0.8 $(mg/kg/day)^{-1}$ (USEPA 1986d).

4.2.3.3.13. NITROGEN OXIDES

4.2.3.3.13.1. Systemic Toxicity

Acute Toxicity. Exposure to nitrogen dioxide (NO_2) has been found to adversely affect the respiratory system in humans. Increased airway resistance and impaired pulmonary function have been demonstrated to occur in healthy adults exposed to single 2-hour NO_2 concentrations ranging from 2.5 to 7 ppm. Some studies also suggest that exposure to nitrogen dioxide concentrations below 2 ppm for shorter durations (3-10 minutes) also affects pulmonary function in healthy and sensitive subjects (USEPA 1982a). A number of investigators reported no differences in pulmonary function tests in separate

epidemiological studies that involved NO_2 concentrations in the range of 0.02 and 0.51 ppm and other complex pollutant mixtures in ambient air (USEPA 1982a). Certain biochemical changes, such as significant decreases in hemoglobin, hematocrit, and erythrocyte acetylcholinesterase, were reported in 10 healthy adults following exposure to 1 ppm NO_2 for 2.5 hours (Posin et al. 1978). In experimental animals, short-term exposure to nitrogen dioxide has also been reported to result in changes in pulmonary function (USEPA 1982a). Exposure of animals to nitrogen dioxide has been associated with increased susceptibility to respiratory infection, decreased mucociliary transport, interference with hormone metabolism in the lung, morphological alterations of the lung tissue, and increased respiratory rate (USEPA 1982a).

Subchronic and Chronic Toxicity. Biochemical alterations have been reported in experimental animals following subchronic exposure to nitrogen dioxide. In guinea pigs exposed to 0.5 ppm NO_2 for 7 days, glutathione (GSH) peroxidase levels of red blood cells were increased (Donovan et al. 1976). Limited evidence from studies in experimental animals also suggests that exposure to nitrogen dioxide may result in alteration of brain enzyme levels (Drodzdz et al. 1975) and kidney, heart, and liver damage (USEPA 1982a).

4.2.3.3.13.2. Teratogenicity and Reproductive Effects

Inhalation exposure to nitrogen dioxide has not been found to be teratogenic in experimental animals (USEPA 1982a).

4.2.3.3.13.3. Immunotoxicity

No information concerning the immunotoxicity of nitrogen dioxide was found in the available literature.

4.2.3.3.13.4. Mutagenicity

Inhalation exposure to nitrogen dioxide has not been found to be mutagenic in experimental animals (USEPA 1982a).

4.2.3.3.13.5. Carcinogenicity

Inhalation exposure to nitrogen dioxide has not been found to be carcinogenic in experimental animals (USEPA 1982a).

4.2.3.3.13.6. Standards and Criteria

USEPA (1982a) noted that the existing annual standard for nitrogen dioxide (0.053 ppm) was based largely on a community epidemiological study (Shy et al. 1970) suggesting respiratory effects in children exposed to long-term low level NO_2 concentrations. However, because of the questionable accuracy of ambient monitoring for NO_2 used in the study, USEPA does not consider the study to be an accurate basis for retention of the existing standard.

4.2.3.3.14. PARTICULATE MATTER

The chemical composition of particulate matter is variable depending on its source, meteorology, and on the length of time it has been airborne (USEPA 1982b). The primary components of fine atmosphere particles are sulfates, carbonaceous material, ammonium, lead, and nitrate. Coarse particles include re-entrained surface dust, salt spray, and large particles produced in mechanical processes such as grinding. Oxides of silicon, aluminum, calcium, iron, calcium carbonate, sea salt, pollen, and spores are commonly occurring

coarse particles (USEPA 1982b). The toxicity of particulate matter depends to some extent upon its chemical composition. However, particulate matter comprised of nontoxic constituents may also have adverse health effects (USEPA 1982b).

4.2.3.3.14.1. Systemic Toxicity

Acute Toxicity. Brief exposure to particulate matter has been reported to adversely affect respiratory system function in humans and experimental animals (USEPA 1982c). Increased pulmonary resistance was reported in guinea pigs exposed to various particulate matter mixtures at concentrations of 100-1,000 ug/m^3 for 1 hour (Amdur and Underhill 1968, 1970, Amdur et al. 1978). Camner et al. (1973) found that human exposure to carbon particles in air accelerated mucociliary clearance in various regions of the respiratory tract.

Subchronic and Chronic Toxicity. A number of epidemiological studies suggest that populations living in areas with higher particulate pollution tend to have a higher prevalence of respiratory symptoms and reduced respiratory capability compared with populations living in less polluted areas (USEPA 1982c). Chronic exposure to a variety of different types of particles (direct acting irritants and insoluble dusts) is associated with morphological damage to the respiratory tract (USEPA 1982c). Insoluble particles accumulate in the alveolar region of the lung and may indirectly produce fibrosis through macrophage damage (USEPA 1982c). Autopsies of humans and experimental animals naturally exposed to a variety of particulates in ambient air have revealed fibrotic lesions, interstitial inflammation, and accumulation of crystal-laden macrophages in the lungs, and incidences of silicosis were found in workers occupationally exposed to high levels of silica (USEPA 1982b). In addition, daily fluctuations in air pollution in a number of cities were found to result in increased illness among individuals with preexisting cardio-respiratory disease (USEPA 1982c).

Silicon dioxide (SiO_2) is found in airborne coarse-mode particulate matter, and silicosis has been observed in experimental animals exposed to high concentrations of quartz and other SiO_2 dusts (USEPA 1982b).

4.2.3.3.14.2. Teratogenicity and Reproductive Effects

No information concerning the teratogenicity or reproductive toxicity of particulate matter was located in the available literature.

4.2.3.3.14.3. Immunotoxicity

Immunosuppression, characterized as depressed number of antibody-producing cells in the spleen, was reported in mice associated with intramuscular injections of nickel chloride (Graham et al. 1975). Graham et al. (1978) calculated that exposure to an aerosol of 0.25 mg/m^3 nickel for 2 hours results in a maximum deposition of 0.98 mg nickel in the mouse; this concentration was found to be the lowest tested that was found to produce a significant depression in immune response. Inhalation exposure to 0.19 mg/m^3 cadmium (as cadmium dichloride) for 2 hours resulted in significant suppression of immune response in mice (USEPA 1982b).

4.2.3.3.14.4. Mutagenicity

The mutagenic potential of particulate matter (specifically, matter retained by the filter medium used) has been examined (Kolbier et al. 1981). However, because of the possible interactions among the many compounds present

in any fraction of the abstracts, it is difficult to identify those compounds that act as direct mutagens. Data obtained with mammalian cell transformation assays and the Ames Salmonella assays suggest that the moderately polar and neutral fractions of the extracts contain significant portions of the total mutagenic activity (USEPA 1982b).

4.2.3.3.14.5. Carcinogenicity

Studies on the carcinogenicity of particles from atmospheres with high levels of coal combustion and industrial emissions reported that particulate organic matter, which often contains polycyclic aromatic hydrocarbons, were tumorigenic (Stern 1968, Wynder and Hoffman 1965). In addition, particulate matter may act as carriers for carcinogens such as polycyclic organics, chromium, arsenic, nickel, beryllium, asbestos, iron ore, and carbon, or these insoluble particles could delay lung clearance, which would allow the carcinogens to remain in contact with bronchial mucous membranes for longer periods of time (USEPA 1982c).

4.2.3.3.14.6. Standards and Criteria

USEPA has developed primary ambient air quality standards for the protection of public health for particulate matter of 50 ug/m^3 annual arithmetic mean and 150 ug/m^3 maximum 24-hour concentration (not to be exceeded more than once per year) (40 CFR 50) (EPA 1986e).

4.2.3.3.15. POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

Polycyclic aromatic hydrocarbons (PAHs) are a diverse class of compounds that are formed during the incomplete combustion of materials containing carbon and hydrogen. PAHs occur in the environment as complex mixtures of many components with widely varying noncarcinogenic and carcinogenic potencies. Only a few components of these mixtures have been characterized adequately, and only limited information is available on the relative potencies of different compounds. In addition, the toxicity of PAH mixtures has not been adequately characterized. The approach adopted by USEPA (1984j) as the basis of risk assessment is to divide the PAHs into two subclasses, "carcinogenic" PAHs and "noncarcinogenic" PAHs, and to apply a carcinogenic potency factor derived from assays on benzo[a]pyrene (B[a]P) to the subclass of carcinogeinc PAHs.

Use of the USEPA approach relies on the assumptions that (1) the toxic effects of all members of the class of carcinogenic PAHs are qualitatively and quantitatively similar to those of B[a]P and (2) that the toxic effects of different compounds of the mixtures are additive. With regard to the first assumption, B[a]P is one of the most potent carcinogens in the group of carcinogenic PAHs; based on limited experimental evidence, the other carcinogenic PAHs potentially emitted from the proposed incinerator are probably not as potent carcinogens as B[a]P. Risks would probably be overestimated from applying the potency factor for B[a]P to summed concentrations of these compounds. Assumption 2 is more substantially supported by the limited results of studies in which B[a]P was coadministered in combination with other carcinogenic PAHs. Therefore, the final estimates of exposure and risk from carcinogenic PAHs are likely to be near to or higher than (perhaps much higher than) the upper end of the range of actual exposure and risks. Classification of the carcinogenic potential of PAHs follows the guidance of the International Agency for Research on Cancer (IARC). Compounds determined to have sufficient or limited evidence for their carcinogenicity in animals are classified as carcinogenic PAHs.

4.2.3.3.15.1. Systemic Toxicity

Acute Toxicity. Acute effects from direct contact with PAHs and related materials are limited primarily to phototoxicity. Phototoxicity is caused by exposure to a toxic substance followed by exposure to sunlight. The primary effects are dermatitis--skin reddening, itching, and burning. NIOSH (1977) reviewed the phototoxic effects of exposure to coal tar and found that phototoxicity can result from a single 90-minute exposure to a 1% solution of coal tar. These dermatoses usually disappear when contact with the sensitizer is eliminated.

Subchronic and Chronic Toxicity. The polycyclic aromatic hydrocarbons have been shown to cause cytotoxicity in rapidly proliferating cells throughout the body, with the hematopoietic system, lymphoid system, and testes frequently noted as targets (Santodonato et al. 1981). This effect appears to be due to inhibition of DNA replication by the PAHs. Destruction of the sebaceous glands, hyperkeratosis, hyperplasia, and ulceration have been observed in mouse skin following dermal application of the carcinogenic PAHs, with the degree of induced morphological changes appearing to correlate with the carcinogenic activity. However, it does not seem that this dermal toxicity is necessary or sufficient for carcinogenesis (Santodonato et al. 1981). It should be noted that similar types of dermatitis have been observed in workers exposed to such PAH-containing materials as coal tar and mineral oil.

Some of the noncarcinogenic PAHs have been shown to cause systemic toxicity, but these effects are generally seen only at rather high doses (Santodonato et al. 1981). For example, slight morphological changes in the livers and kidneys of rats have been reported following oral administration of 0.6 g/kg/day acenaphthene in olive oil for 40 days (Knobloch et al. 1969). Aplastic anemia and, ultimately, death was reported in mice exposed to 120 mg/kg/day benzo[a]pyrene in the diet for 180 days (Robinson et al. 19 mating with control males delivered a litter; two of the pups were stillborn and one of the stillborn pups was malformed. Rigdon and Neal (1965) reported no deleterious reproductive effects in Swiss mice fed diets containing as much as 1 g/kg benzo[a]pyrene during mating, gestation, or postpartum. Mackenzie and Angevine (1981) found reduced fertility and reproductive capacity in the offspring of CD-1 mice exposed to an oral dose of 10 mg/kg benzo[a]pyrene by gavage on days 7-16 of gestation.

4.2.3.3.15.3. Immunotoxicity

The carcinogenic PAHs have been shown to have an immunosuppressive effect in animals. Wojdani et al. (1984) reported significant decreases in the percent of lymphocytes binding to target cells or killing target cells in mice exposed to 5 mg/kg benzo[a]pyrene via single intraperitoneal injection.

4.2.3.3.15.4. Mutagenicity

Many short-term genotoxicity tests have been performed with PAHs. A number of PAHs were found to be positive for mutation in bacterial assays and to cause DNA damage, mutation, and chromosomal effects in mammalian cells *in vitro* and *in vivo* (USEPA 1986f). Benzo[a]pyrene has been reported to be positive in assays for bacterial DNA repair and bacterial mutation, mutation in *Drosophila melanogaster*, and DNA repair, sister-chromatid exchange, chromosomal aberrations, point mutations, and transformation in mammalian cells (USEPA 1986f).

4.2.3.3.15.5. Carcinogenicity

Carcinogenicity is the toxic effect of greatest public health concern following exposure to materials containing carcinogenic PAHs. A number of the PAHs have been shown to be potent carcinogens, producing tumors both at the site of application and systemically, in several different animal species, when administered by any of a number of routes. Thyssen et al. (1981) reported treatment-related tumors of the nasal cavity, larynx, trachea, and pharynx in hamsters exposed to 9.5 mg/m^3 benzo[a]pyrene in air for 4.5 hours/day for 10 weeks followed by 3 hours/day, 7 days/week for up to 675 days; no tumors of these types were seen in control animals. In addition, IARC (1984, 1985) noted that occupational exposure to coal soot, coal tar and pitch, coal tar fumes, and some impure mineral oils causes cancer in humans at several sites, including the skin, and concluded that there is sufficient evidence that soots, tars, and some mineral oils are carcinogenic in humans. Fractionation procedures have demonstrated that PAHs are the carcinogenic agents in coal tar.

Oral exposure to carcinogenic PAHs is also associated with gastric tumor formation (Neal and Rigdon 1967). Rigdon and Neal (1969) reported gastric tumors, pulmonary adenomas, and leukemias in mice fed benzo[a]pyrene.

Benzo[a]pyrene is considered representative of the carcinogenic PAHs and is classified by USEPA (1986f) in Group B2--Probable Human Carcinogen. This category applies to agents for which there is sufficient animal evidence in the absence of human data.

4.2.3.3.15.6. Standards and Criteria

USEPA (1984j) previously published an inhalation cancer risk estimate of 6.1 $(mg/kg/day)^{-1}$ for benzo[a]pyrene based on the study of Thyssen et al. (1981).

USEPA (1984j) calculated a value of 11.5 $(mg/kg/day)^{-1}$ as the oral cancer potency factor for benzo[a]pyrene based on a study by Neal and Rigdon (1967) in which oral administration of B[a]P lead to forestomach tumors in mice.

4.2.3.3.16. POLYCHLORINATED BIPHENYLS

In considering the health effects of human exposure to polychlorinated biphenyls (PCBs) exposure observed in humans, it is important to note that used PCBs associated with exposure incidents are often contaminated with highly toxic impurities, particularly polychlorinated dibenzofurans (PCDFs). At least some of the reported effects may be due to the PCDF impurities.

Polychlorinated biphenyls are mixtures of chlorobiphenyls with varying degrees of chlorination. After release into the environment, the composition of PCB mixtures changes because of differential persistence, mobility, biouptake, and/or metabolism of their components. Conventionally, environmental residues of PCBs are analyzed by gas chromatography, compared to standards of various Aroclor mixtures, and quantitated by matching of major peaks to those in a standard of the most similar Arochlor (Webb and McCall 1973).

For most toxic end points, most of the toxicity of the PCB mixtures is associated with minor components which are not tested for or reported specifically in standard methods of analysis. The composition of mixtures changes after release into the environment, so that environmental residues rarely match any of the mixtures for which toxicity data are available. The cancer potency factor for PCBs is derived from a chronic bioassay using Aroclor 1260. This potency factor is used by USEPA for the risk assessment of

environmental concentrations of PCBs even though the composition of the mixture used in the laboratory bioassay is unlikely to be identical with the PCB mixtures found in the environment.

4.2.3.3.16.1. Systemic Toxicity

Acute Toxicity. PCBs are not highly toxic when given as a single oral dose to mammals (Kimbrough et al. 1978), and would be classified as only slightly toxic based on acute oral toxicity (Hodge and Sterner 1949).

Subchronic and Chronic Toxicity. Occupational exposure to PCBs is associated with hepatotoxicity, neurotoxicity, abnormal hematological findings, and chloracne (a disfiguring and long-term skin disease) and dermatitis (USEPA 1984k). Liver toxicity is a primary effect of PCB exposure in humans. Several studies examining liver function in exposed humans have reported disturbances in blood levels of liver enzymes. Maroni et al. (1981), Chase et al. (1982), Smith et al. (1982), and Emmett et al. (1983) found statistically significant associations between blood PCB levels and elevated levels of GGTP, SGOT, or both.

The liver is also a primary target organ for PCB toxicity in experimental animals. Fatty deposits and increased liver sizes are commonly reported. Individual hepatocytes may appear foamy and vacuolated. Cellular necrosis can occur at high doses, and the location of lesions is often centrilobular (Sleight 1983). Comparative studies have shown that Aroclor 1254 is usually the most hepatotoxic mixture. In rats, Aroclor 1260 was significantly less toxic to the liver than Aroclor 1254 (Kimbrough et al. 1972). Aroclors 1248, 1242, and 1016 are progressively less toxic than Aroclor 1254 (Koller 1977, Burse et al. 1974).

Subchronic oral exposure to PCBs in monkeys is also associated with degenerative change of the central nervous system and a skin syndrome similar to the effects seen in humans (USEPA 1984k). Barsotti et al. (1976) and Allen et al. (1979) fed diets containing 2.5 and 5 ppm Aroclor 1248 to rhesus monkeys for 7 months. Weight loss, followed by hair loss, acne of the face and neck, eyelid edema and erythema, and keratinization of hair follicles were reported.

4.2.3.3.16.2. Teratogenicity and Reproductive Effects

Reproductive effects of PCBs have been demonstrated in animals, but there appear to be major differences in the susceptibility of different species. Mink and monkeys appear to be relatively sensitive; mice, rats, and rabbits appear relatively insensitive.

Dietary administration of PCBs for 9 months prior to whelping caused lowered reproductive success in mink. Feeding Aroclor 1254 at 2 ppm or Aroclor 1243 at 5 ppm was detrimental to reproduction. Aroclor 1016 at concentrations as high as 10 ppm did not lower the number of young born per female. The PCBs were embryotoxic to developing embryos, but spermatogenesis, oogenesis, and implantation were unaffected. Transfer of PCBs to the surviving pups through mother's milk was a significant contributing factor to offspring exposure (Ringer 1983).

Monkeys also appear to be sensitive to the reproductive effects of PCBs. Altered menstrual cycles, reduced breeding success, and lowered birth rates were reported in monkeys exposed to dietary levels of 2.5 and 5 ppm Aroclor 1248 for 6-7 months before breeding (Barsotti et al. 1976). When female monkeys were exposed to 0.5 or 1 ppm Aroclor 1248 (three times per week) for

7 months prior to breeding (Allen et al. 1979, Bowman and Heironimus 1981), no toxic signs were observed in the mothers. The infants, however, had reduced birth weights, developed hyperpigmentation during nursing, and were marginally hyperactive at age 12 months.

Reproductive outcomes of women exposed to PCBs from high consumption of PCB-contaminated fish from Lake Michigan were compared with births from women who reported no such exposure (Fein et al. 1984a,b, Jacobson et al. 1983, Jacobson et al. 1984). Reduced birth weights, slow weight gain, reduced gestational ages, and behavioral deficits in infants were reported in a methodologically sound study. The study did not, however, rigorously establish that the causative factor was exposure to PCBs rather than other contaminants present in Lake Michigan fish.

4.2.3.3.16.3. Immunotoxicity

Immunotoxic and immunosuppressive effects have been reported in most experiments in which these end points have been investigated and are among the more sensitive indicators of PCB exposure (USEPA 1985f). Monkeys seem to be the most sensitive species; guinea pigs are relatively sensitive; and rabbits, rats, and mice appear relatively insensitive. Rhesus monkeys chronically exposed to Aroclor 1248 exhibited atrophy or hypercellularity of the thymus, spleen, lymph nodes, and bone marrow; these effects were particularly marked in infants after chronic exposures of their mothers to diets containing 2.5 ppm Aroclor 1248 (Allen and Barsotti 1976, Allen et al. 1980). Reductions in cell-mediated immunity, humoral immunity, or both were reported in monkeys chronically exposed to diets containing 1.5 or 5 ppm Aroclor 1248 (Thomas and Hinsdill 1978), and in monkeys dosed with 100 or 400 ug/kg/day Aroclor 1254 (Truelove et al. 1982). Increased susceptibilities to infectious diseases in PCB-exposed monkeys have been reported by Allen et al. (1979), McNulty et al. (1980), and Barsotti et al. (1976).

4.2.3.3.16.4 Mutagenicity

PCB mixtures and several isomers have been studied for genotoxic effects, and the results have been generally negative (USEPA 1985fa). Studies of the metabolic activation of PCBs and their ability to induce microsomal enzymes have demonstrated that PCB isomers and their metabolites bind to macromolecules, including DNA, with varying degrees of affinity. This suggests that PCBs are capable of damaging DNA, but it is unclear whether the damage is repairable or likely to produce permanent alterations in the genome. Their ability to induce enzymes responsible for the metabolic activation of other known mutagens and carcinogens may be the greatest genetic hazard posed by PCBs.

4.2.3.3.16.5. Carcinogenicity

A number of studies have suggested that PCB mixtures are capable of increasing the frequency of tumors in animals exposed to the mixtures for long periods. Kimbrough et al. (1975) exposed female rats to 0 and 100 ppm Aroclor 1260 in the diet for 20-21 months, and the treated animals had a high frequency of liver tumors. The National Cancer Institute (NCI 1978) reported significant dose-related increases of liver tumors and hyperplastic nodules in the livers of rats fed diets containing 0, 25, 50, or 100 ppm Aroclor 1254 for 104-105 weeks. Schaeffer et al. (1984) reported on a comparative study in male rats of the carcinogenicity and chronic toxicity of Clophen A60 (equivalent to Aroclor 1260) and Clophen A30 (equivalent to Aroclor 1242) fed at concentrations of 100 ppm in the diets for up to 832 days. A strong carcinogenic effect of Clophen

A60 (hepatocellular carcinomas and/or neoplastic nodules) and a weaker carcinogenic effect of Clophen A30 (neoplastic nodules) were reported. Studies have also suggested that PCB mixtures can act to promote or inhibit the action of other carcinogens in rats and mice (USEPA 1984k).

USEPA (1984k) classified the weight of the evidence for carcinogenicity as B2--Probable Human Carcinogen--based on sufficient evidence in animal bioassays and inadequate evidence from studies in humans. Based on the Schaeffer et al. (1984) study, one would expect less chlorinated PCB mixtures to be less potent, but potency factors for mixtures other than Aroclor 1260 have not been calculated.

4.2.3.3.16.6. Standards and Criteria

The USEPA Carcinogen Assessment Group (USEPA 1985f) calculated a low-level cancer potency factor for PCBs based on a study of rats exposed to Aroclor 1260 (Kimbrough et al. 1975). The data on liver tumor incidence in the rat study was used in the linearized multistage model to calculate 95% upper confidence limits on risk. The carcinogenic potency factor for lifetime oral exposure to PCBs is 4.34 $(mg/kg/day)^{-1}$.

4.2.3.3.17 POLYCHLORINATED DIBENZO-P-DIOXINS AND DIBENZOFURANS

The polychlorinated dibenzo-p-dioxins (PCDDs) comprise a family of 75 related organic compounds that contain between one and eight chlorine atoms. The polychlorinated dibenzofurans (PCDFs) comprise a family of 135 compounds containing one to eight chlorine atoms. A specific chemical nomenclature has been developed to describe these compounds. A congener is any member of the family with a unique chemical structure. Thus, we speak of 75 congeners of PCDD. Isomers are members of the family which possess the same number of chlorine atoms but with specified arrangements of the chlorine atoms (i.e., 2,3,7,8-tetrachlorodibenzo-p-dioxin). Homologues are groups of isomers with the same number of chlorine atoms. The hexachlorinated dibenzofuran homologue consists of 16 isomers, for example.

There is considerable variation among the congeners with regard to their distribution in emissions, chemical and physical properties, and toxic effects. In emissions from municipal incinerators, not all congeners are equally distributed. For example, trichlorinated congeners are present in much lower concentrations than the other homologues (NRCC 1984).

4.2.3.3.17.1 Systemic Toxicity

The isomer-specific quantifications of biological samples shows preferential uptake, absorption, and stroage of PCDDs and PCDFs having halogen substitution on the 2,3,7, and 8 positions to the molecule. Most toxicological studies of PCDDs and PCDFs have been conducted with the compounds 2,3,7,8-T_4CDD and 2,3,7,8-T_4CDF, respectively. The focus of this report is, therefore, on those congeners which have chlorine substituted in these positions in the molecules, as these are considered to be of greatest biological significance.

Acute Toxicity. In humans, the most consistently observed consequence of acute exposure to PCDDs is the skin disease chloracne (Crow 1981). 2,3,7,8-T_4CDD is an exceptionally potent toxin in several animal species; most animals die 20-40 days after exposure in a "wasting syndrome," which is characterized by progressive weight loss (USEPA 1985g). Other effects of acute exposure to 2,3,7,8-T_4CDD in animals include hypoplasia or atrophy of the thymus gland and skin and gastrointestinal lesions (USEPA 1985g).

PCDFs were found to cause fatigue; nausea; vomiting; ocular symptoms, including irritation and swelling (Higuchi 1976); and swelling of the extremities in individuals exposed to PCBs containing PCDFs in the "Yusho" accident in Japan in 1968 (Higuchi 1976). In experimental animals, signs of PCDF toxicity generally include slight growth retardation (Saeki et al. 1977); "wasting syndrome"; lymphoid depletion in the thymus, spleen, and bone marrow; renal hyperplasia; keratosis of the skin; thymic atrophy; and hepatomegaly (Moore et al. 1976, 1979).

Subchronic and Chronic Toxicity. Information on the potential health effects of subchronic and chronic exposure to 2,3,7,8-T_4CDD in humans has been primarily based on industrial and occupational exposures to herbicides and chlorophenol. Several studies of occupationally exposed workers or workers exposed in industrial accidents have shown porphyria cutanea tarda and elevated serum levels of triglycerides and gastrointestinal disease associated with 2,3,7,8-T_4CDD exposure (USEPA 1985g). In experimental animals, chronic exposure to 2,3,7,8-T_4CDD has been reported to result in effects on lymphoid tissues (Schwetz et al. 1973), skin and gastrointestinal lesions in monkeys (Schantz et al. 1979), and hepatotoxicity in rodents (Kociba et al. 1978).

Information on the potential health effects of subchronic and chronic exposure to PCDFs in humans has been based on studies of the "Yusho" poisoning incident in Japan and the "Yu-Cheng" poisoning incident in Taiwan. Reported effects following the Yusho incident included myocardial damage, chronic bronchitis, bursitis, persistent headaches, and dermal reactions (USEPA 1985g). In experimental animals, subchronic and chronic oral exposure to PCDFs resulted in dermal effects in mice (Nagayama et al. 1979), "chloracne-like" lesions and altered organ weights in rats (Oishi et al. 1978), and skin and gastrointestinal lesions in monkeys (McNulty et al. 1981, 1982).

4.2.3.3.17.2. Teratogenicity and Reproductive Effects

A number of studies have attempted to investigate reproductive outcomes in human populations putatively exposed to 2,3,7,8-T_4CDD, but these studies are severely compromised by difficulties in documenting exposure and in establishing rates of adverse reproductive outcomes in comparison populations. Lathrop et al. (1984) found significant increases in spontaneous abortions and in birth defects among wives of personnel directly involved in spraying "agent orange" in Southeast Asia. Hanify et al. (1981) found a statistical association between incidence of birth defects (heart defects and talipes) and wide-area spraying of 2,4,5-T. Several other studies have suggested a possible increase in spontaneous abortions among putatively exposed populations (Bruzzi et al. 1981, Matheson et al. 1981, Australian Veterans Heath Studies 1983). A case-control study of birth defects in the Atlanta area suggested that Vietnam veterans who had been exposed to "agent orange" had increased risks of fathering babies with spina bifida and cleft lip (Erickson et al. 1984). Overall, the evidence for an association between exposure to 2,3,7,8-T_4CDD and adverse reproductive outcomes in humans is inconclusive.

Many studies have demonstrated that 2,3,7,8-T_4CDD interferes with reproduction or causes birth defects in experimental animals. These reproductive studies involved a wide range of species and several routes of administration. Smith et al. (1976) reported that fetuses of mother CD-1 rats exposed by oral gavage to doses as low as 1 ug/kg from day 6 to day 15 of gestation exhibited an increase in cleft palate and kidney abnormalities; no adverse effects were observed in fetuses from animals exposed at lower dose levels. Fetal effects, including skeletal abnormalities and an increased incidence of kidney abnormalities, were observed in the offspring of rabbits

exposed by oral gavage to 0.1 ug/kg 2,3,7,8-T_4CDD on days 6-15 of gestation; this was the lowest dose tested in this study (Giavini et al. 1982). Murray et al. (1979) exposed male and female rats to 0.0, 0.001, 0.01, or 0.1 ug/kg/day 2,3,7,8-T_4CDD in the diet from 7 weeks of age to mating (F0 generation); subsequent generations (F1, F2, F3) were exposed from conception for lifetime. Low fertility was observed in F0 animals exposed to 0.1 ug/kg/day. Fetotoxicity and decreased reproductive capacity as adults were observed in F1 through F3 generations at the 0.01 ug/kg/day exposure level. An increased incidence of slightly dilated renal pelves was noted at 0.001 ug/kg in the F1 generation but not at any higher doses or in any subsequent generation. Schantz et al. (1979) reported that female rhesus macaques exposed to an average of 1.8 ng/kg/day TCDD for 7 months exhibited reduced fertility and fetotoxicity. Only two of seven pregnant females were able to produce viable offspring; all eight control females delivered live offspring.

Exposure to PCDFs is also associated with reproductive toxicity in humans and teratogenicity in experimental animals. Fetuses born to Yusho women frequently had reduced birth weights along with transient pigmentation of the skin (Funatsu et al. 1971, 1972). Still-births and cardiac anomalies have also been reported (Kikuchi et al. 1977).

In experimental animals, Weber et al. (1984) treated groups of six pregnant C57B1/6N mice by gavage with 0, 250, 500, or 1,000 ug/kg 2,3,7,8-T_4CDF on day 10 of gestation and groups of 8-11 pregnant mice with 0, 10, 30, 50, or 100 ug/kg/day on days 10-13 of gestation. A dose-related increase in fetal mortality was found in the groups receiving a single treatment. Both the singly and multiply treated groups showed dose-related increases in cleft palate and in kidney changes (largely hydronephrosis) on both a litter and fetus basis. In the high-dose groups receiving either the single or multiple treatments, 100% of the fetuses had kidney changes; this effect was significantly higher on a fetus basis in all treatment groups. The authors noted, however, that these effects are likely to be reversible with further development. Although the control group for the single treatments had no fetal mortality or kidney changes, nearly 12% of the fetuses in the other control group were affected by each of these end points. No cleft palates were observed, however, in any of the control litters. The incidence of cleft palate was significantly higher after maternal treatment with one dose of 1,000 ug/kg (87% of fetuses, 100% of litters) or four treatments of 50 ug/kg/day (16% of fetuses, 67% of litters). Despite the variability between the control groups and the possibility that the kidney changes may be transient, this experiment demonstrates that 2,3,7,8-T_4CDF can produce a clear teratogenic response when given to mice at doses of 50 ug/kg/day on days 10-13 of gestation or produce fetal loss when given as a single dose of 250 ug/kg on day 10. Comparing the results of this experiment with those of Moore et al. (1973), who used an almost identical protocol, the doses of 2,3,7,8-T_4CDF and 2,3,7,8-T_4CDD required to cause cleft palate in 50% of the litters when administered on days 10-13 were about 40 ug/kg and 2 ug/kg, respectively. Thus, 2,3,7,8-T_4CDF produces a similar teratogenic response to that of 2,3,7,8-T_4CDD but is about 20 times less potent.

4.2.3.3.17.3. Immunotoxicity

The effect of TCDDs on the human immune system has been extensively studied on residents of the Quail Run Mobile Home Park in Gray Summit, Missouri. Individuals at the mobile home park were exposed to TCDD when sludge wastes contaminated with TCDD were sprayed on dirt roads to keep the dust down. Hoffman et al. (1986) compared exposed and unexposed persons in the area and reported an increased frequency of anergy (diminished reactivity to specific

antigens) and relative anergy and decreased percentages of T3, T4, and T11 cells in the exposed population.

Many studies have shown that 2,3,7,8-T_4CDD is immunosuppressive in experimental animals. 2,3,7,8-T_4CDD appears to have a broad-spectrum immunosuppressive effect that includes hypoplasia or atrophy of the thymus, spleen, and bone marrow and interference with cell-mediated and antibody-mediated immune functions (USEPA 1985g). One important aspect of this effect is that 2,3,7,8-T_4CDD appears to depress the generation of cytotoxic T-lymphocytes at doses lower than those known to cause other biological effects in intact animals (Clark et al. 1983).

Few studies on the effect of PCDFs on the immune system have been reported. Luster et al. (1979a,b) exposed groups of four to eight female Hartley guinea pigs to six weekly oral doses of 0, 0.05, 0.17, 0.5, or 1 ug/kg of 2,3,7,8-T_4CDF (stated to be more than 98% pure). Thymus-to-body-weight ratios were reduced in the two or three highest dose groups, but spleen weights did not decline significantly. Exposure to the two highest dose levels significantly depressed cell-mediated immune functions, as indicated by decreased lymphocyte blastogenesis when cultured with T-mitogens, delayed hypersensitivity reactions, and reduced productions of the macrophage inhibitor factor. Effects at the two lower doses were not statistically significant. No significant effects on humoral immunity, as indicated by serum IgG levels and antibody response following immunization with bovine gamma globulin, were observed. The effects were similar both qualitatively and quantitatively to those induced by 2,3,7,8-T_4CDD under similar circumstances (Vos et al. 1973). The authors noted that neither 2,3,7,8-T_4CDF nor 2,3,7,8-T_4CDD induced severe immunotoxic effects in adult guinea pigs, but the effects of 2,3,7,8-T_4CDD were much more severe in young animals.

Vecchi et al. (1983a,b) compared the immunosuppressive effects of 2,3,7,8-T_4CDF and 2,3,7,8-T_4CDD in strains of mice that are "responsive" (C57B1/6) and "nonresponsive" (DBA/2) to AHH induction. 2,3,7,8-T_4CDF was dissolved in corn oil and administered to groups of seven to eight mice by intraperitoneal injection or gavage in single doses of 0, 5, 11, 22.5, 45, 90, 180, or 900 ug/kg. Thymus weights and spleen cell counts fell significantly in animals given the two highest dose levels. Humoral antibody production, as indicted by the number of spleen hemolytic plaque-forming cells in response to a challenge with sheep red blood cells, was significantly reduced in "responsive" mice at all doses from 11.5 ug/kg upwards. The effect was much smaller in "nonresponsive" mice (37% versus 85% inhibition at a dose of 180 ug/kg). The authors stated that the effects of 2,3,7,8-T_4CDF were similar to those produced by 2,3,7,8-T_4CDD at doses 30 times smaller. However, their data indicate that the ratio of effective doses was actually about 1:100. The effects of 2,3,7,8-T_4CDF had almost disappeared 42 days after treatment, in contrast to those of 2,3,7,8-T_4CDD, which were only slightly reduced after 42 days.

4.2.3.3.17.4. Mutagenicity

2,3,7,8-T_4CDD was found to be nonmutagenic in Salmonella typhimurium strain TA1532 with or without metabolic activation (USEPA 1985g). 2,3,7,8-T_4CDD has been found to be mutagenic in yeast and in the mouse lymphoma assay system. The available data suggest little potential for the interaction of 2,3,7,8-T_4CDD with nucleic acids or the ability of 2,3,7,8-T_4CDD to produce chromosomal aberrations (USEPA 1985g).

Schoeny (1982) tested five PCDFs for their ability to induce point

mutations in the Ames test. The chemicals were tested in up to 11 strains of Salmonella, usually with and without metabolic activation with S9 (rat liver microsomes induced with various chemicals, including Aroclor 1254 or 2,3,7,8-T_4CDF). None of the PCDFs induced a significant increase in revertant colonies.

4.2.3.3.17.5. Carcinogenicity

Several Swedish epidemiological studies have reported an association between occupational exposure to phenoxy acid herbicides or chlorophenols and increased incidence of certain cancers, including soft tissue sarcomas, non-Hodgkins's lymphomas, and nasopharyngeal cancers (USEPA 1985g). The presumptive link between these exposures and cancer is the presence of 2,3,7,8-T_4CDD or other PCDDs as impurities in phenoxy acids and chlorophenols. However, a case-control study of similar design in New Zealand on individuals exposed to phenoxy herbicides or chlorophenols (Smith et al. 1982, 1983, 1984) and two case-control studies investigating possible relationships between the occurrence of soft-tissue sarcomas and prior military service in Vietnam (Greenwald et al. 1984, Kogan and Clapp 1985) have yielded conflicting results. There have been several case reports of soft-tissue sarcomas among U.S. workers exposed to phenoxy acids and/or PCDDs (USEPA 1985g). However, Fingerhut et al. (1984) showed that some of these reports were based on erroneous pathological diagnoses. In a small but well-controlled study, Thiess et al. (1982) reported a significant excess of stomach cancers among workers presumptively exposed to 2,3,7,8-T_4CDD in a chemical reactor accident 23 years earlier. Other cancer studies have been inadequate to show either positive or negative results. Although some of these results suggest a possible association between exposure to 2,3,7,8-T_4CDD and increased risk of cancer, the evidence taken as a whole is inconclusive.

Three experiments have shown that 2,3,7,8-T_4CDD increases the frequency of cancer in rats and mice exposed to it for most or all of their lifetimes (Kociba et al. 1978, NTP 1982a,b). Two other studies (Van Miller et al. 1977, Toth et al. 1979) yielded similar but less definitive results. These studies indicated that the liver is an important target site for the carcinogenic action of 2,3,7,8-T_4CDD when it is administered orally. However, neoplastic responses were also seen in the thyroid, lymphatic system, lung, adrenal cortex, tongue, and hard palate and nasal turbinates in one or more studies. At least four studies (Van Miller et al. 1977, NTP 1982a,b in both mice and rats) showed a suggestive increase in soft-tissue tumors of histologic types similar to those reported in workers presumptively exposed to 2,3,7,8-T_4CDD. Thus, 2,3,7,8-T_4CDD appears to have a broad-spectrum neoplastic effect in at last two species, rats and mice. In addition to increasing tumor frequency when administered alone, 2,3,7,8-T_4CDD can promote or inhibit the effect of other cancer initiators (USEPA 1985g).

Poland et al. (1982, 1983) reported that 2,3,7,8-T_4CDF was a potent promoter of skin tumors in HRS/J hairless mice. A group of 20 female mice was given a single skin application of a tumor initiator (N-methyl-N'-nitro-N-nitroso-guanidine, or MNNG; 5 umol) and was then exposed to applications of 1 ug 2,3,7,8-T_4CDF in acetone, twice weekly for 20 weeks. All of the 19 surviving mice developed papillomas, with an average multiplicity of 4.9 tumors/mouse. None of the 23 mice treated with MNNG only and 1/20 mice treated with 2,3,7,8-T_4CDF only developed tumors. This appears to be the only report of testing of PCDFs for carcinogenicity.

USEPA (1985g) categorized 2,3,7,8-T_4CDD alone in Group B2--Probable Human Carcinogen. This category applies to agents for which there is sufficient

evidence of carcinogenicity in experimental animals and inadequate evidence in humans.

4.2.3.3.17.6. Standards and Criteria

USEPA (1987h) developed an RfD for 2,3,7,8-T_4CDD based on the three-generation reproduction study in rats by Murray et al. (1979). Although the authors considered the lowest dose tested, 0.001 ug/kg to be a no-observed-effect level, a reevaluation by Nisbet and Paxton (1982), using different statistical methods, revealed that toxic effects were observed at the 0.001 ug/kg dose. USEPA (1987h) considered 0.001 ug/kg to be a lowest-observed-adverse-effect level and, using an uncertainty factor of 1,000, derived a reference dose of $1x10^{-6}$ ug/kg/day for 2,3,7,8-T_4CDD.

For 2,3,7,8-T_4CDD, USEPA (1985g) calculated a low-level cancer potency factor based on a feeding study in female rats that induced a statistically significant increased incidence of tumors in the liver, lungs, hard palate, and nasal turbinates (Kociba et al. 1978). The data on tumor incidence in the rat study were used in the linearized multistage model to calculate 95% upper confidence limits on risk. The carcinogenic potency factor for lifetime exposure to 2,3,7,8-T_4CDD is $1.56x10^{5}$ $(mg/kg/day)^{-1}$.

Specific risk criteria have not yet been developed for PCDFS.

4.2.3.3.18. SULFUR OXIDES

4.2.3.3.18.1. Systemic Toxicity

Acute Toxicity. Nasal and eye irritation is associated with short-term (less than 30-minute) inhalation exposure to sulfur dioxide (SO_2). The primary effect of inhaled sulfur compounds is on the respiratory tract. Frank et al. (1964) exposed resting subjects to 1, 5, or 15 ppm sulfur dioxide for 30 minutes and reported increases in pulmonary flow resistance for concentrations of 5 ppm SO_2 or higher. Other investigators have also reported increased airway resistance or other bronchoconstrictive effects associated with sulfur dioxide exposures of 5 ppm SO_2 or higher (USEPA 1982b). Sulfuric acid and other sulfates have also been found to affect respiratory function in study subjects. Exposure to sulfuric acid mist (0.35 to 0.5 mg/m^3) has been reported to increase respiratory rate and decrease maximal inspiratory and expiratory flow rates and tidal volume (Amdur et al. 1952). However, several studies on human subjects since then have reported little effect on respiratory function following exposure to 0.1 to 1 mg/m^3 sulfuric acid for 10-120 minutes (USEPA 1982b). Short-term SO_2 exposure has been reported to result in bronchoconstruction in experimental animals at levels below 2.6 mg/m^3 (1 ppm) (USEPA 1982b). Transient decreases in respiratory rate following SO_2 exposure have also been reported (Alarie et al. 1973). Short-term exposure to 100 ug/m^3 has been reported to increase airway resistance in guinea pigs, the most sensitive laboratory animal species to aerosols of sulfuric acid (Amdur et al. 1978). There is also some evidence from controlled human exposure studies and studies in experimental animals to suggest that acute sulfur dioxide exposure affects respiratory defense mechanisms such as mucus clearance processes (USEPA 1982b).

Subchronic and Chronic Toxicity. Subchronic inhalation exposure is also associated with effects on mucociliary transport in experimental animals. In experimental animals, the effects of sulfur dioxide and sulfuric acid on mucociliary transport or immunological processes is variable and species dependent. Ferin and Leach (1973) noted that the rats exposed to SO_2 at

0.26 mg/m^3 (0.1 ppm), 7 hours/day, 5 days/week for either 2 or 3 weeks had increased mucociliary clearance. However, mucus flow in the trachea of dogs was reported to decrease following intermittent exposure for 1 year to SO_2 (Hirsch et al. 1975). Exposure to sulfuric acid mist at 500 mg/m^3 for 1 hour increased mucociliary transport in dogs, but exposure to 1,000 ug/m^3 sulfuric acid for 1-2 hours decreased clearance transport in rats and hamsters (USEPA 1982b).

4.2.3.3.18.2. Teratogenicity and Reproductive Effects

No information concerning the teratogenicity or reproductive toxicity of sulfur dioxide was located in the available literature.

4.2.3.3.18.3. Immunotoxicity

No information concerning the immunotoxicity of sulfur dioxide was located in the available literature.

4.2.3.3.18.4. Mutagenicity

Data concerning the mutagenicity of sulfur dioxide are rare. Sulfur dioxide mutagenicity has been demonstrated only in microorganisms *in vitro* at acidic pH levels (USEPA 1982b).

4.2.3.3.18.5. Carcinogenicity

USEPA (1982b) reanalyzed a study by Peacock and Spence (1967) in which increased incidences of primary pulmonary neoplasia were reported in male and female mice exposed to 500 ppm SO_2 for 5 minutes/day, 5 days/week for lifetime. USEPA concluded that SO_2 significantly increased the incidence of primary lung carcinoma in females and the incidence of primary lung adenomas in males and females. However, despite these results, USEPA (1982b) has concluded that SO_2 is suspect as a carcinogen.

4.2.3.3.18.6. Standards and Criteria

The primary national anbient air quality standard for sulfur oxides is an annual arithmetic mean of 80 mg/m^3 (40 CFR 50).

4.2.3.3.19. 2,4,6-TRICHLOROPHENOL

Chlorophenols expected to be released in incinerator emissions occur in mixtures. 2,4,6-Trichlorophenol is considered a probable human carcinogen and most likely to adversely affect human health; therefore it is discussed in the hazard assessment.

4.2.3.3.19.1. Systemic Toxicity

Acute Toxicity. Single intraperitoneal injection of 2,4,6-trichlorophenol produced convulsions in rats (Farquharson et al. 1958).

Subchronic and Chronic Toxicity. Relatively little information concerning the subchronic and chronic systemic toxicity of 2,4,6-trichlorophenol is available. In a subchronic range-finding experiment, NCI (1979) fed rats and mice of both sexes diets containing 2,4,6-trichlorophenol at dose levels ranging from 6,800 to 46,000 ppm for 7 weeks followed by an additional week of observation. Mean body weights of treated animals were considerably lower than controls. Marked increases in splenic hematopoiesis and midzonal vacuolization

of hepatocytes were observed in rats. No other remarkable changes were noted for treated animals (USEPA 1984l).

4.2.3.3.19.2. Teratogenicity and Reproductive Effects

No information concerning the teratogenicity or reproductive toxicity of 2,4,6-trichlorophenol was found in the available literature.

4.2.3.3.19.3. Immunotoxicty

No information concerning the immunotoxicity of 2,4,6-trichlorophenol was found in the available literature.

4.2.3.3.19.4. Mutagenicity

2,4,6-trichlorophenol was found to be negative in the Ames mutagenicity assay with and without metabolic activation (USEPA 1984l). In Saccharomyces cerevisiae, 2,4,6-trichlorophenol increased the mutation rate but not the rate of intragenic recombination (Fahrig et al. 1978). Recently, 2,4,6-trichlorophenol tested positive for mutagenicity in L517B7 mouse lymphoma cells (NTP 1987b).

4.2.3.3.19.5. Carcinogenicity

Results of at least three bioassays suggest that 2,4,6-trichlorophenol is carcinogenic in experimental animals (USEPA 1984l). In the NCI (1979) bioassay, the incidence of neoplasms of the hematopoietic system, lymphoma, and monocytic leukemia appeared to be treatment related in Fischer 344 rats exposed to 2,4,6-trichlorophenol in the diet. In B6C3F1 mice fed 2,4,6-trichlorophenol in the diet, the incidences of hepatocellular neoplasms (carcinomas or adenomas) were significantly elevated.

USEPA has classified 2,4,6-trichlorophenol in Group B2--Probable Human Carcinogen--based on the increased incidence of lymphomas or leukemias in male rats and hepatocellular adenomas or carcinomas in male and female mice reported in the NCI (1979) bioassay (USEPA 1984l).

4.2.3.3.19.6. Standards and Criteria

USEPA (1984l) calculated a cancer potency factor (q_1^*) of 1.98×10^{-2} $(mg/kg/day)^{-1}$ for oral exposure to 2,4,6-trichlorophenol from the NCI bioassay.

4.2.3.4. HEALTH EFFECTS CRITERIA

Health effects criteria for chronic inhalation of the selected contaminants of concern are presented in Table 4.2.3e and criteria for chronic ingestion of contaminants are presented in Table 4.2.3f.

As described in the previous section, it is anticipated that PAHs, PCDDs/PCDFs, and adehydes will be present as complex mixtures. To simplify the difficult task of criteria development and risk assessment for these mixtures, their toxicities will be evaluated based on the toxicity of B[a]P for PAHs, 2,3,7,8-T_4CDD for PCDDs/PCDFs, and formaldehyde for aldehydes.

Reference criteria have also been developed to evaluate the potential for acute inhalation effects associated with short-term exposures to aldehydes (treated as formaldehyde) and hydrogen chloride (HCl). The reference criterion for HCl of 70 mg/m^3 was derived from the ceiling threshold limit value (TLV) of

7,000 mg/m^3 (ACGIH 1986) divided by a safety factor of 100. The reference criterion for formaldehyde of 150 mg/m^3 was derived from the 8-hour time-weighted average TLV of 1,500 mg/m^3 (ACGIH 1986) divided by a safety factor of 10.

TABLE 4.2.3B

HEALTH EFFECTS ASSOCIATED WITH EXPOSURE TO SELECTED CHEMICALS OF CONCERN

Selected Chemicals Of Concern	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Aldehydes (treated as formaldehyde)		X	X	X	X	X		X						X	X
Antimony and compounds	X(a)		X		X		X	X	X	X				X	
Arsenic and compounds			X	X		X	X				X			X	X
Beryllium and compounds		X(a)						X						X	X
Cadmium and compounds				X	X	X	X				X	X		X	X
Carbon monoxide				X		X							X		
Chlorobenzenes (treated as 1,4-dichlorobenzene)		X	X(a)		X	X		X		X				X	X
Chlorophenols (treated as 2,4,6-trichlorophenol)		X	X							X					X
Chromium (VI) and compounds			X					X		X	X			X	X
Hydrogen chloride					X			X			X			X	
Lead and compounds				X	X	X(a)	X						X		
Mercury (inorganic) and compounds				X	X(a)	X								X	
Nickel and compounds		X(a)		X	X		X	X			X	X		X	X
Nitrogen oxides			X		X		X						X(a)	X	
Particulate matter														X	
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)		X		X	X		X	X			X	X			X
Polychlorinated biphenyls (PCBs)		X	X	X		X		X				X	X		X
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)				X				X	X			X	X		X
Sulfur oxides (treated as sulfur dioxide)														X	

(a) RfD based on upon footnoted health effect (endpoint).

1 = Decreased Longevity 2 = Growth Reduction 3 = Liver Effects 4 = Teratogenic/Reproductive Effects 5 = Kidney Effects
6 = Nervous System Effects 7 = Cardiovascular Effects 8 = Dermatologic Effects 9 = Lymphoid Necrosis 10 = Spleen Effects
11 = Gastrointestinal Effects 12 = Immunotoxic Effects 13 = Red Blood Cell Effects 14 = Respiratory Effects 15 = Carcinogenic Effects

TABLE 4.2.3C

OBSERVED ADVERSE EFFECT LEVELS ASSOCIATED WITH
THE SELECTED CHEMICALS OF CONCERN

Selected Chemicals Of Concern	Observed Adverse Effect Level Lowest	No	Species	Source
Aldehydes (treated as formaldehyde)		0.98 ppm	Rat	Rusch et al. 1983
Antimony and compounds mg/kg/day	0.35		Rat	Schroeder et al. 1970
Arsenic and compounds	a		--	--
Beryllium and compounds		0.54 mg/kg/day	Rat	Schroeder and Mitchner 1975
Cadmium and compounds	0.5 mg/kg/day		Mouse	Koller et al. 1975
Carbon monoxide	25 ppm		Human	USEPA 1979
Chlorobenzenes (treated as 1,4-dichlorobenzene)		150 mg/kg	Rat	NTP 1986
Chlorophenols (treated as 2,4,6-trichlorophenol)	b		--	--
Chromium (VI) and compounds		2.4 mg/kg/day	Rat	Mackenzie et al. 1958
Hydrogen chloride	c		--	--
Lead and compounds	5 mg/l		Rat	USEPA 1984b
Mercury (inorganic) and compounds	2 mg/kg/day		Rat	Fitzhugh et al. 1950
Nickel and compounds		5 mg/kg/day	Rat	Ambrose et al. 1976
Nitrogen oxides		1.0 mg/kg/day	Human	Walton 1951

TABLE 4.2.3C (Continued)

OBSERVED ADVERSE EFFECT LEVELS ASSOCIATED WITH
THE SELECTED CHEMICALS OF CONCERN

Selected Chemicals Of Concern	Observed Adverse Effect Level Lowest	No	Species	Source
Particulate matter	NDd		--	--
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)		0.001 mg/kg	Rat	Murray et al. 1979
Polychlorinated biphenyls (PCBs)	0.25 mg/kg		Monkey	Barsotti 1981
Polycyclic aromatic hydrocarbons (treated as benzo[a]pyrene)	e		--	--
Sulfur oxides (treated as sulfur dioxide)	2.6 ppm		Uf	USEPA 1982

a Value not place in table because status of LOAEL uncertain: 15 mg/kg in rodents reported by ICF-Clement (1987).

b Value not placed in table because status of LOAEL uncertain: 6,800 ppm in rats by NCI (1979).

c Value not placed in table because status of LOAEL uncertain: 5 ppm in humans by Elkins (1959).

d No data available.

e Value not place in table because status of LOAEL uncertain: 10 mg/kg in mice by MacKenzie and Angevine (1981).

f Uncertain of species tested.

TABLE 4.2.3D

REFERENCE DOSE LEVEL OF CONFIDENCEa REPORTED IN USEPA'S INTEGRATED RISK INFORMATION SYSTEM FOR THE SELECTED CHEMICALS OF CONCERN

	ORAL		
Selected Chemicals Of Concern	Reference Dose (mg/kg/day)	Source	Confidence
Aldehydes (treated as formaldehyde)	NAb	--	--
Antimony and compoundsc	4E-4c,d	Schroeder et al. 1970	Low
Arsenic and compounds	NAb	--	--
Beryllium and compounds	5E-3d	Schroeder and Mitchner 1975a	Low
Cadmium and compoundse,f	NA in IRISb	--	--g
Carbon monoxide	NAb	--	--
Chlorobenzenes (treated as 1,4-dichlorobenzene)h,i	NAb	--	--
Chlorophenols (treated as 2,4,6-trichlorophenol)	NC in IRISb	--	--
Chromium (VI) and compounds	5E-3d	Mackenzie et al. 1958	Low
Hydrogen chloride	NAb	--	--
Lead and compoundsj,k,f	NA in IRISb	--	--
Mercury (inorganic) and compoundsl,m	2E-3d	Fitzhugh et al. 1950	Medium
Nickel and compoundsn	2E-2d	Ambroze et al. 1976	Medium
Nitrogen oxidesf	1p	Walton 1951	High
Particulate matterf	NAb	--	--
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	NA in IRISb	--	--
Polychlorinated biphenyls (PCBs)	NAb	--	--
Polycyclic aromatic hydrocarbons (treated as benzo[a]pyrene)q	NC in IRISb	--	--
Sulfur oxides (treated as sulfur dioxide)f	NAb	--	--

TABLE 4.2.3D (Continued)

a Low, Medium or High. High confidence indicates the judgment that the RfD is unlikely to change in the future because there is consistency among the toxic responses observed in difference sexes, species, study designs, or in dose-response relationships, or that the reasons for existing differences are well understood. High confidence is often given to RfDs that are based on human data for the exposure route of concern, since in such cases the problems of interspecies extrapolation have been avoided. Low confidence indicates the judgment that the data supporting the RfD may be of limited quality and/or quantity and that additional information could result in a change in the RfD.

b NA = RfD Not available,
NA in IRIS = RfD not available in IRIS,
NC in IRIS = RfD not calculated in IRIS.

c A RfD has been calculated based on inhalation data of 3.0x10-5 mg/kg/day.

d RfD based on a chronic animal study.

e RfD calculated for the cadmium health advisory was 5.0X10-4 mg/kg/day.

f Regulated under the Clean Air Act.

g Review by USEPA pending.

h RfD calculated for the 1,4-dichlorobenzene health advisory was 0.1 mg/kg/day.

i An inhalation RfD has been calculated to be 0.1 mg/kg/day.

j RfD calculated for the Lead MCLG was 6.0x10-4 mg/kg/day.

k An inhalation RfD has been calculated to be 4.3x10-4 mg/kg/day.

l At the time of this report's publishing a RfD value of 1.6x10-4 mg/kg/day was used. The information came from the Health Advisory for Mercury. IRIS has since come out with its own RfD.

m An inhalation RfD has been calculated to be 5.0x10-5 mg/kg/day.

n At the time of this report's publication, a RfD value of 1.0x10-2 mg/kg/day was used. The information came from the Health Advisory for Nickel. IRIS has since come out with its own RfD.

p RfD based on human data.

q At the time of this report's publication, a RfD value of 1.0x10-6 mg/kg/day was used. This information came from the Health and Environmental Assessment for 2,3,7,8-TCDD.

TABLE 4.2.3E

HEALTH EFFECTS CRITERIA FOR INHALATION EXPOSURE TO CONTAMINANTS OF CONCERN

Contaminant	Reference Dose (RfD) (mg/kg/day)	Source a	EPA/CAG Cancer Potency Factor (mg/kg/day)-1	Weight of Evidence b
Antimony	3.0x10-5	TLV c	—	—
Arsenic	—	—	50	A
Beryllim	—	—	8.4	B1
Cadmium	—	—	6.1	B1
Carbon monoxide	—	—	—	—
Chromium VI	—	—	41	A
1,4-Dichlorobenzene f	0.1	HA	2.0x10-2	C(B2) g
Formaldehyde	—	—	3.3x10-2 e	B2
Hydrogen chloride	—	—	—	—
Lead	4.3x10-4	NAAQS d	—	—
Mercury (inorganic)	5.0x10-5	HEA	—	—
Nickel	—	—	0.8	A
Nitrogen oxides (NO2)	—	—	—	—
Particulate matter	—	—	—	—
PAHs:				
Carcinogenic(B[a]P)	—	—	6.1	B2
Noncarcinogenic	—	—	—	—
PCBs f	—	—	4.34	B2
PCDDs/PCDFs (2,3,7,8-T4CDD) f	—	—	1.56x10 5	B2
Sulfur oxides (SO2)	—	—	—	—
2,4,6-Trichloro-phenol f	—	—	2.0x10-2	B2

a HEA = Health Effects Assessment; IRIS = chemical files of the Integrated Risk Information System; NAAQS = National Ambient Air Quality Standards; TLV = ACGIH Threshold Limit Value.

b Weight of evidence classification scheme for carcinogens: A—Human Carcinogen, sufficient evidence from human epidemiological studies; B1—Probable Human Carcinogen, limited evidence from epidemiological studies and adequate evidence from animal studies; B2—Probable Human Carcinogen, inadequte evidence from epidemiological studies and adequate evidence from animal studies; C—Possible Human Carcinogen, limited evidence in animals in the absence of human data; D—Not Classified as to human carcinogenicity; and E—Evidence of Noncarcinogenicity.

c Conversion of TLV to mg/kg/day assumed an uncertainty factor of 100 to account for the fact workers are a relatively healthy (insensitive) subgroup of the population and a factor of 2.8 to account for workers inhaling 10 m3 air in an 8-hour work day for 5 days/week while the average person breathes 20 m3 air in a 24-hour day for 7 days/week.

$$\text{Mg/kg/day} = \text{TLV (mg/m3)} \times \frac{5 \text{ days}}{7} \times \frac{10 \text{ m3/day}}{20} \times \frac{1}{100 \text{ (uncertainty factor)}} \times \frac{1}{70\text{kg}}$$

d Conversion of NAAQS to units of mg/kg/day assumed that a 70-kg individual breathes 20 m3 air each day.

e Cancer potency factor calculated by Brown (1985) based on results of inhalation bioassay in rats (Kerns et al. 1983a,b) and adjusted for differences in laboratory exposure regimen and expected environmental exposure in humans.

f Ingestion health effects criteria are used for inhalation health effects criteria in this risk assessment.

g Originally classified in Group C (USEPA 1987d); ICF-Clement Associates considers 1,4-dichlorobenzene to be in Group B2 based on recent evidence of carcinogenicity in an animal bioassay.

TABLE 4.2.3F

HEALTH EFFECTS CRITERIA FOR ORAL EXPOSURE TO CONTAMINANTS OF CONCERN

Contaminant	Reference Dose (RfD) (mg/kg/day)	Source a	EPA/CAG Cancer Potency Factor (mg/kg/day)-1	Weight of Evidence b
Antimony	4.0x10-4	RfD/IRIS	—	—
Arsenic	—	—	15	A
Beryllim	5.0x10-3	RfD/IRIS	—	—
Cadmium	5.0x10-4	HA	—	—
Carbon monoxide c	—	—	—	—
Chromium VI	5.0x10-3	HEA	—	—
1,4-Dichlorobenzene	0.1	HA	2.0x10-2	C(B2) d
Formaldehyde c	—	—	—	—
Hydrogen chloride c	—	—	—	—
Lead	6.0x10-4	MCLG	—	—
Mercury (inorganic)	1.6x10-4	HA	—	—
Nickel	1.0x10-2	HA	—	—
Nitrogen oxides (NO2)	—	—	—	—
Particulate matter c	—	—	—	—
PAHs:				
Carcinogenic (B[a]P)	—	—	11.5	B2
Noncarcinogenic	—	—	—	—
PCBs	—	—	4.34	B2
PCDDs/PCDFs (2,3,7,8-T4CDD)	1.0x10-9	RfD/IRIS	1.56x10 5	B2
Sulfur oxides (SO2) c	—	—	—	—
2,4,6-Trichlorophenol	—	—	2.0x10-2	B2

a HEA = Health Effects Assessment; IRIS = chemical files of the Integrated Risk Information System; NAAQS = National Ambient Air Quality Standards; TLV = ACGIH Threshold Limit Value.

b Weight of evidence classification scheme for carcinogens: A—Human Carcinogen, sufficient evidence from human epidemiological studies; B1—Probable Human Carcinogen, limited evidence from epidemiological studies and adequate evidence from animal studies; B2—Probable Human Carcinogen, inadequte evidence from epidemiological studies and adequate evidence from animal studies; C—Possible Human Carcinogen, limited evidence in animals in the absence of human data; D—Not Classified as to human carcinogenicity; and E—Evidence of Noncarcinogenicity.

c Not evaluated by the oral route.

d Originally classified in Group C (USEPA 1987d); ICF-Clement Associates considers 1,4-dichlorobenzene to be in Group B2 based on recent evidence of carcinogenicity in an animal bioassay.

4.2.4. EXPOSURE ASSESSMENT

An exposure assessment was conducted for the each of the two proposed Winona facility sites to determine the potential intake of the chemicals of concern by receptors. Nine exposure pathways were selected for detailed evaluation in the exposure assessment:

1. Inhalation;
2. Ingestion via inhalation;
3. Ingestion of outdoor soil;
4. Ingestion of indoor dust;
5. Ingestion of fish;
6. Ingestion of cow's milk;
7. Ingestion of beef;
8. Ingestion of home-grown produce; and
9. Ingestion of human breast-milk.

For each pathway, potential chemical concentrations in the environmental media of concern were estimated for an average and a maximum case. Intakes were then calculated for average case and maximum case exposure scenarios based on site-specific information describing the potential magnitude, frequency and duration of exposure.

4.2.4.1. INTRODUCTION

Contaminants emitted from the proposed Winona facility will be distributed widely in the air and may be deposited at varying rates onto a wide area of land and water. As a result, residents may be exposed to these contaminants in varying quantities and proportions via a wide variety of exposure routes. These routes include inhalation of air; ingestion of outdoor soil, produce, fish, milk, beef, and indoor dust; and direct dermal contact with outdoor soil. The frequency, extent, and duration of exposure for any individual person will depend on many factors, including location of residence, age, sex, and activity patterns.

The complexity of exposure to environmental contaminants requires a series of simplifying steps in the process of exposure assessment. This sectionis based on ICF/Clement TWP and follows a general approach to exposure assessment which has been developed by specialists in this field and has been codified in general terms by the U.S. Environmental Protection Agency (USEPA 1986a,b,c). In this general approach, exposure assessment is conducted in two general steps. The first step (Section IX-C,TWP) is a pathway analysis in which the exposure pathways most likely to be significant are extracted from a list of all possible pathways. This screening process is necessary to focus the assessment on the most significant exposure pathways and is justified by experience, which shows that in most circumstances exposure via a few exposure pathways to one or a few chemicals dominates estimates of potential risks.

The second step(Section IX.D,TWP) is a detailed assessment of exposures. For each exposure pathway, an exposure scenario is developed based on assumptions about the environmental behavior and transport of the contaminants, and the extent, frequency, and duration of exposures. These factors are used to predict potential intakes of the chemicals of concern for both an average and a maximum exposure case.

4.2.4.2. IDENTIFICATION OF EXPOSURE PATHWAYS

The purpose of this section is to identify the most significant potential pathways through which humans may be exposed to the chemicals released from the proposed Winona plant. These emitted contaminants may be suspended in the ambient air, transported in the atmosphere, and deposited on the ground.

An exposure pathway is composed of the following four elements (USEPA 1986b):

(1) a source and mechanism of chemical release to the environment;

(2) an environmental transport medium (e.g., air) for the released chemical, and/or a mechanism of transfer of the chemical from one medium to another (e.g., deposition of particles onto soil);

(3) a point of potential contact of humans or biota with the contaminated medium (the exposure point); and

(4) an exposure route (e.g., inhalation) at the exposure point.

All four elements must be present for a pathway to be considered complete.

One of the most important exposure pathways associated with resource recovery facilities involves the direct inhalation of airborne contaminants. The inhalation of chemicals which are associated with fly ash particles can result in exposures through two different routes depending upon the area of the lung in which they are deposited. Some of the inhaled particles will be deposited in the lower pulmonary region (alveoli) where functional gas exchange occurs. Another portion of the inhaled particles will be deposited in the upper respiratory tract (trachea and terminal bronchioles) where they may be cleared via the mucociliary clearance system and subsequently swallowed. A third portion of the inhaled particles will be deposited in the nasal and pharyngeal areas from which they also may be swallowed. The final portion of inhaled particles will be exhaled. Accordingly, in this assessment, potential ingestion exposures as a result of inhalation and direct inhalation exposures will both be evaluated. Two exposure points will be focused on for these exposure pathways: the location of the greatest airborne concentrations in Winona (to include the highest possible individual exposure) and the overall Winona area (to include average population exposures).

Some fraction of the chemicals emitted from the proposed incinerator will be deposited onto the ground or onto water, for example as a result of the settling of dry particles. The deposited chemicals may result in indirect exposures through several pathways. Deposited chemicals may become mixed into soil where individuals, such as children or gardeners, may come into contact with them. Children in particular are known to ingest soils either incidentally or intentionally (i.e., pica) while playing. Thus potential exposures due to the incidental ingestion of soil by both children and adults (e.g., a gardener) will be evaluated.

Chemicals in soils which contact the exposed skin of children or backyard gardeners may also be absorbed. As described in Section IV.C, TWP, the exposures and risks potentially associated with this pathway are usually small in comparison to the potential exposures and risks associated with other indirect pathways of exposure. In addition, available methodologies for quantifying exposures and risks associated with the dermal absorption of chemicals from a soil matrix, as a result of short duration exposures, are not well developed, primarily due to the lack of relevant experimental data. Thus potential exposures to deposited chemicals adsorbed to soils via dermal

absorption will not be quantitatively evaluated in this risk assessment.

The fourth exposure pathway to be evaluated in the risk assessment involves the incidental ingestion of household dust. Ingestion of dust inside the home is considered because outdoor airborne particulate matter or tracking of surface soil contribute to a large percentage of the dust inside homes. Children playing or crawling on all fours inside the home collect dust on their hands. This dust may then be ingested when the children put their hands in their mouths. Thus potential exposures to children as a result of incidental dust ingestion in the home will be evaluated in this risk assessment.

As described in the TWP, (Section VIII), a wide variety of crops are grown in the Winona area, with many Winona residents maintaining home gardens. Produce grown in the vicinity of the proposed resource recovery facility may be exposed to emitted chemicals both directly via deposition onto exposed surfaces and indirectly via uptake from soils. Individuals may then be exposed to these contaminants via ingestion of their own produce and commercial produce. The highest exposures are likely to occur among those residents who grow and rely on their own produce as a major food source. Accordingly, potential exposures to individuals who may ingest homegrown produce will be evaluated in this exposure assessment.

Another potential exposure pathway involves the biouptake of contaminants by fish in the Mississippi River. When the chemicals emitted from the proposed facility are deposited onto the river, they will mix into the water. Resident fish may then biologically take up the chemicals, accumulating them in their tissues. Individuals who fish from the Mississippi River in the vicinity of the facility and use their catch as a food source may then ingest these chemicals. Thus exposures due to ingestion of fish caught from the Mississippi River will be evaluated in this risk assessment.

Exposures may also result from ingestion of locally-produced dairy products, especially cow's milk. Chemicals deposited on dairy farms may contaminate feed and subsequently bioaccumulate in the fat present in cow's milk. There are approximately 30,000 dairy cows in Winona County. approximately 750 of which are within a five-mile radius of the two proposed facility sites. There are, according to the Minnesota Department of Agriculture (MDA 1987), approximately twenty commercial milk-producing farms within a five-mile radius of the two facility sites. Milk from these farms, though, is shipped out of Winona for processing, according to the one dairy processor identified in the vicinity of Winona, Ridgeway Foods, Inc. Dairy products (primarily cheese) produced by Ridgeway Foods are conservatively estimated to be composed of no more than five percent local milk (Ridgeway 1987). The milk sold in Winona stores is estimated to be comprised of less than one percent locally-produced milk (Ridgeway 1987, Broadwater 1987). In this risk assessment, therefore, exposures to individuals via ingestion of locally-produced cow's milk will be evaluated.

Indirect exposures may also result from ingestion of locally raised livestock products, such as beef, pork, and chicken. Winona ranchers raise their own feed, and thus chemicals emitted from the facility may be deposited onto animal feed and subsequently bioaccumulate in animal tissues. There are approximately 8,400 head of beef cattle in Winona County (Broadwater 1987). Of these, roughly 600 are within a five-mile radius of the two proposed facility sites, broadly distributed on more than 15 farms. No known farm in the facilities' vicinity has more than 200 head (Pavek 1987). Beef for commercial distribution is transported to large and relatively distant plants, such as in St. Paul, MN and Greenbay, WI, where beef from the entire region is slaughtered

and processed. These plants supply beef to stores in Winona and thus only a small fraction of beef sold in Winona is actually raised locally. Slaughterhouses do exist in the Winona area, however, where local ranchers may process their own beef for home consumption. The highest exposures associated with beef ingestion are likely to occur among ranchers who ingest their own beef, and thus this exposure pathway will also be evaluated.

Only one commercial hog farm is known to exist within a five-mile radius of the two proposed facility sites (Broadwater 1987). Similar to beef, Winona hogs are shipped to distant plants for processing and packaging. Due to the low number of hogs likely to be raised in the facilities' area and considering the large number of hogs handled by commercial processors, the fraction of pork for sale in Winona that is locally-raised will be very low (no more than one percent). Winona farmers may, however, raise hogs for personal consumption and thus exposures via this pathway may occur. The chemical-specific information that would be required to quantify exposures via this pathway is less abundant than that for the cow's milk and beef ingestion pathways. However, the overall methodology applied in quantifying exposures to cow's milk and beef is similar to that for quantifying exposures to pork. Therefore in this risk assessment, exposures occurring as a result of pork ingestion will be qualitatively evaluated by analogy to the results obtained for the two livestock-related pathways likely to be of greatest concern to human health, cow's milk ingestion and beef ingestion.

There are no known commercial poultry operations in the Winona area. However, some Winona farmers are likely to raise chickens for personal consumption. A risk assessment conducted by Stevens and Gerbec (1987) indicates, though, that exposures via ingestion of locally-raised chicken are likely to be significantly lower than those associated with milk or beef ingestion. Thus this exposure pathway is not evaluated in the risk assessment.

As indicated in the Section VIII TWP, hunting of game is a popular activity in the Winona area, and game is therefore an occassional food source. Since information on the types and amounts of game consumed in the Winona area were unavailable, potential human exposures via ingestion of locally caught game will be addressed qualitatively in this report. People exposed by this pathway are dealt with as a special risk group. Potential ecological impacts to Winona game will be addressed in detail in Section XI, TWP.

The last exposure pathway that will be evaluated in the risk assessment involves the ingestion of human breast-milk by an infant. Among the chemicals of concern identified in Section VI.B, TWP, PCDDs/PCDFs and PCBs are known to bioaccumulate in the fat present in human breast-milk. As a result, a mother who is exposed to PCDDs/PCDFs and PCBs may subsequently expose her infant to these chemicals as a result of breast-feeding.

In conclusion, nine exposure pathways were evaluated in detail in this risk assessment. These pathways represent both direct and indirect routes of exposure:

1. Inhalation;
2. Ingestion via inhalation;
3. Ingestion of outdoor soil;
4. Ingestion of indoor dust;
5. Ingestion of fish;
6. Ingestion of cow's milk;
7. Ingestion of beef;
8. Ingestion of home-grown produce; and

9. Ingestion of human breast-milk.

4.2.4.3 EXPOSURES ASSOCIATED WITH EACH SELECTED PATHWAY

4.2.4.3.1. Exposure Assessment Methodology

At MPCA's request, three scenarios were modeled: I) a typical Winona resident; II) a self-sustaining farmer; and III) consumption parameters for scenarios I, II, and III are presented in Tables 2, 3, and 4, respectively. For the first scenario, the typical Winona resident, MPCA provided revised consumption values for locally produced beef, dairy milk, vegetables, and fish. Exposure parameters for the other five pathways (inhalation, ingestion via inhalation, and ingestion of outdoor soil, indoor dust, and human breast-milk) remain unchanged except for the items noted above. Different consumption rates were also provided by MPCA for use in scenario II, the self-sustaining farmer, for ingestion of beef, dairy milk, and vegetables. Exposure via ingestion of human breast-milk was also calculated for scenario II. Estimates of exposure via the other five pathways in scenario II were the same as in scenario I. In scenario III, the avid sport fisherman, MPCA provided different fish consumption rates. Furthermore, for scenario III, in the case of fish consumption only adult ingestion rates differed; child and adolescent exposure estimates were kept the same as in scenario I. Based on these differences, exposure via ingestion of human breast-milk also varied slightly from scenario I.

The consumption rates provided by MPCA were broken down into four categories -- infants, toddles, adoescents, and adults, corresponding (according to MPCA) to ages <1, 1-5, 6-15, and 16+. In order to use these values in the spreadsheet models already prepared by Clement, time weighted averages were interpolated from MPCA's values where necessary to adjust for differences in age categories. For example,, the first age category modeled by Clement for the TWP for many of the ingestion scenarios is 1-8 years of age. A consumption rate for this age period was interpolated from MPCA's values by the following formula:

$$C_{1-8} = \frac{[(C_{1-5})(5 \text{ years})] + [(C_{6-15})(3 \text{ years})]}{8 \text{ years}}$$

where

C_{1-8} = consumption rate during eight year period ages 1-8,
C_{1-5} = consumption rate during toddler period ages 1-5, and
C_{6-15} = consumption rate during adolescent period ages 6-15.

Consumption rates presented in Tables 4.2.4a, 4.2.4b, and 4.2.4c reflect this adjustment for relevent age periods.

In the case of locally grown produce, MPCA provided consumption rates for five groups of vegetables. Consumption of potatoes (and similar tubers) and carrots (and similar tubers) were combined for modeling purposes because they were considered to be in the root crop category. Likewise, consumption of legumes and fruits including tomatoes were combined as they were considered to be in the vine crop category. These changes are also reflected in the values presented in Tables 4.2.4a, 4.2.4b, and 4.2.4c.

MPCA's revised consumption rates were presented as annual averages, and thus, as directed in MPCA's letter,, the frequency of exposure for each pathway affected by the revisions was set at 365 days/year.

The exposure assessment results based on these revisions are shown in Tables 5 through 16 as follows:

Scenario I - Typical Winona resident

Table 4.2.4d. Total body burden (intake), maximum case, Riverview site
Table 4.2.4c. Total body burden (intake), maximum case, Riverbend site
Table 4.2.4f. Total body burden (intake), average case, Riverview site
Table 4.2.4g. Total body burden (intake), average case, Riverbend site

Scenario II - Self-sufficient farmer

Table 4.2.4h. Total body burden (intake), maximum case, Riverview site
Table 4.2.4i. Total body burden (intake), maximum case, Riverbend site
Table 4.2.4j. Total body burden (intake), average case, Riverview site
Table 4.2.4k. Total body burden (intake), average case, Riverbend site

Scenario III - Avid sport fisherman

Table 4.2.4l. Total body burden (intake), maximum case, Riverview site
Table 4.2.4m. Total body burden (intake), maximum case, Riverbend site
Table 4.2.4n. Total body burden (intake), average case, Riverview site
Table 4.2.4o. Total body burden (intake), average case, Riverbend site

Table 4.2.4A

EXPOSURE PARAMETERS USED TO ESTIMATE INTAKES
FOR THE PROPOSED WINONA RESOURCE RECOVERY FACILITY:
TYPICAL WINONA RESIDENT SCENARIO

Exposure Pathway	Age Period (years)	Average Body Weight Over Period (kg) (a)	Exposure Rate Per Exposed Day		Assumed Duration of Exposure (years)	Assumed Frequency of Exposure (days/year)	
			Average Case	Plausible Maximum Case		Average Case	Plausible Maximum Case
Inhalation of Ambient Air (b)	1-4	14	5 m3	5 m3	4	365	365
	5-11	29	10 m3	12 m3	7	365	365
	12-18	57	17 m3	26 m3	7	365	365
	19+	70	14 m3	22 m3	52	365	365
Ingestion via Inhalation of Ambient Air (b)	1-4	14	5 m3	5 m3	4	365	365
	5-11	29	10 m3	12 m3	7	365	365
	12-18	57	17 m3	26 m3	7	365	365
	19+	70	14 m3	22 m3	52	365	365
Incidental Ingestion of Outdoor Soil (c)	1-6	17	100 mg	500 mg	6	92	215
	7-11	30	50 mg	250 mg	5	61	215
	12+	70	50 mg	100 mg	59	61	215
Incidental Ingestion of Household Dust (d)	1-6	17	50 mg	100 mg	6	180	365
Ingestion of Fish (e)	1-5	14	0.1 g	0.1 g	5	365	365
	6-14	37	0.3 g	0.3 g	9	365	365
	15-34	68	0.3 g	0.3 g	20	365	365
	35-74	70	0.3 g	0.3 g	36	365	365
Ingestion of Fresh Produce (e)							
Raw Tomatoes	1-8	20	3.5 g	3.5 g	8	365	365
	9-74	70	5.2 g	5.2 g	62	365	365
Raw Carrots	1-8	20	0.9 g	0.9 g	8	365	365
	9-74	70	1.3 g	1.3 g	62	365	365
Lettuce	1-8	20	5.7 g	5.7 g	8	365	365
	9-74	70	8.8 g	8.8 g	62	365	365
Ingestion of Beef (e)	1-8	20	1.6 g	1.6 g	8	365	365
	9-74	70	3.4 g	3.4 g	62	365	365
Ingestion of Dairy Milk (e)	1-8	20	0.38 l	0.38 l	8	365	365
	9-18	50	0.35 l	0.35 l	10	365	365
	19-74	70	0.35 l	0.35 l	52	365	365
Ingestion of Human Breast-Milk (f)	0-1	9	0.8 kg	0.8 kg	1	365	365

(a) Based on USEPA (1985a).
(b) Based on NRCP (1984) and USEPA (1985a). Assumes resting ventilization rates.
(c) Based on information provided in Appendix E.
(d) Based on Hawley (1985).
(e) Provided by MPCA (1988).
(f) Based on USEPA (1985a) and Butte et al. (1984).

Table 4.2.4B

EXPOSURE PARAMETERS USED TO ESTIMATE INTAKES
FOR THE PROPOSED WINONA RESOURCE RECOVERY FACILITY:
SELF-SUSTAINING FARMER SCENARIO

Exposure Pathway	Age Period (years)	Average Body Weight Over Period (kg) (a)	Exposure Rate Per Exposed Day		Assumed Duration of Exposure (years)	Assumed Frequency of Exposure (days/year)	
			Average Case	Plausible Maximum Case		Average Case	Plausible Maximum Case
Inhalation of Ambient Air (b)	1-4	14	5 m3	5 m3	4	365	365
	5-11	29	10 m3	12 m3	7	365	365
	12-18	57	17 m3	26 m3	7	365	365
	19+	70	14 m3	22 m3	52	365	365
Ingestion via Inhalation of Ambient Air (b)	1-4	14	5 m3	5 m3	4	365	365
	5-11	29	10 m3	12 m3	7	365	365
	12-18	57	17 m3	26 m3	7	365	365
	19+	70	14 m3	22 m3	52	365	365
Incidental Ingestion of Outdoor Soil (c)	1-6	17	100 mg	500 mg	6	92	215
	7-11	30	50 mg	250 mg	5	61	215
	12+	70	50 mg	100 mg	59	61	215
Incidental Ingestion of Household Dust (d)	1-6	17	50 mg	100 mg	6	180	365
Ingestion of Fish (e)	1-5	14	0.1 g	0.1 g	5	365	365
	6-14	37	0.3 g	0.3 g	9	365	365
	15-34	68	0.3 g	0.3 g	20	365	365
	35-74	70	0.3 g	0.3 g	36	365	365
Ingestion of Fresh Produce (e)							
Raw Tomatoes	1-8	20	33 g	33 g	8	365	365
	9-74	70	62 g	62 g	62	365	365
Raw Carrots	1-8	20	98 g	98 g	8	365	365
	9-74	70	206 g	206 g	62	365	365
Lettuce	1-8	20	25 g	25 g	8	365	365
	9-74	70	50 g	50 g	62	365	365
Ingestion of Beef (e)	1-8	20	53 g	53 g	8	365	365
	9-74	70	125 g	125 g	62	365	365
Ingestion of Dairy Milk (e)	1-8	20	0.63 l	0.63 l	8	365	365
	9-18	50	1.00 l	1.00 l	10	365	365
	19-74	70	1.00 l	1.00 l	52	365	365
Ingestion of Human Breast-Milk (f)	0-1	9	0.8 kg	0.8 kg	1	365	365

(a) Based on USEPA (1985a).
(b) Based on NRCP (1984) and USEPA (1985a). Assumes resting ventilization rates.
(c) Based on information provided in Appendix E.
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6. Ingestion of cow's milk;
7. Ingestion of beef;
8. Ingestion of home-grown produce; and
9. Ingestion of human breast-milk.

For each pathway, potential chemical concentrations in the environmental media of concern were estimated for an average and a maximum case. Intakes were then calculated for average case and maximum case exposure scenarios based on site-specific information describing the potential magnitude, frequency and duration of exposure.

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Contaminants emitted from the proposed Winona facility will be distributed widely in the air and may be deposited at varying rates onto a wide area of land and water. As a result, residents may be exposed to these contaminants in varying quantities and proportions via a wide variety of exposure routes. These routes include inhalation of air; ingestion of outdoor soil, produce, fish, milk, beef, and indoor dust; and direct dermal contact with outdoor soil. The frequency, extent, and duration of exposure for any individual person will depend on many factors, including location of residence, age, sex, and activity patterns.

The complexity of exposure to environmental contaminants requires a series of simplifying steps in the process of exposure assessment. This sectionis based on ICF/Clement TWP and follows a general approach to exposure assessment which has been developed by specialists in this field and has been codified in general terms by the U.S. Environmental Protection Agency (USEPA 1986a,b,c). In this general approach, exposure assessment is conducted in two general steps. The first step (Section IX-C,TWP) is a pathway analysis in which the exposure pathways most likely to be significant are extracted from a list of all possible pathways. This screening process is necessary to focus the assessment on the most significant exposure pathways and is justified by experience, which shows that in most circumstances exposure via a few exposure pathways to one or a few chemicals dominates estimates of potential risks.

The second step(Section IX.D,TWP) is a detailed assessment of exposures. For each exposure pathway, an exposure scenario is developed based on assumptions about the environmental behavior and transport of the contaminants, and the extent, frequency, and duration of exposures. These factors are used to predict potential intakes of the chemicals of concern for both an average and a maximum exposure case.

4.2.4.2. IDENTIFICATION OF EXPOSURE PATHWAYS

absorption will not be quantitatively evaluated in this risk assessment.

The fourth exposure pathway to be evaluated in the risk assessment involves the incidental ingestion of household dust. Ingestion of dust inside the home is considered because outdoor airborne particulate matter or tracking of surface soil contribute to a large percentage of the dust inside homes. Children playing or crawling on all fours inside the home collect dust on their hands. This dust may then be ingested when the children put their hands in their mouths. Thus potential exposures to children as a result of incidental dust ingestion in the home will be evaluated in this risk assessment.

As described in the TWP, (Section VIII), a wide variety of crops are grown in the Winona area, with many Winona residents maintaining home gardens. Produce grown in the vicinity of the proposed resource recovery facility may be exposed to emitted chemicals both directly via deposition onto exposed surfaces and indirectly via uptake from soils. Individuals may then be exposed to these contaminants via ingestion of their own produce and commercial produce. The highest exposures are likely to occur among those residents who grow and rely on their own produce as a major food source. Accordingly, potential exposures to individuals who may ingest homegrown produce will be evaluated in this exposure assessment.

Another potential exposure pathway involves the biouptake of contaminants by fish in the Mississippi River. When the chemicals emitted from the proposed facility are deposited onto the river, they will mix into the water. Resident fish may then biologically take up the chemicals, accumulating them in their tissues. Individuals who fish from the Mississippi River in the vicinity of the facility and use their catch as a food source may then ingest these chemicals. Thus exposures due to ingestion of fish caught from the Mississippi River will be evaluated in this risk assessment.

Exposures may also result from ingestion of locally-produced dairy products, especially cow's milk. Chemicals deposited on dairy farms may contaminate feed and subsequently bioaccumulate in the fat present in cow's milk. There are approximately 30,000 dairy cows in Winona County. approximately 750 of which are within a five-mile radius of the two proposed facility sites. There are, according to the Minnesota Department of Agriculture (MDA 1987), approximately twenty commercial milk-producing farms within a five-mile radius of the two facility sites. Milk from these farms, though, is shipped out of Winona for processing, according to the one dairy processor identified in the vicinity of Winona, Ridgeway Foods, Inc. Dairy products (primarily cheese) produced by Ridgeway Foods are conservatively estimated to be composed of no more than five percent local milk (Ridgeway 1987). The milk sold in Winona stores is estimated to be comprised of less than one percent locally-produced milk (Ridgeway 1987, Broadwater 1987). In this risk assessment, therefore, exposures to individuals via ingestion of locally-produced cow's milk will be evaluated.

Indirect exposures may also result from ingestion of locally raised livestock products, such as beef, pork, and chicken. Winona ranchers raise their own feed, and thus chemicals emitted from the facility may be deposited onto animal feed and subsequently bioaccumulate in animal tissues. There are approximately 8,400 head of beef cattle in Winona County (Broadwater 1987). Of these, roughly 600 are within a five-mile radius of the two proposed facility sites, broadly distributed on more than 15 farms. No known farm in the facilities' vicinity has more than 200 head (Pavek 1987). Beef for commercial distribution is transported to large and relatively distant plants, such as in St. Paul, MN and Greenbay, WI, where beef from the entire region is slaughtered

9. Ingestion of human breast-milk.

4.2.4.3 EXPOSURES ASSOCIATED WITH EACH SELECTED PATHWAY

4.2.4.3.1. Exposure Assessment Methodology

At MPCA's request, three scenarios were modeled: I) a typical Winona resident; II) a self-sustaining farmer; and III) consumption parameters for scenarios I, II, and III are presented in Tables 2, 3, and 4, respectively. For the first scenario, the typical Winona resident, MPCA provided revised consumption values for locally produced beef, dairy milk, vegetables, and fish. Exposure parameters for the other five pathways (inhalation, ingestion via inhalation, and ingestion of outdoor soil, indoor dust, and human breast-milk) remain unchanged except for the items noted above. Different consumption rates were also provided by MPCA for use in scenario II, the self-sustaining farmer, for ingestion of beef, dairy milk, and vegetables. Exposure via ingestion of human breast-milk was also calculated for scenario II. Estimates of exposure via the other five pathways in scenario II were the same as in scenario I. In scenario III, the avid sport fisherman, MPCA provided different fish consumption rates. Furthermore, for scenario III, in the case of fish consumption only adult ingestion rates differed; child and adolescent exposure estimates were kept the same as in scenario I. Based on these differences, exposure via ingestion of human breast-milk also varied slightly from scenario I.

The consumption rates provided by MPCA were broken down into four categories -- infants, toddles, adoescents, and adults, corresponding (according to MPCA) to ages <1, 1-5, 6-15, and 16+. In order to use these values in the spreadsheet models already prepared by Clement, time weighted averages were interpolated from MPCA's values where necessary to adjust for differences in age categories. For example,, the first age category modeled by Clement for the TWP for many of the ingestion scenarios is 1-8 years of age. A consumption rate for this age period was interpolated from MPCA's values by the following formula:

$$C_{1-8} = \frac{[(C_{1-5})(5 \text{ years})] + [(C_{6-15})(3 \text{ years})]}{8 \text{ years}}$$

where

C_{1-8} = consumption rate during eight year period ages 1-8,
C_{1-5} = consumption rate during toddler period ages 1-5, and
C_{6-15} = consumption rate during adolescent period ages 6-15.

Consumption rates presented in Tables 4.2.4a, 4.2.4b, and 4.2.4c reflect this adjustment for relevent age periods.

In the case of locally grown produce, MPCA provided consumption rates for five groups of vegetables. Consumption of potatoes (and similar tubers) and carrots (and similar tubers) were combined for modeling purposes because they were considered to be in the root crop category. Likewise, consumption of legumes and fruits including tomatoes were combined as they were considered to be in the vine crop category. These changes are also reflected in the values presented in Tables 4.2.4a, 4.2.4b, and 4.2.4c.

Table 4.2.4A

EXPOSURE PARAMETERS USED TO ESTIMATE INTAKES
FOR THE PROPOSED WINONA RESOURCE RECOVERY FACILITY:
TYPICAL WINONA RESIDENT SCENARIO

Exposure Pathway	Age Period (years)	Average Body Weight Over Period (kg) (a)	Exposure Rate Per Exposed Day		Assumed Duration of Exposure (years)	Assumed Frequency of Exposure (days/year)	
			Average Case	Plausible Maximum Case		Average Case	Plausible Maximum Case
Inhalation of Ambient Air (b)	1-4	14	5 m3	5 m3	4	365	365
	5-11	29	10 m3	12 m3	7	365	365
	12-18	57	17 m3	26 m3	7	365	365
	19+	70	14 m3	22 m3	52	365	365
Ingestion via Inhalation of Ambient Air (b)	1-4	14	5 m3	5 m3	4	365	365
	5-11	29	10 m3	12 m3	7	365	365
	12-18	57	17 m3	26 m3	7	365	365
	19+	70	14 m3	22 m3	52	365	365
Incidental Ingestion of Outdoor Soil (c)	1-6	17	100 mg	500 mg	6	92	215
	7-11	30	50 mg	250 mg	5	61	215
	12+	70	50 mg	100 mg	59	61	215
Incidental Ingestion of Household Dust (d)	1-6	17	50 mg	100 mg	6	180	365
Ingestion of Fish (e)	1-5	14	0.1 g	0.1 g	5	365	365
	6-14	37	0.3 g	0.3 g	9	365	365
	15-34	68	0.3 g	0.3 g	20	365	365
	35-74	70	0.3 g	0.3 g	36	365	365
Ingestion of Fresh Produce (e)							
Raw Tomatoes	1-8	20	3.5 g	3.5 g	8	365	365
	9-74	70	5.2 g	5.2 g	62	365	365
Raw Carrots	1-8	20	0.9 g	0.9 g	8	365	365
	9-74	70	1.3 g	1.3 g	62	365	365
Lettuce	1-8	20	5.7 g	5.7 g	8	365	365
	9-74	70	8.8 g	8.8 g	62	365	365
Ingestion of Beef (e)	1-8	20	1.6 g	1.6 g	8	365	365
	9-74	70	3.4 g	3.4 g	62	365	365
Ingestion of Dairy Milk (e)	1-8	20	0.38 l	0.38 l	8	365	365
	9-18	50	0.35 l	0.35 l	10	365	365
	19-74	70	0.35 l	0.35 l	52	365	365
Ingestion of Human Breast-Milk (f)	0-1	9	0.8 kg	0.8 kg	1	365	365

(a) Based on USEPA (1985a).
(b) Based on NRCP (1984) and USEPA (1985a). Assumes resting ventilization rates.
(c) Based on information provided in Appendix E.
(d) Based on Hawley (1985).
(e) Provided by MPCA (1988).
(f) Based on USEPA (1985a) and Butte et al. (1984).

Table 4.2.4C

EXPOSURE PARAMETERS USED TO ESTIMATE INTAKES
FOR THE PROPOSED WINONA RESOURCE RECOVERY FACILITY:
AVID SPORT FISHERMAN SCENARIO

Exposure Pathway	Age Period (years)	Average Body Weight Over Period (kg) (a)	Exposure Rate Per Exposed Day		Assumed Duration of Exposure (years)	Assumed Frequency of Exposure (days/year)	
			Average Case	Plausible Maximum Case		Average Case	Plausible Maximum Case
Inhalation of Ambient Air (b)	1-4	14	5 m3	5 m3	4	365	365
	5-11	29	10 m3	12 m3	7	365	365
	12-18	57	17 m3	26 m3	7	365	365
	19+	70	14 m3	22 m3	52	365	365
Ingestion via Inhalation of Ambient Air (b)	1-4	14	5 m3	5 m3	4	365	365
	5-11	29	10 m3	12 m3	7	365	365
	12-18	57	17 m3	26 m3	7	365	365
	19+	70	14 m3	22 m3	52	365	365
Incidental Ingestion of Outdoor Soil (c)	1-6	17	100 mg	500 mg	6	92	215
	7-11	30	50 mg	250 mg	5	61	215
	12+	70	50 mg	100 mg	59	61	215
Incidental Ingestion of Household Dust (d)	1-6	17	50 mg	100 mg	6	180	365
Ingestion of Fish (e)	1-5	14	0.1 g	0.1 g	5	365	365
	6-14	37	0.3 g	0.3 g	9	365	365
	15-34	68	30 g	30 g	20	365	365
	35-74	70	30 g	30 g	36	365	365
Ingestion of Fresh Produce (e)							
Raw Tomatoes	1-8	20	3.5 g	3.5 g	8	365	365
	9-74	70	5.2 g	5.2 g	62	365	365
Raw Carrots	1-8	20	0.9 g	0.9 g	8	365	365
	9-74	70	1.3 g	1.3 g	62	365	365
Lettuce	1-8	20	5.7 g	5.7 g	8	365	365
	9-74	70	8.8 g	8.8 g	62	365	365
Ingestion of Beef (e)	1-8	20	1.6 g	1.6 g	8	365	365
	9-74	70	3.4 g	3.4 g	62	365	365
Ingestion of Dairy Milk (e)	1-8	20	0.38 l	0.38 l	8	365	365
	9-18	50	0.35 l	0.35 l	10	365	365
	19-74	70	0.35 l	0.35 l	52	365	365
Ingestion of Human Breast-Milk (f)	0-1	9	0.8 kg	0.8 kg	1	365	365

(a) Based on USEPA (1985a).
(b) Based on NRCP (1984) and USEPA (1985a). Assumes resting ventilization rates.
(c) Based on information provided in Appendix E.
(d) Based on Hawley (1985).
(e) Provided by MPCA (1988).
(f) Based on USEPA (1985a) and Butte et al. (1984).

Table 4.2.4D

ESTIMATED MAXIMUM BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	3.52E-05	NA	NA	NA	NA	NA	NA	NA	NA	4E-05	NA
Arsenic and compounds	5.85E-10	9.38E-11	1.13E-09	5.69E-09	2.28E-11	1.95E-12	9.28E-15	1.59E-09	NA	6E-10	9E-09
Beryllium and compounds	6.61E-12	NA	NA	NA	NA	NA	NA	NA	NA	7E-12	NA
Cadmium and compounds	2.88E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	5.12E-10	1.64E-09	9.23E-09	4.66E-08	3.72E-09	5.31E-09	6.05E-13	1.75E-08	NA	5E-10	8E-08
Chlorophenols (treated as 2,4,6-trichlorophenol)	4.29E-10	1.38E-09	7.74E-09	3.91E-08	1.56E-10	1.56E-10	4.62E-12	2.16E-08	NA	4E-10	7E-08
Chromium (VI) and compounds	3.74E-08	NA	NA	NA	NA	NA	NA	NA	NA	4E-08	NA
Nickel and compounds	2.76E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Polychlorinated Biphenyls	1.59E-11	3.94E-12	4.74E-11	2.39E-10	1.91E-11	2.73E-11	6.28E-12	7.67E-11	3.51E-10	2E-11	8E-10
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	2.38E-12	3.44E-12	1.93E-11	9.75E-11	3.73E-11	4.36E-11	2.20E-12	2.97E-11	1.75E-10	2E-12	4E-10
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	2.03E-08	5.06E-09	6.08E-08	3.07E-07	NA	NA	7.42E-11	9.84E-08	NA	2E-08	5E-07

ESTIMATED MAXIMUM BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	8.24E-08	1.28E-09	1.79E-07	7.74E-08	1.55E-10	4.41E-11	3.27E-13	2.87E-08	NA	8E-08	3E-07
Beryllium and compounds	NA	1.02E-13	1.43E-11	6.20E-12	1.24E-14	3.18E-17	4.97E-16	1.77E-12	NA	NA	2E-11
Cadmium and compounds	NA	1.75E-08	2.46E-06	1.06E-06	1.17E-09	6.09E-09	1.72E-10	6.61E-07	NA	NA	4E-06
Chromium (VI) and compounds	NA	6.20E-11	8.70E-09	3.76E-09	4.14E-11	3.22E-11	5.04E-13	1.55E-09	NA	NA	1E-08
Lead and compounds	6.62E-07	5.11E-08	3.35E-06	1.45E-06	8.68E-10	2.06E-09	3.21E-10	4.32E-07	NA	7E-07	5E-06
Mercury (inorganic) and compounds	4.46E-07	1.58E-07	1.03E-05	4.47E-06	2.41E-07	1.15E-08	2.40E-08	1.35E-06	NA	4E-07	2E-05
Nickel and compounds	NA	7.32E-09	1.03E-06	4.45E-07	5.33E-09	2.53E-09	1.20E-12	1.69E-07	NA	NA	2E-06

NA = Not Applicable.

Table 4.2.4E

ESTIMATED MAXIMUM BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	5.18E-05	NA	NA	NA	NA	NA	NA	NA	NA	5E-05	NA
Arsenic and compounds	8.61E-10	1.38E-10	1.66E-09	8.37E-09	5.69E-11	4.87E-12	9.82E-15	2.10E-09	NA	9E-10	1E-08
Beryllium and compounds	9.71E-12	NA	NA	NA	NA	NA	NA	NA	NA	1E-11	NA
Cadmium and compounds	4.24E-07	NA	NA	NA	NA	NA	NA	NA	NA	4E-07	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	7.52E-10	2.42E-09	1.36E-08	6.85E-08	9.31E-09	1.33E-08	6.41E-13	2.30E-08	NA	8E-10	1E-07
Chlorophenols (treated as 2,4,6-trichlorophenol)	6.31E-10	2.03E-09	1.14E-08	5.75E-08	3.91E-10	3.90E-10	4.89E-12	2.84E-08	NA	6E-10	1E-07
Chromium (VI) and compounds	5.50E-08	NA	NA	NA	NA	NA	NA	NA	NA	5E-08	NA
Nickel and compounds	4.06E-07	NA	NA	NA	NA	NA	NA	NA	NA	4E-07	NA
Polychlorinated Biphenyls	2.33E-11	5.80E-12	6.97E-11	3.52E-10	4.78E-11	6.82E-11	6.65E-12	1.01E-10	5.35E-10	2E-11	1E-09
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	3.50E-12	5.06E-12	2.84E-11	1.43E-10	9.33E-11	1.09E-10	2.33E-12	3.91E-11	3.00E-10	3E-12	7E-10
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	2.99E-08	7.44E-09	8.95E-08	4.52E-07	NA	NA	7.85E-11	1.30E-07	NA	3E-08	7E-07

ESTIMATED MAXIMUM BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	1.21E-07	1.88E-09	2.63E-07	1.14E-07	3.87E-10	1.10E-10	3.46E-13	3.77E-08	NA	1E-07	4E-07
Beryllium and compounds	NA	1.50E-13	2.11E-11	9.12E-12	3.10E-14	7.96E-17	5.27E-16	2.32E-12	NA	NA	3E-11
Cadmium and compounds	NA	2.57E-08	3.61E-06	1.56E-06	2.94E-09	1.52E-08	1.82E-10	8.69E-07	NA	NA	6E-06
Chromium (VI) and compounds	NA	9.12E-11	1.28E-08	5.53E-09	1.03E-10	8.04E-11	5.33E-13	2.04E-09	NA	NA	2E-08
Lead and compounds	9.74E-07	7.52E-08	4.92E-06	2.13E-06	2.17E-09	5.16E-09	3.40E-10	5.68E-07	NA	1E-06	8E-06
Mercury (inorganic) and compounds	6.56E-07	2.32E-07	1.52E-05	6.57E-06	6.03E-07	2.87E-08	2.54E-08	1.78E-06	NA	7E-07	2E-05
Nickel and compounds	NA	1.08E-08	1.51E-06	6.54E-07	1.33E-08	6.33E-09	1.27E-12	2.22E-07	NA	NA	2E-06

NA = Not Applicable.

Table 4.2.4F

ESTIMATED AVERAGE BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	5.68E-06	NA	NA	NA	NA	NA	NA	NA	NA	6E-06	NA
Arsenic and compounds	9.43E-11	1.51E-11	6.54E-11	1.30E-10	8.83E-12	7.68E-13	1.16E-15	1.64E-10	NA	9E-11	4E-10
Beryllium and compounds	1.06E-12	NA	NA	NA	NA	NA	NA	NA	NA	1E-12	NA
Cadmium and compounds	4.64E-08	NA	NA	NA	NA	NA	NA	NA	NA	5E-08	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	8.24E-11	2.65E-10	5.35E-10	1.07E-09	1.45E-09	2.09E-09	1.54E-13	2.27E-09	NA	8E-11	8E-09
Chlorophenols (treated as 2,4,6-trichlorophenol)	6.91E-11	2.22E-10	4.49E-10	8.95E-10	6.06E-11	6.15E-11	1.18E-12	3.36E-09	NA	7E-11	5E-09
Chromium (VI) and compounds	6.02E-09	NA	NA	NA	NA	NA	NA	NA	NA	6E-09	NA
Nickel and compounds	4.45E-08	NA	NA	NA	NA	NA	NA	NA	NA	4E-08	NA
Polychlorinated Biphenyls	2.55E-12	2.12E-13	9.17E-13	1.83E-12	2.48E-12	3.59E-12	1.23E-12	4.35E-12	1.76E-12	3E-12	2E-11
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	3.83E-13	1.85E-13	3.74E-13	7.44E-13	4.72E-12	5.68E-12	3.18E-13	1.28E-12	1.35E-12	4E-13	1E-11
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	3.28E-09	2.72E-10	1.18E-09	2.34E-09	NA	NA	1.22E-11	5.58E-09	NA	3E-09	9E-09

ESTIMATED AVERAGE BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	1.33E-08	2.05E-10	1.04E-08	1.77E-09	6.01E-11	1.74E-11	4.09E-14	3.70E-09	NA	1E-08	2E-08
Beryllium and compounds	NA	1.65E-14	8.32E-13	1.42E-13	4.81E-15	1.26E-17	1.25E-16	1.85E-13	NA	NA	1E-12
Cadmium and compounds	NA	2.82E-09	1.43E-07	2.43E-08	4.56E-10	2.40E-09	1.17E-11	1.07E-07	NA	NA	3E-07
Chromium (VI) and compounds	NA	9.98E-12	5.05E-10	8.61E-11	1.61E-11	1.27E-11	2.52E-14	2.14E-10	NA	NA	8E-10
Lead and compounds	1.07E-07	8.23E-09	1.94E-07	3.31E-08	3.37E-10	8.14E-10	1.99E-11	4.73E-08	NA	1E-07	3E-07
Mercury (inorganic) and compounds	7.19E-08	2.54E-08	5.99E-07	1.02E-07	9.36E-08	4.52E-09	2.08E-09	1.50E-07	NA	7E-08	1E-06
Nickel and compounds	NA	1.18E-09	5.96E-08	1.02E-08	2.07E-09	1.00E-09	1.15E-13	2.22E-08	NA	NA	1E-07

Table 4.2.4G

ESTIMATED AVERAGE BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	8.04E-06	NA	NA	NA	NA	NA	NA	NA	NA	8E-06	NA
Arsenic and compounds	1.34E-10	2.14E-11	9.27E-11	1.85E-10	1.25E-11	1.09E-12	1.07E-15	2.33E-10	NA	1E-10	5E-10
Beryllium and compounds	1.51E-12	NA	NA	NA	NA	NA	NA	NA	NA	2E-12	NA
Cadmium and compounds	6.57E-08	NA	NA	NA	NA	NA	NA	NA	NA	7E-08	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	1.17E-10	3.75E-10	7.58E-10	1.51E-09	2.05E-09	2.97E-09	1.42E-13	3.22E-09	NA	1E-10	1E-08
Chlorophenols (treated as 2,4,6-trichlorophenol)	9.79E-11	3.15E-10	6.36E-10	1.27E-09	8.59E-11	8.71E-11	1.09E-12	4.77E-09	NA	1E-10	7E-09
Chromium (VI) and compounds	8.53E-09	NA	NA	NA	NA	NA	NA	NA	NA	9E-09	NA
Nickel and compounds	6.30E-08	NA	NA	NA	NA	NA	NA	NA	NA	6E-08	NA
Polychlorinated Biphenyls	3.62E-12	3.00E-13	1.30E-12	2.59E-12	3.51E-12	5.08E-12	1.13E-12	6.17E-12	2.44E-12	4E-12	2E-11
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	5.43E-13	2.62E-13	5.29E-13	1.05E-12	6.69E-12	8.04E-12	2.94E-13	1.81E-12	1.90E-12	5E-13	2E-11
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	4.64E-09	3.85E-10	1.67E-09	3.32E-09	NA	NA	1.12E-11	7.91E-09	NA	5E-09	1E-08

ESTIMATED AVERAGE BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	1.88E-08	2.91E-10	1.47E-08	2.51E-09	8.51E-11	2.47E-11	3.78E-14	5.25E-09	NA	2E-08	2E-08
Beryllium and compounds	NA	2.33E-14	1.18E-12	2.01E-13	6.82E-15	1.78E-17	1.16E-16	2.62E-13	NA	NA	2E-12
Cadmium and compounds	NA	3.99E-09	2.02E-07	3.45E-08	6.46E-10	3.40E-09	1.08E-11	1.52E-07	NA	NA	4E-07
Chromium (VI) and compounds	NA	1.41E-11	7.15E-10	1.22E-10	2.27E-11	1.80E-11	2.32E-14	3.03E-10	NA	NA	1E-09
Lead and compounds	1.51E-07	1.17E-08	2.75E-07	4.69E-08	4.77E-10	1.15E-09	1.83E-11	6.71E-08	NA	2E-07	4E-07
Mercury (inorganic) and compounds	1.02E-07	3.60E-08	8.49E-07	1.45E-07	1.33E-07	6.41E-09	1.92E-09	2.13E-07	NA	1E-07	1E-06
Nickel and compounds	NA	1.67E-09	8.45E-08	1.44E-08	2.93E-09	1.42E-09	1.06E-13	3.14E-08	NA	NA	1E-07

Table 4.2.4H

ESTIMATED MAXIMUM BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	3.52E-05	NA	NA	NA	NA	NA	NA	NA	NA	4E-05	NA
Arsenic and compounds	5.85E-10	9.38E-11	1.13E-09	5.69E-09	8.22E-10	4.83E-10	9.28E-15	1.04E-08	NA	6E-10	2E-08
Beryllium and compounds	6.61E-12	NA	NA	NA	NA	NA	NA	NA	NA	7E-12	NA
Cadmium and compounds	2.88E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	5.12E-10	1.64E-09	9.23E-09	4.66E-08	1.35E-07	1.32E-06	6.05E-13	7.31E-07	NA	5E-10	2E-06
Chlorophenols (treated as 2,4,6-trichlorophenol)	4.29E-10	1.38E-09	7.74E-09	3.91E-08	5.64E-09	3.87E-08	4.62E-12	1.63E-06	NA	4E-10	2E-06
Chromium (VI) and compounds	3.74E-08	NA	NA	NA	NA	NA	NA	NA	NA	4E-08	NA
Nickel and compounds	2.76E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Polychlorinated Biphenyls	1.59E-11	3.94E-12	4.74E-11	2.39E-10	6.91E-10	6.76E-09	6.28E-12	1.86E-09	5.09E-09	2E-11	1E-08
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	2.38E-12	3.44E-12	1.93E-11	9.75E-11	1.35E-09	1.08E-08	2.20E-12	5.33E-10	6.75E-09	2E-12	2E-08
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	2.03E-08	5.06E-09	6.08E-08	3.07E-07	NA	NA	7.42E-11	2.38E-06	NA	2E-08	3E-06

ESTIMATED MAXIMUM BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	8.24E-08	1.28E-09	1.79E-07	7.74E-08	5.59E-09	1.09E-08	3.27E-13	1.16E-06	NA	8E-08	1E-06
Beryllium and compounds	NA	1.02E-13	1.43E-11	6.20E-12	4.48E-13	7.89E-15	4.97E-16	1.54E-11	NA	NA	4E-11
Cadmium and compounds	NA	1.75E-08	2.46E-06	1.06E-06	4.24E-08	1.51E-06	1.72E-10	4.53E-05	NA	NA	5E-05
Chromium (VI) and compounds	NA	6.20E-11	8.70E-09	3.76E-09	1.49E-09	7.97E-09	5.04E-13	8.02E-08	NA	NA	1E-07
Lead and compounds	6.62E-07	5.11E-08	3.35E-06	1.45E-06	3.14E-08	5.11E-07	3.21E-10	6.50E-06	NA	7E-07	1E-05
Mercury (inorganic) and compounds	4.46E-07	1.58E-07	1.03E-05	4.47E-06	8.71E-06	2.84E-06	2.40E-08	2.30E-05	NA	4E-07	5E-05
Nickel and compounds	NA	7.32E-09	1.03E-06	4.45E-07	1.93E-07	6.28E-07	1.20E-12	7.31E-06	NA	NA	1E-05

NA = Not Applicable.

Table 4.2.4I

ESTIMATED MAXIMUM BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	5.18E-05	NA	NA	NA	NA	NA	NA	NA	NA	5E-05	NA
Arsenic and compounds	8.61E-10	1.38E-10	1.66E-09	8.37E-09	2.06E-09	1.21E-09	9.82E-15	1.37E-08	NA	9E-10	3E-08
Beryllium and compounds	9.71E-12	NA	NA	NA	NA	NA	NA	NA	NA	1E-11	NA
Cadmium and compounds	4.24E-07	NA	NA	NA	NA	NA	NA	NA	NA	4E-07	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	7.52E-10	2.42E-09	1.36E-08	6.85E-08	3.36E-07	3.29E-06	6.41E-13	9.62E-07	NA	8E-10	5E-06
Chlorophenols (treated as 2,4,6-trichlorophenol)	6.31E-10	2.03E-09	1.14E-08	5.75E-08	1.41E-08	9.66E-08	4.89E-12	2.14E-06	NA	6E-10	2E-06
Chromium (VI) and compounds	5.50E-08	NA	NA	NA	NA	NA	NA	NA	NA	5E-08	NA
Nickel and compounds	4.06E-07	NA	NA	NA	NA	NA	NA	NA	NA	4E-07	NA
Polychlorinated Biphenyls	2.33E-11	5.80E-12	6.97E-11	3.52E-10	1.73E-09	1.69E-08	6.65E-12	2.45E-09	1.14E-08	2E-11	3E-08
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	3.50E-12	5.06E-12	2.84E-11	1.43E-10	3.37E-09	2.70E-08	2.33E-12	7.01E-10	1.65E-08	3E-12	5E-08
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	2.99E-08	7.44E-09	8.95E-08	4.52E-07	NA	NA	7.85E-11	3.14E-06	NA	3E-08	4E-06

ESTIMATED MAXIMUM BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	1.21E-07	1.88E-09	2.63E-07	1.14E-07	1.40E-08	2.73E-08	3.46E-13	1.53E-06	NA	1E-07	2E-06
Beryllium and compounds	NA	1.50E-13	2.11E-11	9.12E-12	1.12E-12	1.97E-14	5.27E-16	2.03E-11	NA	NA	5E-11
Cadmium and compounds	NA	2.57E-08	3.61E-06	1.56E-06	1.06E-07	3.77E-06	1.82E-10	5.96E-05	NA	NA	7E-05
Chromium (VI) and compounds	NA	9.12E-11	1.28E-08	5.53E-09	3.74E-09	1.99E-08	5.33E-13	1.06E-07	NA	NA	1E-07
Lead and compounds	9.74E-07	7.52E-08	4.92E-06	2.13E-06	7.84E-08	1.28E-06	3.40E-10	8.55E-06	NA	1E-06	2E-05
Mercury (inorganic) and compounds	6.56E-07	2.32E-07	1.52E-05	6.57E-06	2.18E-05	7.10E-06	2.54E-08	3.03E-05	NA	7E-07	8E-05
Nickel and compounds	NA	1.08E-08	1.51E-06	6.54E-07	4.81E-07	1.57E-06	1.27E-12	9.61E-06	NA	NA	1E-05

NA = Not Applicable.

Table 4.2.4J

ESTIMATED AVERAGE BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	5.68E-06	NA	NA	NA	NA	NA	NA	NA	NA	6E-06	NA
Arsenic and compounds	9.43E-11	1.51E-11	6.54E-11	1.30E-10	3.19E-10	1.90E-10	1.16E-15	1.27E-09	NA	9E-11	2E-09
Beryllium and compounds	1.06E-12	NA	NA	NA	NA	NA	NA	NA	NA	1E-12	NA
Cadmium and compounds	4.64E-08	NA	NA	NA	NA	NA	NA	NA	NA	5E-08	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	8.24E-11	2.65E-10	5.35E-10	1.07E-09	5.22E-08	5.19E-07	1.54E-13	1.46E-07	NA	8E-11	7E-07
Chlorophenols (treated as 2,4,6-trichlorophenol)	6.91E-11	2.22E-10	4.49E-10	8.95E-10	2.19E-09	1.52E-08	1.18E-12	3.36E-07	NA	7E-11	4E-07
Chromium (VI) and compounds	6.02E-09	NA	NA	NA	NA	NA	NA	NA	NA	6E-09	NA
Nickel and compounds	4.45E-08	NA	NA	NA	NA	NA	NA	NA	NA	4E-08	NA
Polychlorinated Biphenyls	2.55E-12	2.12E-13	9.17E-13	1.83E-12	8.94E-11	8.89E-10	1.23E-12	3.18E-10	1.16E-10	3E-12	1E-09
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	3.83E-13	1.85E-13	3.74E-13	7.44E-13	1.70E-10	1.41E-09	3.18E-13	5.72E-11	1.48E-10	4E-13	2E-09
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	3.28E-09	2.72E-10	1.18E-09	2.34E-09	NA	NA	1.22E-11	4.08E-07	NA	3E-09	4E-07

ESTIMATED AVERAGE BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	1.33E-08	2.05E-10	1.04E-08	1.77E-09	2.17E-09	4.32E-09	4.09E-14	2.33E-07	NA	1E-08	3E-07
Beryllium and compounds	NA	1.65E-14	8.32E-13	1.42E-13	1.74E-13	3.11E-15	1.25E-16	2.25E-12	NA	NA	3E-12
Cadmium and compounds	NA	2.82E-09	1.43E-07	2.43E-08	1.65E-08	5.96E-07	1.17E-11	9.36E-06	NA	NA	1E-05
Chromium (VI) and compounds	NA	9.98E-12	5.05E-10	8.61E-11	5.80E-10	3.15E-09	2.52E-14	1.63E-08	NA	NA	2E-08
Lead and compounds	1.07E-07	8.23E-09	1.94E-07	3.31E-08	1.22E-08	2.02E-07	1.99E-11	1.13E-06	NA	1E-07	2E-06
Mercury (inorganic) and compounds	7.19E-08	2.54E-08	5.99E-07	1.02E-07	3.38E-06	1.12E-06	2.08E-09	4.13E-06	NA	7E-08	9E-06
Nickel and compounds	NA	1.18E-09	5.96E-08	1.02E-08	7.48E-08	2.48E-07	1.15E-13	1.47E-06	NA	NA	2E-06

Table 4.2.4K

ESTIMATED AVERAGE BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	8.04E-06	NA	NA	NA	NA	NA	NA	NA	NA	8E-06	NA
Arsenic and compounds	1.34E-10	2.14E-11	9.27E-11	1.85E-10	4.52E-10	2.70E-10	1.07E-15	1.79E-09	NA	1E-10	3E-09
Beryllium and compounds	1.51E-12	NA	NA	NA	NA	NA	NA	NA	NA	2E-12	NA
Cadmium and compounds	6.57E-08	NA	NA	NA	NA	NA	NA	NA	NA	7E-08	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	1.17E-10	3.75E-10	7.58E-10	1.51E-09	7.40E-08	7.36E-07	1.42E-13	2.07E-07	NA	1E-10	1E-06
Chlorophenols (treated as 2,4,6-trichlorophenol)	9.79E-11	3.15E-10	6.36E-10	1.27E-09	3.10E-09	2.16E-08	1.09E-12	4.77E-07	NA	1E-10	5E-07
Chromium (VI) and compounds	8.53E-09	NA	NA	NA	NA	NA	NA	NA	NA	9E-09	NA
Nickel and compounds	6.30E-08	NA	NA	NA	NA	NA	NA	NA	NA	6E-08	NA
Polychlorinated Biphenyls	3.62E-12	3.00E-13	1.30E-12	2.59E-12	1.27E-10	1.26E-09	1.13E-12	4.51E-10	1.64E-10	4E-12	2E-09
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	5.43E-13	2.62E-13	5.29E-13	1.05E-12	2.42E-10	1.99E-09	2.94E-13	8.10E-11	2.10E-10	5E-13	3E-09
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	4.64E-09	3.85E-10	1.67E-09	3.32E-09	NA	NA	1.12E-11	5.78E-07	NA	5E-09	6E-07

ESTIMATED AVERAGE BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	1.88E-08	2.91E-10	1.47E-08	2.51E-09	3.07E-09	6.11E-09	3.78E-14	3.29E-07	NA	2E-08	4E-07
Beryllium and compounds	NA	2.33E-14	1.18E-12	2.01E-13	2.46E-13	4.41E-15	1.16E-16	3.18E-12	NA	NA	5E-12
Cadmium and compounds	NA	3.99E-09	2.02E-07	3.45E-08	2.33E-08	8.44E-07	1.08E-11	1.33E-05	NA	NA	1E-05
Chromium (VI) and compounds	NA	1.41E-11	7.15E-10	1.22E-10	8.22E-10	4.46E-09	2.32E-14	2.31E-08	NA	NA	3E-08
Lead and compounds	1.51E-07	1.17E-08	2.75E-07	4.69E-08	1.72E-08	2.86E-07	1.83E-11	1.61E-06	NA	2E-07	2E-06
Mercury (inorganic) and compounds	1.02E-07	3.60E-08	8.49E-07	1.45E-07	4.79E-06	1.59E-06	1.92E-09	5.85E-06	NA	1E-07	1E-05
Nickel and compounds	NA	1.67E-09	8.45E-08	1.44E-08	1.06E-07	3.51E-07	1.06E-13	2.08E-06	NA	NA	3E-06

Table 4.2.4L

ESTIMATED MAXIMUM BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	3.52E-05	NA	NA	NA	NA	NA	NA	NA	NA	4E-05	NA
Arsenic and compounds	5.85E-10	9.38E-11	1.13E-09	5.69E-09	2.28E-11	1.95E-12	6.44E-13	1.59E-09	NA	6E-10	9E-09
Beryllium and compounds	6.61E-12	NA	NA	NA	NA	NA	NA	NA	NA	7E-12	NA
Cadmium and compounds	2.88E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	5.12E-10	1.64E-09	9.23E-09	4.66E-08	3.72E-09	5.31E-09	4.20E-11	1.75E-08	NA	5E-10	8E-08
Chlorophenols (treated as 2,4,6-trichlorophenol)	4.29E-10	1.38E-09	7.74E-09	3.91E-08	1.56E-10	1.56E-10	3.20E-10	2.16E-08	NA	4E-10	7E-08
Chromium (VI) and compounds	3.74E-08	NA	NA	NA	NA	NA	NA	NA	NA	4E-08	NA
Nickel and compounds	2.76E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Polychlorinated Biphenyls	1.59E-11	3.94E-12	4.74E-11	2.39E-10	1.91E-11	2.73E-11	4.36E-10	7.67E-11	4.50E-10	2E-11	1E-09
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	2.38E-12	3.44E-12	1.93E-11	9.75E-11	3.73E-11	4.36E-11	1.53E-10	2.97E-11	2.10E-10	2E-12	6E-10
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	2.03E-08	5.06E-09	6.08E-08	3.07E-07	NA	NA	5.14E-09	9.84E-08	NA	2E-08	5E-07

ESTIMATED MAXIMUM BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	8.24E-08	1.28E-09	1.79E-07	7.74E-08	1.55E-10	4.41E-11	2.27E-11	2.87E-08	NA	8E-08	3E-07
Beryllium and compounds	NA	1.02E-13	1.43E-11	6.20E-12	1.24E-14	3.18E-17	3.45E-14	1.77E-12	NA	NA	2E-11
Cadmium and compounds	NA	1.75E-08	2.46E-06	1.06E-06	1.17E-09	6.09E-09	1.20E-08	6.61E-07	NA	NA	4E-06
Chromium (VI) and compounds	NA	6.20E-11	8.70E-09	3.76E-09	4.14E-11	3.22E-11	3.49E-11	1.55E-09	NA	NA	1E-08
Lead and compounds	6.62E-07	5.11E-08	3.35E-06	1.45E-06	8.68E-10	2.06E-09	2.22E-08	4.32E-07	NA	7E-07	5E-06
Mercury (inorganic) and compounds	4.46E-07	1.58E-07	1.03E-05	4.47E-06	2.41E-07	1.15E-08	1.67E-06	1.35E-06	NA	4E-07	2E-05
Nickel and compounds	NA	7.32E-09	1.03E-06	4.45E-07	5.33E-09	2.53E-09	8.35E-11	1.69E-07	NA	NA	2E-06

NA = Not Applicable.

Table 4.2.4M

ESTIMATED MAXIMUM BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	5.18E-05	NA	NA	NA	NA	NA	NA	NA	NA	5E-05	NA
Arsenic and compounds	8.61E-10	1.38E-10	1.66E-09	8.37E-09	5.69E-11	4.87E-12	6.81E-13	2.10E-09	NA	9E-10	1E-08
Beryllium and compounds	9.71E-12	NA	NA	NA	NA	NA	NA	NA	NA	1E-11	NA
Cadmium and compounds	4.24E-07	NA	NA	NA	NA	NA	NA	NA	NA	4E-07	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	7.52E-10	2.42E-09	1.36E-08	6.85E-08	9.31E-09	1.33E-08	4.45E-11	2.30E-08	NA	8E-10	1E-07
Chlorophenols (treated as 2,4,6-trichlorophenol)	6.31E-10	2.03E-09	1.14E-08	5.75E-08	3.91E-10	3.90E-10	3.39E-10	2.84E-08	NA	6E-10	1E-07
Chromium (VI) and compounds	5.50E-08	NA	NA	NA	NA	NA	NA	NA	NA	5E-08	NA
Nickel and compounds	4.06E-07	NA	NA	NA	NA	NA	NA	NA	NA	4E-07	NA
Polychlorinated Biphenyls	2.33E-11	5.80E-12	6.97E-11	3.52E-10	4.78E-11	6.82E-11	4.61E-10	1.01E-10	5.35E-10	2E-11	2E-09
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	3.50E-12	5.06E-12	2.84E-11	1.43E-10	9.33E-11	1.09E-10	1.62E-10	3.91E-11	3.00E-10	3E-12	9E-10
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	2.99E-08	7.44E-09	8.95E-08	4.52E-07	NA	NA	5.45E-09	1.30E-07	NA	3E-08	7E-07

ESTIMATED MAXIMUM BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	1.21E-07	1.88E-09	2.63E-07	1.14E-07	3.87E-10	1.10E-10	2.40E-11	3.77E-08	NA	1E-07	4E-07
Beryllium and compounds	NA	1.50E-13	2.11E-11	9.12E-12	3.10E-14	7.96E-17	3.65E-14	2.32E-12	NA	NA	3E-11
Cadmium and compounds	NA	2.57E-08	3.61E-06	1.56E-06	2.94E-09	1.52E-08	1.27E-08	8.69E-07	NA	NA	6E-06
Chromium (VI) and compounds	NA	9.12E-11	1.28E-08	5.53E-09	1.03E-10	8.04E-11	3.70E-11	2.04E-09	NA	NA	2E-08
Lead and compounds	9.74E-07	7.52E-08	4.92E-06	2.13E-06	2.17E-09	5.16E-09	2.35E-08	5.68E-07	NA	1E-06	8E-06
Mercury (inorganic) and compounds	6.56E-07	2.32E-07	1.52E-05	6.57E-06	6.03E-07	2.87E-08	1.76E-06	1.78E-06	NA	7E-07	3E-05
Nickel and compounds	NA	1.08E-08	1.51E-06	6.54E-07	1.33E-08	6.33E-09	8.84E-11	2.22E-07	NA	NA	2E-06

NA = Not Applicable.

Table 4.2.4N

ESTIMATED AVERAGE BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	5.68E-06	NA	NA	NA	NA	NA	NA	NA	NA	6E-06	NA
Arsenic and compounds	9.43E-11	1.51E-11	6.54E-11	1.30E-10	8.83E-12	7.68E-13	8.06E-14	1.64E-10	NA	9E-11	4E-10
Beryllium and compounds	1.06E-12	NA	NA	NA	NA	NA	NA	NA	NA	1E-12	NA
Cadmium and compounds	4.64E-08	NA	NA	NA	NA	NA	NA	NA	NA	5E-08	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	8.24E-11	2.65E-10	5.35E-10	1.07E-09	1.45E-09	2.09E-09	1.07E-11	2.27E-09	NA	8E-11	8E-09
Chlorophenols (treated as 2,4,6-trichlorophenol)	6.91E-11	2.22E-10	4.49E-10	8.95E-10	6.06E-11	6.15E-11	8.16E-11	3.36E-09	NA	7E-11	5E-09
Chromium (VI) and compounds	6.02E-09	NA	NA	NA	NA	NA	NA	NA	NA	6E-09	NA
Nickel and compounds	4.45E-08	NA	NA	NA	NA	NA	NA	NA	NA	4E-08	NA
Polychlorinated Biphenyls	2.55E-12	2.12E-13	9.17E-13	1.83E-12	2.48E-12	3.59E-12	8.52E-11	4.35E-12	5.12E-12	3E-12	1E-10
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	3.83E-13	1.85E-13	3.74E-13	7.44E-13	4.72E-12	5.68E-12	2.21E-11	1.28E-12	2.22E-12	4E-13	4E-11
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	3.28E-09	2.72E-10	1.18E-09	2.34E-09	NA	NA	8.45E-10	5.58E-09	NA	3E-09	1E-08

ESTIMATED AVERAGE BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	1.33E-08	2.05E-10	1.04E-08	1.77E-09	6.01E-11	1.74E-11	2.84E-12	3.70E-09	NA	1E-08	2E-08
Beryllium and compounds	NA	1.65E-14	8.32E-13	1.42E-13	4.81E-15	1.26E-17	8.70E-15	1.85E-13	NA	NA	1E-12
Cadmium and compounds	NA	2.82E-09	1.43E-07	2.43E-08	4.56E-10	2.40E-09	8.10E-10	1.07E-07	NA	NA	3E-07
Chromium (VI) and compounds	NA	9.98E-12	5.05E-10	8.61E-11	1.61E-11	1.27E-11	1.75E-12	2.14E-10	NA	NA	8E-10
Lead and compounds	1.07E-07	8.23E-09	1.94E-07	3.31E-08	3.37E-10	8.14E-10	1.38E-09	4.73E-08	NA	1E-07	3E-07
Mercury (inorganic) and compounds	7.19E-08	2.54E-08	5.99E-07	1.02E-07	9.36E-08	4.52E-09	1.44E-07	1.50E-07	NA	7E-08	1E-06
Nickel and compounds	NA	1.18E-09	5.96E-08	1.02E-08	2.07E-09	1.00E-09	7.98E-12	2.22E-08	NA	NA	1E-07

Table 4.2.40
ESTIMATED AVERAGE BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	8.04E-06	NA	NA	NA	NA	NA	NA	NA	NA	8E-06	NA
Arsenic and compounds	1.34E-10	2.14E-11	9.27E-11	1.85E-10	1.25E-11	1.09E-12	7.44E-14	2.33E-10	NA	1E-10	5E-10
Beryllium and compounds	1.51E-12	NA	NA	NA	NA	NA	NA	NA	NA	2E-12	NA
Cadmium and compounds	6.57E-08	NA	NA	NA	NA	NA	NA	NA	NA	7E-08	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	1.17E-10	3.75E-10	7.58E-10	1.51E-09	2.05E-09	2.97E-09	9.88E-12	3.22E-09	NA	1E-10	1E-08
Chlorophenols (treated as 2,4,6-trichlorophenol)	9.79E-11	3.15E-10	6.36E-10	1.27E-09	8.59E-11	8.71E-11	7.53E-11	4.77E-09	NA	1E-10	7E-09
Chromium (VI) and compounds	8.53E-09	NA	NA	NA	NA	NA	NA	NA	NA	9E-09	NA
Nickel and compounds	6.30E-08	NA	NA	NA	NA	NA	NA	NA	NA	6E-08	NA
Polychlorinated Biphenyls	3.62E-12	3.00E-13	1.30E-12	2.59E-12	3.51E-12	5.08E-12	7.86E-11	6.17E-12	2.44E-12	4E-12	1E-10
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	5.43E-13	2.62E-13	5.29E-13	1.05E-12	6.69E-12	8.04E-12	2.04E-11	1.81E-12	1.90E-12	5E-13	4E-11
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	4.64E-09	3.85E-10	1.67E-09	3.32E-09	NA	NA	7.80E-10	7.91E-09	NA	5E-09	1E-08

ESTIMATED AVERAGE BODY BURDEN (INTAKE) OF CHEMICALS EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS
(all values in mg/kg/day)

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL INTAKE	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	1.88E-08	2.91E-10	1.47E-08	2.51E-09	8.51E-11	2.47E-11	2.62E-12	5.25E-09	NA	2E-08	2E-08
Beryllium and compounds	NA	2.33E-14	1.18E-12	2.01E-13	6.82E-15	1.78E-17	8.03E-15	2.62E-13	NA	NA	2E-12
Cadmium and compounds	NA	3.99E-09	2.02E-07	3.45E-08	6.46E-10	3.40E-09	7.48E-10	1.52E-07	NA	NA	4E-07
Chromium (VI) and compounds	NA	1.41E-11	7.15E-10	1.22E-10	2.27E-11	1.80E-11	1.61E-12	3.03E-10	NA	NA	1E-09
Lead and compounds	1.51E-07	1.17E-08	2.75E-07	4.69E-08	4.77E-10	1.15E-09	1.27E-09	6.71E-08	NA	2E-07	4E-07
Mercury (inorganic) and compounds	1.02E-07	3.60E-08	8.49E-07	1.45E-07	1.33E-07	6.41E-09	1.33E-07	2.13E-07	NA	1E-07	2E-06
Nickel and compounds	NA	1.67E-09	8.45E-08	1.44E-08	2.93E-09	1.42E-09	7.36E-12	3.14E-08	NA	NA	1E-07

4.2.5. RISK CHARACTERIZATION

4.2.5.1. SUMMARY

In the risk characterization, the chemical intake estimates derived for the nine exposure pathways in the technical work paper (TWP) were integrated with the health criteria presented in the TWP. This results in expressions of upperbound maximum exposed individual (MEI) risks for potential carcinogens and hazard index values for noncarcinogens. An excess lifetime MEI risk represents the probability that an individual may develop cancer as a result of exposure to chemicals over a 70-year lifetime under maximum exposure conditions. The upperbound cancer risk estimates provide a rough but plausible estimate of the upper limit of risk; i.e., it is not likely that the true risk would be much more than the estimated risk, but it could very well be considerably less, even approaching zero. Additionally, hazard index values were calculated for chemicals exhibiting potential noncarcinogen effects. Hazard index values that are greater than the threshold value of one indicate the potential exists for an adverse health effect. In addition to the MEI risk, an upperbound on the population risk (cancer incidence) was calculated by multiplying the number of people potentially impacted by the average MEI risk estimates. Predicted ambient air concentrations caused by emissions from the proposed Winona facility were compared with applicable standards and guidelines, and with background air quality. Finally, uncertainties in the risk characterization were discussed.

Risks are calculated for two cases within each of three scenarios. First an average case which combines MPCA's facility emission and human consumption rates and Clement's modeling assumptions with 95% upperbound cancer potency factors. Although the exposures represented by this case have a high probability of occurring, the resultant risk has a lower probability due to the use of the upperbound cancer potency factors. Second, a maximum plausible case is used which combines maximum values from modeling results and MPCA's facility emission and human consumption rates with 95% upperbound cancer potency factors. This technique is intended to place an ultimate on the resultant risks. Although this case may have a finite probability of occurring, it may be extremely small and actually approach zero, due to the unlikely conjunction of numerous conditions represented by maximum values. The reader should keep in mind that the risks reported here are not actuarial risks, that is, they are not likely to actually occur. Rather, they are upperbounds on estimates, whether for the average or maximum plausible cases. Three exposure scenarios were evaluated: 1) a typical winona resident; 2) a self-sustaining farmer; and 3) an avid sport fisherman. Estimated risks are discussed separately for each scenario. The risk assessment results indicated the risks in general were similar for the Riverbend II and the Riverview Drive sites.

All risk assessments involve the use of assumptions, judgment, and imperfect data to varying degrees. The primary areas of uncertainty affecting the Winona risk assessment are:

- Uncertainties in air dispersion modeling;
- Uncertainties in hazard identification;
- Uncertainties in exposure assessment; and
- Uncertainties in critical toxicity values.

Uncertainty may be magnified in the assessment through a combination of these potential sources of uncertainty, and ultimately this results in uncertainty in the final estimates of risk. In risk assessments in which considerable uncertainty is anticipated, a technique commonly employed to compensate for uncertainty is to bias the assessment in the direction of overestimation of risk. This is often termed a "worst case" or "conservative" analysis. This technique was applied by Clement in their assumptions for the Winona risk assessment. However, it is not known whether or not the consumption rates provided by MPCA represent conservative assumptions, therefore Clement is unable to predict whether or not the final estimates of risk represent a worst case analysis.

4.2.5.2. ESTIMATION OF RISK

The USEPA (1986a,b) has developed guidelines for assessing the potential risks from exposure to carcinogenic and noncarcinogenic chemicals. The USEPA utilizes separate methodologies for estimating the risk from these two different classes of compounds. At low doses (doses corresponding to risks lower than 10^{-2}), the lifetime risk for a chemical exhibiting carcinogenic effects is calculated by multiplying the cancer potency factor [in $(mg/kg/day)^{-1}$] by the estimated chronic chemical intake (in mg/kg/day). The resultant risk value represents the probability that an individual may develop cancer as a result of exposure to chemicals over a 70-year lifetime under maximum exposure conditions. This is often referred to as the maximum exposed individual (MEI) risk. A similar average risk is derived for an individual exposed under average conditions. To calculate the MEI risk from exposure to all carcinogens that might be simultaneously emitted from the Winona facility, the MEI risks for the individual chemicals are summed both within each exposure pathway and across all eight ingestion pathways. The inhalation pathway is kept separate because the mechanisms by which cancer may develop through this route are different than those in the ingestion route. The maximum population risk may then be calculated by multiplying the number of individuals exposed by the total MEI carcinogenic risk.

For noncarcinogens, potential risks are calculated by means of a hazard index technique. First, the ratio of the chronic daily intake (CDI) to the reference dose (RfD) is calculated. Values of these CDI:RfD ratios that are greater than one indicate the potential may exist for adverse health effects. The effects of the mixture are computed by summing the individual ratios for each chemical. This sum, known as the hazard index, serves the same function for the mixture as the concentration ratio does for the individual compound. A hazard index greater than a threshold level of one indicates that an adverse effect may occur. In this risk assessment CDI:RfD ratios were also summed across all eight ingestion pathways, to arrive at a total hazard index for exposure to all chemicals simultaneously through all routes.

Risks are calculated for two cases. First an average case which combines average modeling and exposure assumptions developed by Clement and MPCA's requested facility emission and human consumption rates with 95% upperbound cancer potency factors. Although the exposures represented by this case have a high probability of occurring, the resultant risk has a lower probability due to the use of the upperbound cancer potency factors. Second, a maximum plausible scenario is used which combines maximum values from modeling results and for exposures developed by Clement and MPCA's requested facility emission and human consumption rates with with 95% upperbound cancer potency factors. This technique is intended to place an ultimate upperbound on the resultant risks. Although this case may have a finite probability of occurring, it may be extremely small and actually approach zero, due to the unlikely conjunction

of numerous conditions represented by maximum values. The reader should keep in mind that the risks reported here are not actuarial risks, rather, they are upperbounds on estimates, whether for the average or maximum plausible case.

As requested by MPCA, three exposure scenarios were evaluated: I) a typical winona resident; II) a self-sustaining farmer; and III) an avid sport fisherman. Estimated risks are discussed separately for each scenario. Additionally, total amounts of chemical inhaled and ingested daily, referred to as "total body burden," are calculated by summing estimated average daily intakes, in mg/kg/day, across each pathway in which exposure to those chemicals occurs. Total body burden is estimated for all chemicals using both average and maximum plausible exposure cases. In all cases estimates were made for both the Riverview and Riverbend sites.

4.2.5.3 Risk Characterization Revisions

As requested by MPCA, the following tables were submitted by ICF to summarize the carcinogenic and noncarcinogenic risks associated with each chemical and each pathway (Tables 4.2.5.A through 4.2.5L. These twelve tables correspond to the twelve exposure tables (4.2.1A through 4.2.1L) with respect to scenario, case, and site, as followws:

Scenario I - typical Winona resident

4.2.5A Total estimated risk, maximum case, Riverview site
4.2.5B Total estimated risk, maximum case, Riverbend site
4.2.5C Total estimated risk, average case, Riverview site
4.2.5D Total estimated risk, average case, Riverbend site

Scenario II - Self-sufficient farmer

4.2.5E Total estimated risk, maximum case, Riverview site
4.2.5F Total estimated risk, maximum case, Riverbend site
4.2.5G Total estimated risk, average case, Riverview site
4.3.5H Total estimated risk, average case, Riverbend site

Scenario III - Avid Sport fisherman

4.2.5I Total estimated risk, maximum case, Riverview site
4.2.5J Total estimated risk, maximum case, Riverbend site
4.2.6K Total estimated risk, average case, Riverview site
4.2.5L Total estimated risk, average case, Riverbend site

4.2.5.3.1 Scenario I: Typical Winona Resident

Total body burden (intake) of each chemical estimated for the typical Winona resident for the average and maximum plausible cases are summarized in Tables 4.2.5A through 4.2.5D. Risk estimates based on these exposures are presented in Tables 4.2.1A through 4.2.1F, the highest maximum exposed individual risks for carcinogens are associated with the ingestion of human breast-milk, soil, and dairy milk. In all cases, ingestion of PCDDs/PCDFs contributed the greatest amount of carcinogenic risk.

Risk estimates for the inhalation exposure pathway are shown in the first columns of Tables 4.2.5A through 4.2.5F. For the maximum exposed individual, the upperbound on the lifetime excess cancer risk considering all potential carcinogens is $5x10^{-6}$ for the Riverview site and $8x10^{-6}$ for the Riverbend site. The chemicals that accounted for most of the estimated risks are

aldehydes, cadmium, and hexavalent chromium (CrVI). The average case MEI risk is estimated to be $8x10^{-7}$ for the Riverview site and $1x10^{-6}$ for the Riverbend site. The maximum hazard index for the noncarcinogens is $2x10^{-3}$ for the Riverview site and $3x10^{-3}$ for the Riverbend site, almost three orders of magnitude less than the threshold level of one.

Risk estimates for the ingestion (via inhalation) pathway are shown in the second columns of Tables 4.2.5A through 4.2.5D. The upperbound MEI risk is $6x10^{-7}$ for the Riverview site and $9x10^{-7}$ for the Riverbend site. For the average case, these estimates are $3x10^{-8}$ and $5x10^{-8}$, respectively. Most of the excess cancer risk is associated with exposure to PCDDs/PCDFs. The maximum case hazard index for the ingestion of chemicals via inhalation is $1x10^{-3}$ for the Riverview site and $2x10^{-3}$ for the Riverbend site, three orders of magnitude less than the threshold level of one.

The maximum case risks associated with incidental dust ingestion are estimated to be $4x10^{-6}$ for the Riverview site and $5x10^{-6}$ for the Riverbend site, as shown in the third columns of Tables 4.4.5A through 4.2.5D. These risks are primarily associated with the ingestion of PCDDs/PCDFs and PAHs in household dust. Average case risks are estimated to be $7x10^{-8}$ and $1x10^{-7}$ for the two sites, respectively. The maximum case hazard index is $8x10^{-2}$ for the Riverview site and $1x10^{-1}$ for the Riverbend site. These are the highest hazard index values calculated for the nine exposure pathways, but are still an order of magnitude less than the threshold level of one. Potential ingestion of mercury accounts for the majority of this hazard index.

Risk estimates for the soil ingestion pathway are shown in the fourth columns of Tables 4.2.5A throuth 4.2.5D. Exposure through incidental ingestion of soil is the second highest source of carcinogenic risk for the typical Winona resident, after ingestion of human breast-milk. The upperbound MEI risk is $2x10^{-5}$ for the Riverview site and $3x10^{-5}$ for the Riverbend site based on lifetime ingestion of soil at the maximum deposition point in Winona. The average case risk is $1x10^{-7}$ for the Riverview site and $2x10^{-7}$ for the Riverbend site. These potential carcinogenic risks are primarily associated with incidental ingestion of PCDDs/PCDFs in soils. The maximum hazard index for the soil ingestion pathway is $3x10^{-2}$ for the Riverview site and $5x10^{-2}$ for the Riverbend site, both still significantly less than the threshold level of one.

The fifth columns of Tables 4.2.5A through 4.2.5D, show the estimated risks associated with ingestion of beef. The maximum case upperbound excess lifetime cancer risk is estimated to be $6x10^{-6}$ for the Riverview site and $1x10^{-5}$ for the Riverbend site with the great majority of the risk associated with ingestion of PCDDs/PCDFs in beef. The average case cancer risk is estimated to be $7x10^{-7}$ and $1x10^{-6}$ for the two sites, respectively. The maximum hazard index for exposure to noncarcinogens in beef is $2x10^{-3}$ for the Riverview site and $4x10^{-3}$ for the Riverbend site, 500 and 250 times less than the threshold level of one, respectively.

Upperbound excess lifetime cancer risks for ingestion of chemicals in dairy milk are shown in the sixth columns of Tables 4.2.5A through 4.2.5D. The maximum case risk is calculated to be $7x10^{-6}$ and $2x10^{-5}$ for the Riverview and Riverbend sites, respectively. The corresponding average case risks for the two sites are estimated to be $9x10^{-7}$ and $1x10^{-6}$, respectively. Ingestion of PCDDs/PCDFs in cow's milk accounts for the majority of the estimated risks. The maximum risk is associated with the point of maximum deposition in the farming areas near the two Winona facility sites whereas the average risk is associated with the average deposition rate across the Winona farming areas.

The maximum hazard index for this pathway is $9x10^{-5}$ for the Riverview site and $2x10^{-4}$ for the Riverbend site, approximately four orders of magnitude below the threshold level of one.

The maximum case individual upperbound excess lifetime cancer risk for fish ingestion, shown in the seventh columns of figures in Tables 4.2.5A through 4.2.5D, is estimated to be $3x10^{-7}$ for the Riverview site and $4x10^{-7}$ for the Riverbend site. This risk estimate is conservatively based upon the hypothetical assumption that the maximum deposition rate predicted to occur at one location over the Mississippi River actually occurs equally across the exposed surface of the river. The average case risk, based upon an average deposition rate for the Mississippi River, is estimated to be $5x10^{-8}$ for both the Riverview and Riverbend sites. Both the average and maximum case risk estimates are among the lowest of the nine potential exposure pathway analyzed. The risks are predominantly associated with the ingestion of PCDDs/PCDFs present in fish caught from the Mississippi River. Recent experimental evidence indicates, however, that the concentrations of organic chemicals such as PCDDs/PCDFs and PCBs are eight times lower in fish filets that in whole fish tissues (Hilmann 1987). Thus more realistic risk estimates for the ingestion of fish may be approximately one order of magnitude lower than those listed above. The maximum hazard index estimate for fish ingestion is $2x10^{-4}$ for both facility sites, five thousand times lower than the threshold level of one. The maximum case hazard index was conservatively calculated by assuming that the maximum deposition rate occurs over the exposed surface of the Mississippi River.

The risks associated with consumption of homegrown vegetables are presented in the eighth columns of Tables 4.2.5A through 4.2.5D The maximum case risk is estimated to be $6x10^{-6}$ for the Riverview site and $8x10^{-6}$ for the Riverbend site, also primarily due to the ingestion of PCDDs/PCDFs present in vegetables. The maximum case estimate is derived for the point of maximum deposition in the Winona area. The average case risk is estimated to be $3x10^{-7}$ for the Riverview site and $4x10^{-7}$ for the Riverbend site, also predominantly due to ingestion of PCDDs/PCDFs, as well as PAHs. The maximum case hazard index is $1x10^{-2}$ for both the Riverview site and the Riverbend site, two orders of magnitude lower than the threshold value of one.

The risks associated with ingestion of human breast-milk by an infant were estimated for PCDDs/PCDFs and PCBs and are shown in the ninth columns in Tables 4.2.5A through 4.2.5C The risks were based on the chronic daily intakes estimated for an infant whose mother was conservatively assumed to be exposed via all the exposure pathways reviewed above. The estimated maximum case risks for the infant are estimated to be $3x10^{-5}$ for the Riverview site and $5x10^{-5}$ for the Riverbend site. Ingestion of PCDDs/PCDFs contained in human breast-milk contributes to the vast majority of the incremental cancer risk. For the average case, the excess lifetime cancer risks are estimated to be $2x10^{-7}$ for the Riverview site and $3x10^{-7}$ for the Riverbend site.

Total upperbound excess individual lifetime cancer risks and hazard indices for the nine exposure pathways evaluated in this risk assessment are summarized in the last two columns of Tables 4.2.5A through 4.2.5C. Risks posed via the inhalation route are presented separately from the eight ingestion routes, which are summed in the last column. The total estimated maximum exposed individual risk obtained when the incremental risks from all eight ingestion pathways are summed is $7x10^{-5}$ for the Riverview site and $1x10^{-4}$ for the Riverbend site. The total risk for the inhalation route is more than an order of magnitude less, estimated to be $5x10^{-6}$ and $8x10^{-6}$ for the two sites, respectively. For the average case, total estimated risk for

the eight ingestion pathways is $2x10^{-6}$ for the Riverview site and $3x10^{-6}$ for the Riverbend site. Ingestion of PCDDs/PCDFs is by far the greatest source of cancer risk at both sites and for both the average and maximum cases.

When a total maximum hazard index value is derived by summing the CDI:RfD ratios for all potential noncarcinogens for all eight ingestion pathways the result is still less than the threshold value of one for both sites. For the Riverview site this total maximum ingestion hazard index is 0.1, while for the Riverbend site the total maximum ingestion hazard index is 0.2. For the average case, the total ingestion hazard indices are 0.007 and 0.01 for the Riverview and Riverbend sites, respectively. Ingestion of mercury contained in the various sources produces the highest estimated CDI:RfD ratio of all the chemicals exhibiting potential systemic effects, while still being lower than the threshold level of one.

4.2.5.3.2 Scenario II: Self-sustaining Farmer

Total body burden (intake) estimated for the nine exposure pathways in the self-sustaining farmer scenario is summarized in Tables 4.2.5H through 4.2.5K. Risks estimated for exposure parameters set forth by MPCA in the self-sustaining farmer scenario are presented in Tables 4.2.5E through 4.2.5H. Exposure parameters differed from the typical Winona resident scenario for only the ingestion of beef, dairy milk and locally-grown produce pathways. Based on these calculations, exposure via ingestion of human breast-milk under the self-sustaining former scenario was also estimated. These four are the only pathways which have estimated exposures and risks different from those presented under the the typical Winona resident scenario. Exposure and risk estimates for the other five pathways (inhalation, ingestion via inhalation, and ingestion of dust, soil, and fish) were the same as in the first scenario, and therefore will not be reviewed here.

Estimated risks associated with ingestion of locally-raised beef under conditions in the self-sustaining farmer scenario are presented in the fifth columns of Tables 4.2.5E through 4.2.5H. The maximum case upperbound excess lifetime cancer risk is estimated to be $2x10^{-4}$ for the Riverview site and $5x10^{-4}$ for the Riverbend site, with the great majority of the risk associated with ingestion of PCDDs/PCDFs in beef. The average case cancer risk is estimated to be $3x10^{-5}$ and $4x10^{-5}$ for the two sites, respectively. The maximum hazard index for exposure to noncarcinogens in locally-raised beef is $5x10^{-2}$ for the Riverview site and $1x10^{-1}$ for the Riverbend site.

Upperbound excess lifetime cancer risks for ingestion of chemicals in locally-produced dairy milk are shown in the sixth columns of Tables 4.2.5E through 4.2.5H The maximum case risk is calculated to be $2x10^{-3}$ and $4x10^{-3}$ for the Riverview and Riverbend sites, respectively. The corresponding average case risks for the two sites are estimated to be $2x10^{-4}$ and $3x10^{-4}$, respectively. Ingestion of PCDDs/PCDFs in dairy milk accounts for the majority of the estimated risks. The maximum risk is associated with the point of maximum deposition in the farming areas near the two Winona facility sites whereas the average risk is associated with the average deposition rate across the Winona farming areas. The maximum hazard index for the ingestion of dairy milk under the self-sustaining farmer scenario is $2x10^{-2}$ for the Riverview site and $5x10^{-2}$ for the Riverbend site.

The risk estimates for consumption of locally-produced dairy milk are the highest of any pathway analyzed in the self-sustaining farmer scenario. This relatively high risk is due in part to the assumption that all the milk consumed is produced from locally raised cows, unlike the typical Winona

resident scenario in which it was assumed that local milk is only one percent of the total milk consumed. Telephone conversations with various sources in the area (Ridgeway 1987, Wisconsin Dairies Co-op 1987, Broadwater 1987) indicated, however, that even self-sustaining farmers obtain the majority (and commonly all) of their milk commercially, rather than from their dairy cows. Therefore, assuming that all milk consumed by individuals categorized in the self-sustaining farmer scenario is produced locally may result in an overly conservative estimate of risk derived from this pathway.

The risks associated with consumption of homegrown vegetables under conditions in the self-sustaining farmer scenario are presented in the eighth columns of Tables 4.2.5E through 4.2.5H. The maximum case risk is estimated to be $1x10^{-4}$ for the both the Riverview and Riverbend sites, primarily due to the ingestion of PCDDs/PCDFs and PAHs present in vegetables. The maximum case estimate is derived for the point of maximum deposition in the Winona area. The average case risk is estimated to be $1x10^{-5}$ for the Riverview site and $2x10^{-5}$ for the Riverbend site, also predominantly due to ingestion of PCDDs/PCDFs. The maximum case hazard indices are $2x10^{-1}$ and $3x10^{-1}$ for the Riverview and Riverbend sites, respectively.

The risks associated with ingestion of human breast-milk by an infant in the self-sustaining farmer scenario were estimated for PCDDs/PCDFs and PCBs and are shown in the ninth columns in Tables 4.2.5E through 4.2.5H. The risks were based on the chronic daily intakes estimated for an infant whose mother was assumed to be exposed for 25 years via all the exposure pathways in the self-sustaining farmer scenario. The estimated maximum case risks for the infant are estimated to be $1x10^{-3}$ for the Riverview site and $3x10^{-3}$ for the Riverbend site. Ingestion of PCDDs/PCDFs contained in human breast-milk contributes to the majority of the incremental cancer risk. For the average case, the excess lifetime cancer risks are estimated to be $2x10^{-5}$ for the Riverview site and $3x10^{-5}$ for the Riverbend site. Exposure via this pathway assumes that the mother consumed only locally-produced milk for 25 years prior to giving birth. As discussed previously, this may be an overly conservative assumption.

Total excess individual lifetime cancer risks and hazard indices for the nine exposure pathways evaluated in this risk assessment for the self-sustaining farmer scenario are summarized in the last two columns of Tables 4.2.5E through 4.2.5H. Risks posed via the inhalation route are presented separately from the eight ingestion routes, which are summed in the last column. The total estimated maximum exposed individual risk obtained when the incremental risks from all eight ingestion pathways are summed is $3x10^{-3}$ for the Riverview site and $8x10^{-3}$ for the Riverbend site. The total risk for the inhalation route is approximately three orders of magnitude less, estimated to be $5x10^{-6}$ and $8x10^{-6}$ for the two sites, respectively. For the average case, total estimated risk for the eight ingestion pathways is $3x10^{-4}$ for the Riverview site and $4x10^{-4}$ for the Riverbend site. Ingestion of PCDDs/PCDFs is by far the greatest source of cancer risk at both sites and for both the average and maximum cases.

When a total maximum hazard index value is derived by summing the ratios of exposure concentrations to reference doses for all potential noncarcinogens for all eight ingestion pathways the result is still less than one for both sites. For the Riverview site this total maximum ingestion hazard index is 0.4, while for the Riverbend site the total maximum ingestion hazard index is 0.7. For the average case, the total ingestion hazard indices are 0.08 and 0.1 for the Riverview and Riverbend sites, respectively. Ingestion of mercury contained in the various sources produces the highest estimated CDI:RfD ratio

of all the chemicals exhibiting potential systemic effects.

4.2.5.3.3 Scenario III: Avid Sport Fisherman

Total body burden (intake) estimates for the nine exposure pathways in the avid sport fisherman scenario are summarized in Tables 4.2.4.L through 4.2.4O. Risks estimated for exposure parameters set forth by MPCA for the avid sport fisherman scenario are presented in Tables 4.2.5I through 4.2.5L. Ingestion of fish was the only pathway which had different exposure parameters for this scenario than the typical Winona resident scenario. Risks via the human breast-milk pathway were also estimated. Exposure and risk from the other seven pathways were the same for both scenarios and therefore will not be reviewed here.

The maximum case individual upperbound excess lifetime cancer risk for fish ingestion, shown in the seventh columns in Tables 4.2.5I through 4.2.5L, is estimated to be $2x10^{-5}$ for the Riverview site and $3x10^{-5}$ for the Riverbend site. This risk estimate is conservatively based upon the hypothetical assumption that the maximum deposition rate predicted to occur at one location over the Mississippi River actually occurs equally across the exposed surface of the river. The average case risk, based upon an average deposition rate for the Mississippi River, is estimated to be $3x10^{-6}$ for both the Riverview and Riverbend sites. These risks are approximately two orders of magnitude higher than those calculated for the typical Winona resident scenario. The risks are predominantly associated with the ingestion of PCDDs/PCDFs present in fish caught from the Mississippi River. Recent experimental evidence indicates, however, that the concentrations of organic chemicals such as PCDDs/PCDFs and PCBs are eight times lower in fish filets that in whole fish tissues (Hilmann 1987). Thus more realistic risk estimates for the ingestion of fish may be approximately one order of magnitude lower than those listed above. The maximum hazard index estimate for fish ingestion is $1x10^{-2}$ for both facility sites, one hundred times lower than the threshold level of one. The maximum case hazard index was conservatively calculated by assuming that the maximum deposition rate occurs over the exposed surface of the Mississippi River.

Total excess individual lifetime cancer risks and hazard indices for the nine exposure pathways evaluated in this risk assessment for the avid sport fisherman scenario are summarized in the last two columns of Tables 4.2.5I through 4.2.5L. Risks posed via the inhalation route are presented separately from the eight ingestion routes, which are summed in the last column. The total estimated maximum exposed individual risk obtained when the incremental risks from all eight ingestion pathways are summed is $1x10^{-4}$ for the Riverview site and $2x10^{-4}$ for the Riverbend site. The total risk for the inhalation route is much less, estimated to be $5x10^{-6}$ and $8x10^{-6}$ for the two sites, respectively. For the average case, total estimated risk for the eight ingestion pathways is $6x10^{-6}$ for the Riverview site and $7x10^{-6}$ for the Riverbend site. Ingestion of PCDDs/PCDFs is by far the greatest source of cancer risk at both sites and for both the average and maximum cases.

When a total maximum hazard index value is derived by summing the ratios of exposure concentrations to reference doses for all potential noncarcinogens for all eight ingestion pathways the result is still approximately an order of magnitude less than one for both sites. For the Riverview site this total maximum ingestion hazard index is 0.1, while for the Riverbend site the total maximum ingestion hazard index is 0.2. For the average case, the total ingestion hazard indices are 0.008 and 0.01 for the Riverview and Riverbend sites, respectively. Ingestion of mercury contained in the various sources

produces the highest CDI:RfD ratio of all the chemicals exhibiting potential systemic effects.

It should be recognized that the maximum case risk levels shown in Tables 4.2.5A through 4.2.5L, are not associated with realistic exposure scenarios but rather with hypothetical worst-case exposure scenarios. For example, the maximum case cancer risk for the fish ingestion pathway assumes that the maximum deposition rate predicted to occur at one point over the Mississippi River actually occurs equally over the river's surface. The maximum case cancer risk for the ingestion of human breast-milk assumes that a mother who breast-feeds her infant has already: inhaled the maximum predicted air concentrations every day for 25 years; ingested outdoor soil 215 days every year for 25 years; ingested vegetables grown at the point of maximum deposition for 25 years; incidentally ingested household dust every day for a 6 year period as a child; ingested fish caught from the Mississippi River (assuming the maximum deposition rate occurs equally over the river's surface) for 25 years; and ingested locally produced beef and milk for 25 years.

For the nancarcinogenic chemicals, the total hazard index across all chemicals and all pathways is less than one for each scenario for both average and maximum cases. The ICF approach for evaluating potential noncarcinogenic risks was to develop a total hazard index irregardless of endpoint first. If this value is lower than one, then specific endpoint hazard indices, ICF states, should also be lower than one. If the total hazard index were greater than one, the chemicals would be divided by endpoint and hazard indices would be recalculated. Since the hazard indices were all less than one in this revision, ICF did not recalculate endpoint specific hazard indices. Conclusions made by ICF that based on the revised hazard index values (using MPCA assumptions), it does not appear that adverse effects to any specific endpoint would be observed as a result of exposures through the pathways evaluated.

4.2.5.4 Potentially exposed population for each path relative to population size

Revised population risks were calculated based on the new MPCA input parameters as shown in Table 4.2.5M. An upperbound on the population risk (cancer incidence) was calculated by multiplying the number of people potentially impacted by the average MEI risk estimates. Population risk estimates were obtained by using the average case risks calculated for each pathway under the typical Winona resident scenario because these risks were calculated to be roughly representative of the average population exposure for Winona residents. Thus, population risks were calculated for the average case carcinogenic risk using the total population residing within a 10-mile radius of the facility except for the dust and human-breast milk pathways. These two pathways were evaluated assuming exposure to a fraction of the total population, as described in Table 4.2.5M. Cancer incidence was calculated for only the typical Winona resident scenario.

Table 4.2.5M presents the size of the potentially exposed population for each exposure pathway. These populations were based on the updated 1987 estimated Winona population of 38,032 within a ten-mile radius of the proposed facility (CACI 1987) and take into account the percentage of this population that conservatively might be exposed via each pathway. Based on the size of the potentially exposed population for each pathway, the upperbound cancer incidence estimates for each exposure pathway (i.e., the number of excess cancer cases that could occur over a 70-period) were estimated as shown in

Table 31. This table also lists the number of years it would take to observe one excess cancer case as a result of chemical emissions from the proposed Winona facility. This table indicates, for example, that a maximum of 0.002 excess cancer cases would occur over a 70-year period among the exposed Winona population, equivalent to a maximum of one excess cancer case every 40,000 years, as a result of ingesting fish caught from the Mississippi River. The maximum number of excess cancer cases over a 70-year period predicted for the Winona population, assuming exposure through all nine pathways, is 0.1 cases for the Riverview site and 0.2 for the Riverbend site. This corresponds to one additional cancer case occurring among Winona residents every 700 and 400 years, respectively.

4.2.5.5 Comparison of the original TWP with Requested Revisions

In comparing the original TWP and the results from requested revisions, ICF outlined those areas which differed. First, under exposure conditions in the typical Winona resident scenario, the revised risks were higher than Clement's estimates for the inhalation, ingestion via inhalation, and ingestion of soil and dust pathways. The revised risks were lower than Clement's estimates for the ingestion of beef, dairy milk, fish, produce, and human breast-milk pathways. Under the self-sustaining farmer scenario, all pathways which had exposure assumptions differing from the typical Winona resident scenario (ingestion of beef, dairy milk, produce, and human breast-milk) had estimated risks higher than those calculated by Clement in the original report. In the third scenario, the avid sport fisherman, risks calculated for the ingestion of fish were lower than those predicted by Clement in the TWP, as was also the case for the human breast-milk pathway. Population risk estimates, based on the average case in the typical Winona resident scenario, were the same for the Riverview Drive site, and higher for the Riverbend II site, when compared to Clement's estimates in the original report.

4.2.5.6 DISCUSSION OF UNCERTAINTIES

All risk assessments involve the use of assumptions, judgment, and imperfect data to varying degrees. This results in uncertainty in the final estimates of risk. Uncertainty in a risk assessment may arise from many sources including:

- Analytical or statistical variation in measured data;
- Misidentification or failure to be all-inclusive in hazard identification;
- Choice of models and input parameters in exposure assessment and fate and transport modeling;
- Choice of models or evaluation of toxicological data in dose-response quantification; and
- Assumptions concerning exposure scenarios and population distributions.

Uncertainty may be magnified in the assessment through a combination of these sources.

In risk assessments in which considerable uncertainty is anticipated, a technique commonly employed to compensate for uncertainty is to bias the assessment in the direction of overestimation of risk. This is often termed a "worst case" or "conservative" analysis. The net effect of combining numerous conservative assumptions is that the final estimates of risk may be greatly overestimated.

4.2.5.6.1 Uncertainties in Air Dispersion and Deposition Modeling

The parameters which describe the proposed incinerator (e.g., stack height, stack exit gas temperature, and emission rates) were based on design or regulatory values, or information available from other similar resource recovery facilities. As of the time of writing of this report (November 1987), the vendor for the plant had not been selected and specifications had not been written for the air pollution control equipment. It is also possible that minor modifications to the design may have ramifications on the risks calculated in this report, should they occur. The emission rates are a critical input into the dispersion models but they are also uncertain. Emission measurements obtained from operating facilities with similar characteristics will provide the best source of information in the absence of actual trial burn emission test results. Due to the paucity of the data base for facilities of similar characteristics and lack of knowledge of either the vendor of the facility or the design of the air pollution control systems, a wider data base was employed for selection of emission rates. Generally speaking, however, this is not likely to have a great effect on the risks since the emission rates selected were mostly of the same order of magnitude as those from facilities reviewed for this report (Section IV.D).

The ISCLT model is probably the most appropriate model for calculating long-term inhalation exposures to chemicals emitted from the facility at receptors up to stack height. However, predicting concentrations in complex terrain (i.e., at receptors above stack height) adds uncertainty to the air concentration estimates. In this risk assessment, the model output from the Complex I and ISCST models were used as a basis for estimating exposure point concentrations. Furthermore, none of the models took into account chemical removal mechanisms such as particle deposition and chemical reactions in estimating air concentrations.

Meteorological data for the long-term dispersion modeling was taken from the extensive STAR data base for La Crosse, Wisconsin. It represents an average of numerous stability classes, mixing heights, wind speeds and directions and is probably as accurate and precise as may be obtained. The major source of error which may be introduced through the meteorological data results from climatic and topographic differences between La Crosse and Winona. The magnitude of these differences is currently unknown. Uncertainties in dispersion modeling may have been compounded due to the fact that different five-year meteorological data bases were used for ISCLT (and probably Valley) compared to ISCST and Complex I. Although this is technically correct, it does propagate a lack of consistency among the outputs and makes accurate comparisons difficult.

Rather than using the STAR data from La Crosse, a more useful application of the Valley model would have been to use the default screening mode as recommended by USEPA (1986d). This would have the net effect of eliminating the probable lack of correspondence between meteorological conditions at Winona compared to La Crosse. This would also give an upper bound to the maximum concentrations obtainable in complex terrain which was not available from the current outputs of the model.

A major uncertainty affecting all the indirect pathways of exposure evaluated in this risk assessment is the estimation of particle deposition velocities. First, the particle size distribution was based on data from another incinerator (Munich North, West Germany). The degree of correspondence between this facility and the proposed Winona facility is unknown. A weighted-average deposition velocity was calculated for the Mississippi River and for overland areas based on an empirical model and using simplified assumptions to characterize surface roughness characteristics. Plume depletion with distance from the stack was not, however, taken into account in the model. This is a very conservative approach because it assumes that the particle size distribution of the emissions does not change with distance from the facility. In reality the proportion of smaller particles will increase and thus the deposition velocity will decrease with distance from the stack. This expected decrease in deposition velocity was not included in estimating deposition rates.

4.2.5.6.2 Uncertainties in Hazard Identification

In almost all risk assessments, the largest source of uncertainty is in critical toxicity values. Uncertainty in health criteria may significantly affect the magnitude of the risk estimates presented in this report. Health criteria for evaluating long-term exposures such as risk reference doses or cancer potency factors are based on concepts and assumptions which bias an evaluation in the direction of overestimation of health risk. USEPA noted in its Guidelines for Carcinogenic Risk Assessment (USEPA 1986a):

There are major uncertainties in extrapolating both from animals to humans and from high to low doses. There are important species differences in uptake, metabolism, and organ distribution of carcinogens, as well as species and strain differences in target site susceptibility. Human populations are variable with respect to geometric constitution, diet, occupational and home environment, activity patterns, and other cultural factors.

These uncertainties are compensated for by using upper bounds for cancer potency factors for carcinogens and safety factors for reference doses for noncarcinogens. At best, the assumptions used here provide a rough but plausible estimate of the upper limit of risk, i.e., it is not likely that the true risk would be much more than the estimated risk, but it could very well be considerably lower, even approaching zero.

In addition, there are varying degrees of confidence in the weight of evidence for carcinogenicity of a given chemical. USEPA's (1986a) weight-of-evidence classification provides information which can indicate the level of confidence or uncertainty in a cancer risk estimate. For example, several of the carcinogens evaluated in this report are classified in Groups B1 (beryllium and cadmium) or B2 (PCDDs/PCDFs, benzo[a]pyrene, formaldehyde), probable human carcinogens with limited or inadequate evidence from human studies and adequate evidence from animal studies. Some of the uncertainties in the hazard evaluation are further compensated for by assuming that suspected carcinogens behave as true carcinogens. The summation of the risks associated with all potential carcinogens tends to overestimate risk by including probable human carcinogens with demonstrated human carcinogens (Group A).

The chemicals of primary concern at the Winona facility are arsenic, cadmium, chromium (VI), PCDDs/PCDFs, and PAHs. In general, cancer potency factors are very uncertain and usually very conservative, but the sources of uncertainty differ among the chemicals. For inorganic chemicals including

arsenic and cadmium, the major sources of uncertainty are limitations in the data based on the human epidemiological studies and uncertainty about the physical and chemical forms of the inorganic chemicals in the workplace (mostly smelters) from which the data were derived. The cancer potency factor for chromium is associated with only the hexavalent form which was assumed to be 10% of the total chromium as per USEPA guidance (1987). The carcinogenic potency for nickel is based primarily on nickel subsulfide which has a very low probability of being emitted from a resource recovery facility. For the organic carcinogens, including PCDDs/PCDFs, the major sources of uncertainty are from the extrapolation from the carcinogenic effects of the compounds in laboratory test animals exposed to relatively high doses to humans exposed to relatively low doses. For PCDDs/PCDFs, the upper bound cancer potency factor as derived from the linearized multistage model differs from the lower bound by five orders of magnitude, yielding a large window of uncertainty in this interpretation. Recent information suggesting that PCDDs/PCDFs behave as promoters rather than initiators of cancer could lower the calculated risk by a large degree.

Risk assessments for polycyclic aromatic hydrocarbons (PAHs) have many of the problems that are associated with risk assessments for PCDDs and PCDFs. Only a few components of these mixtures have been characterized adequately, and only limited information is available on the relative potencies of different compounds. The approach adopted by USEPA (1980a, 1984a) as the basis for risk assessment is to divide the PAHs into two subclasses, "carcinogenic" PAHs and "noncarcinogenic" PAHs. This approach is a crude version of the toxic equivalency factor (TEF) approach that is used for PCDDs/PCDFs; B[a]P is used as the surrogate compound, a TEF of 1.0 is applied to all carcinogenic PAHs, and a TEF of zero is applied to all noncarcinogenic PAHs. From the results of the emissions analyses presented by Batelle (1986), of the total PAH mixture emitted, 0.75% would be comprised of carcinogenic PAHs (CPAHs). This adjustment should have the net effect of lowering uncertainty for the Winona facility compared to other projects where all PAHs were treated as carcinogenic regardless of their evidence of carcinogenicity. The cancer potency factor for B[a]P was then applied to all carcinogenic PAHs although this may overestimate risk. This is due to the fact that the majority of PAHs that have been studied exhibit potencies lower than B[a]P (Deutsch-Wensel et al. 1983, Bingham and Falk 1969, Wynder and Hoffman 1959).

Toxic equivalency schemes have not been developed for other families of organic chemicals treated in this report. All 209 PCB congeners are assumed to have the same toxicity as Aroclor 1260; all aldehydes are assumed to be as toxic as formaldehyde; all chlorobenzenes are assumed to be as toxic as p-dichlorobenzene; and all chlorophenols are assumed to be as toxic as 2,4,6-trichlorophenol. There is a considerable body of knowledge that suggests that these assumptions lead to overestimation of risk, however, neither the emissions chemistry nor the toxicology of these families have been defined well enough for detailed characterization.

Another factor which can contribute to uncertainties in cancer risk assessment is the number of chemicals evaluated. Since cancer risks are considered to be additive, risks for facilities where a large number of chemicals are evaluated will inevitably be larger than those where a small number are evaluated especially if the products of the emission rates and cancer potency factors are of the same order of magnitude. The suite of chemicals evaluated for Winona is large compared to that evaluated for the typical facility. If the risks associated with these chemicals are accurate, this should decrease the uncertainty, however, the risks may be increased.

4.2.5.6.3 Uncertainties in Exposure Assessment

There are two major areas of uncertainty in the exposure assessment. These include the choice of models and input parameters used to estimate exposure point concentrations and the methods and parameters used to calculate chemical intakes. Very conservative assumptions were used in calculating soil concentrations, and these concentrations provided the basis for estimating chemical intakes via outdoor soil ingestion and indoor dust ingestion. Numerous additional assumptions were applied in estimating chemical concentrations in homegrown vegetables, in livestock, and in fish caught from the Mississippi River. An effort was made to compensate for this uncertainty by applying conservative assumptions.

Similar comments apply to the estimation of chemical intakes. Major uncertainties are associated with the parameters used to characterize maximum case exposures, including the assumed points of exposure, the contact rates with contaminated exposure media, and the frequency and duration of exposure. Again, uncertainties affecting the estimation of chemical intakes were compensated for by making conservative assumptions. For example, the contact rates with each contaminated environmental media (e.g., inhalation or fish ingestion rate) were assumed to be constant throughout the exposure period within each age category. Exposures were also estimated assuming no Winona residents migrated from the city during a 30- or 70-year period. In estimating infant exposures to PCDDs/PCDFs, it was conservatively assumed that the mother was exposed simultaneously throughout her first 40 years to all of the evaluated exposure pathways except breast-milk ingestion.

4.2.5.7 COMPARISON WITH APPLICABLE STANDARDS AND GUIDELINES

In this section, the air concentrations predicted for the Winona facility will be compared with applicable standards for the criteria pollutants. Because the State of Minnesota has not yet adopted standards or guidelines for non-criteria pollutants, the predicted air concentrations will also be compared with applicable standards and guidelines developed for air toxics by other states and countries.

Table 4.2.5N represents critical toxicity values for short term inhalation exposure to hydrogen chloride and aldehydes and are compared with predicted short-term air concentrations.

TABLE 4.2.5N
RISKS RESULTING FROM ACUTE INHALATION EXPOSURES

Chemical	Maximum Predicted Short-Term Concentration (ug/m^3)	Health Criteria (ug/m^3)	Hazard Ratio
Aldehydes (treated as formaldehyde)[a]			
Riverview	0.28	150	<1
Riverbend	0.42	150	<1
Hydrogen chloride[b]			
Riverview	18.1	70	<1
Riverbend	23.5	70	<1

[a] Listed values are 8-hour maximum predicted concentrations because the health criteria was derived from an 8-hour time weighted average threshold limit value (ACGIH 1986). The generic maximum concentrations for a 1 g/sec emission rate for one boiler unit were 8.34 ug/m^3 for the Riverview site and 12.3 ug/m^3 for the Riverbend site.

[b] Listed values are 1-hour maximum predicted concentrations because the health criteria for HCl was derived from a ceiling threshold limit value (ACGIH 1986). The generic maximum concentrations for a 1 g/sec emission rate for one boiler unit were 22.6 ug/m^3 for the Riverview site and 29.4 ug/m^3 for the riverbend site.

4.2.5.7.1 Comparison With Standards For Criteria Pollutants

Among the selected chemicals of concern evaluated in this risk assessment, several are currently regulated under the U.S. Clean Air Act. These chemicals are sulfur dioxide, nitrogen oxides, particulate matter, and carbon monoxide. Table 4.2.50 compares the maximum predicted air concentrations of these chemicals with their applicable national ambient air quality standards (NAAQS). As can be seen, none of the predicted concentrations resulting from the proposed facility exceed the NAAQS. However, the predicted concentrations for the Riverbend site slightly exceed the concentrations predicted for the Riverview site.

Table 4.2.5O represents a comparison of predicted air concentrations with national ambient air quality standards.

4.2.5.8 COMPARISON WITH BACKGROUND AIR QUALITY

In Section V.C, available monitoring data on existing air quality for the chemicals of concern were compiled. Table 4.2.5P compares the maximum predicted air concentrations for the proposed Winona facility with the data on background air quality. This comparison indicates the incremental increases in air concentrations for all evaluated chemicals associated with the proposed Riverview Drive site are lower than existing background levels. Estimates for the proposed Riverbend II site indicate cadmium, nickel, and sulfur dioxide emissions would result in air concentrations within the range of existing background levels.

4.2.5.9 IMPACT ON RESPIRATORY DISEASE INCIDENCE

As described in Section VIII.D, health statistics describing the incidence of cancer and other diseases among Minnesota and Winona residents is not currently available. As a result, the potential impacts of emissions from the proposed facility on the incidence of disease in the potentially exposed Winona population cannot be quantitatively compared to existing incidence data. However, as presented above, results of the risk assessment can be used to predict the excess incidence of cancer (i.e., the number of excess cancer that could occur over a 70-year period) among the exposed population.

4.2.5.10 OFF-NORMAL OPERATING CONDITIONS

The evaluation presented thus far in this document assumes that the facility is operating under design combustion conditions and that all of the air pollution devices are operating at design efficiency. It is possible that these conditions will not always be met and that the level of emissions of the

facility may be increased. These are referred to as off-normal conditions; releases in the off-normal mode are usually of higher concentration than routine emissions, however, they are of much shorter duration. Conceptually, the emissions control train at Winona as envisioned at this writing includes proper control of combustion (temperature, turbulence, residence time), an afterburner, a dry scrubber, and a fabric filter baghouse. Since the specifications for the equipment have not been finalized, this may later be modified. Additionally, the incinerator is composed of two units, therefore, it is assumed that, if problems were to develop with one unit, it could be shut down and the second unit would assume its load.

Few risk assessments have evaluated off-normal conditions in detail. The assessment for the SANDER facility (Signal Environmental 1987) proposed a worst-case scenario in which all of the emission controls would fail at one time and that the failure would last for three hours. These investigators assessed the potential for effects related to off-normal exposures by comparing ambient concentrations to Threshold Limit Values (TLVs) published by the American Conference of Governmental Industrial Hygienists. All of the concentrations for the worst case three hour exposure were less than the adjusted TLVs.

A more sophisticated approach was undertaken by Westinghouse (1986) in their risk assessment for the proposed Bloomington Facility. Westinghouse used techniques of probabilistic risk analysis to develop probabilities of failure of different components in the system. The probabilities were combined via fault tree analysis for the whole system. They concluded for this plant that the annual release frequency for particulate was 0.12, for acid gases was 0.65, and for products of incomplete combustion was 0.30. They also found that the duration of an off-normal release was inversely proportional to the probability of the release--the probability decreased as the release became longer. The effects of the releases were evaluated by comparison to acute health criteria developed either from TLVs or, for PCDD/PCDFs on a health basis. For all the materials analyzed, both the expected maximum concentration and the peak maximum concentration were less than the health based criteria by at least an order of magnitude. Off-normal risks associated with PCDD/PCDFs were evaluated for four different failure scenarios ranging from poor combustion conditions in the combustor and afterburner and failure of the scrubber to simultaneous failure of six bags in a fabric filter. In each case, the resultant concentrations were at least one order of magnitude less than the health based criteria.

In conclusion, based on two studies evaluated in the published risk assessment literature, releases due to off-normal operations are not likely to exceed health based criteria. This is a result both of the short duration of the release and the low probability of these releases occurring.

Table 4.2.5A
SUMMARY OF ESTIMATED MAXIMUM CARCINOGENIC RISKS

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL RISK	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	1.2E-06	NA	NA	NA	NA	NA	NA	NA	NA	1E-06	NA
Arsenic and compounds	2.9E-08	1.4E-09	1.7E-08	8.5E-08	3.4E-10	2.9E-11	1.4E-13	2.4E-08	NA	3E-08	1E-07
Beryllium and compounds	5.5E-11	NA	NA	NA	NA	NA	NA	NA	NA	6E-11	NA
Cadmium and compounds	1.8E-06	NA	NA	NA	NA	NA	NA	NA	NA	2E-06	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	1.0E-11	3.3E-11	1.8E-10	9.3E-10	7.4E-11	1.1E-10	1.2E-14	3.5E-10	NA	1E-11	2E-09
Chlorophenols (treated as 2,4,6-trichlorophenol)	8.6E-12	2.8E-11	1.5E-10	7.8E-10	3.1E-12	3.1E-12	9.2E-14	4.3E-10	NA	9E-12	1E-09
Chromium (VI) and compounds	1.5E-06	NA	NA	NA	NA	NA	NA	NA	NA	2E-06	NA
Nickel and compounds	2.2E-07	NA	NA	NA	NA	NA	NA	NA	NA	2E-07	NA
Polychlorinated Biphenyls	1.2E-10	3.0E-11	3.7E-10	1.8E-09	1.5E-10	2.1E-10	4.8E-11	5.9E-10	2.7E-09	1E-10	6E-09
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	3.7E-07	5.4E-07	3.0E-06	1.5E-05	5.8E-06	6.8E-06	3.4E-07	4.6E-06	2.7E-05	4E-07	6E-05
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	1.2E-07	5.8E-08	7.0E-07	3.5E-06	NA	NA	8.5E-10	1.1E-06	NA	1E-07	5E-06
TOTAL CARCINOGENIC RISK	5E-06	6E-07	4E-06	2E-05	6E-06	7E-06	3E-07	6E-06	3E-05	5E-06	7E-05

SUMMARY OF ESTIMATED MAXIMUM HAZARD INDICES

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL HAZARD INDEX	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	1.6E-04	3.2E-12	4.5E-10	1.9E-10	3.9E-13	1.1E-13	8.2E-16	7.2E-11	NA	2E-04	7E-10
Beryllium and compounds	NA	2.0E-11	2.9E-09	1.2E-09	2.5E-12	6.4E-15	9.9E-14	3.5E-10	NA	NA	4E-09
Cadmium and compounds	NA	3.5E-05	4.9E-03	2.1E-03	2.3E-06	1.2E-05	3.4E-07	1.3E-03	NA	NA	8E-03
Chromium (VI) and compounds	NA	1.2E-08	1.7E-06	7.5E-07	8.3E-09	6.4E-09	1.0E-10	3.1E-07	NA	NA	3E-06
Lead and compounds	1.5E-03	8.5E-05	5.6E-03	2.4E-03	5.4E-09	1.3E-08	5.3E-07	7.2E-04	NA	2E-03	9E-03
Mercury (inorganic) and compounds	4.5E-04	9.9E-04	6.5E-02	2.8E-02	1.5E-03	7.2E-05	1.5E-04	8.5E-03	NA	4E-04	1E-01
Nickel and compounds	NA	7.3E-07	1.0E-04	4.4E-05	8.9E-06	4.2E-06	1.2E-10	1.7E-05	NA	NA	2E-04
TOTAL HAZARD INDEX	2E-03	1E-03	8E-02	3E-02	2E-03	9E-05	2E-04	1E-02	NA	2E-03	1E-01

NA = Not Applicable.

Table 4.2.5B

SUMMARY OF ESTIMATED MAXIMUM CARCINOGENIC RISKS

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL RISK	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	1.7E-06	NA	NA	NA	NA	NA	NA	NA	NA	2E-06	NA
Arsenic and compounds	4.3E-08	2.1E-09	2.5E-08	1.3E-07	8.5E-10	7.3E-11	1.5E-13	3.1E-08	NA	4E-08	2E-07
Beryllium and compounds	8.2E-11	NA	NA	NA	NA	NA	NA	NA	NA	8E-11	NA
Cadmium and compounds	2.6E-06	NA	NA	NA	NA	NA	NA	NA	NA	3E-06	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	1.5E-11	4.8E-11	2.7E-10	1.4E-09	1.9E-10	2.7E-10	1.3E-14	4.6E-10	NA	2E-11	3E-09
Chlorophenols (treated as 2,4,6-trichlorophenol)	1.3E-11	4.1E-11	2.3E-10	1.1E-09	7.8E-12	7.8E-12	9.8E-14	5.7E-10	NA	1E-11	2E-09
Chromium (VI) and compounds	2.3E-06	NA	NA	NA	NA	NA	NA	NA	NA	2E-06	NA
Nickel and compounds	3.2E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Polychlorinated Biphenyls	1.8E-10	4.5E-11	5.4E-10	2.7E-09	3.7E-10	5.3E-10	5.1E-11	7.8E-10	4.1E-09	2E-10	9E-09
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	5.5E-07	7.9E-07	4.4E-06	2.2E-05	1.5E-05	1.7E-05	3.6E-07	6.1E-06	4.7E-05	5E-07	1E-04
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	1.8E-07	8.6E-08	1.0E-06	5.2E-06	NA	NA	9.0E-10	1.5E-06	NA	2E-07	8E-06
TOTAL CARCINOGENIC RISK	8E-06	9E-07	5E-06	3E-05	1E-05	2E-05	4E-07	8E-06	5E-05	8E-06	1E-04

SUMMARY OF ESTIMATED MAXIMUM HAZARD INDICES

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL HAZARD INDEX	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	2.4E-04	4.7E-12	6.6E-10	2.8E-10	9.7E-13	2.8E-13	8.6E-16	9.4E-11	NA	2E-04	1E-09
Beryllium and compounds	NA	3.0E-11	4.2E-09	1.8E-09	6.2E-12	1.6E-14	1.1E-13	4.6E-10	NA	NA	7E-09
Cadmium and compounds	NA	5.1E-05	7.2E-03	3.1E-03	5.9E-06	3.0E-05	3.6E-07	1.7E-03	NA	NA	1E-02
Chromium (VI) and compounds	NA	1.8E-08	2.6E-06	1.1E-06	2.1E-08	1.6E-08	1.1E-10	4.1E-07	NA	NA	4E-06
Lead and compounds	2.3E-03	1.3E-04	8.2E-03	3.5E-03	1.4E-08	3.2E-08	5.7E-07	9.5E-04	NA	2E-03	1E-02
Mercury (inorganic) and compounds	6.6E-04	1.5E-03	9.5E-02	4.1E-02	3.8E-03	1.8E-04	1.6E-04	1.1E-02	NA	7E-04	2E-01
Nickel and compounds	NA	1.1E-06	1.5E-04	6.5E-05	2.2E-05	1.1E-05	1.3E-10	2.2E-05	NA	NA	3E-04
TOTAL HAZARD INDEX	3E-03	2E-03	1E-01	5E-02	4E-03	2E-04	2E-04	1E-02	NA	3E-03	2E-01

NA = Not Applicable.

Table 4.2.5C

SUMMARY OF ESTIMATED AVERAGE CARCINOGENIC RISKS

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL RISK	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	1.9E-07	NA	NA	NA	NA	NA	NA	NA	NA	2E-07	NA
Arsenic and compounds	4.7E-09	2.3E-10	9.8E-10	2.0E-09	1.3E-10	1.2E-11	1.7E-14	2.5E-09	NA	5E-09	6E-09
Beryllium and compounds	8.9E-12	NA	NA	NA	NA	NA	NA	NA	NA	9E-12	NA
Cadmium and compounds	2.8E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	1.6E-12	5.3E-12	1.1E-11	2.1E-11	2.9E-11	4.2E-11	3.1E-15	4.5E-11	NA	2E-12	2E-10
Chlorophenols (treated as 2,4,6-trichlorophenol)	1.4E-12	4.4E-12	9.0E-12	1.8E-11	1.2E-12	1.2E-12	2.4E-14	6.7E-11	NA	1E-12	1E-10
Chromium (VI) and compounds	2.5E-07	NA	NA	NA	NA	NA	NA	NA	NA	2E-07	NA
Nickel and compounds	3.6E-08	NA	NA	NA	NA	NA	NA	NA	NA	4E-08	NA
Polychlorinated Biphenyls	2.0E-11	1.6E-12	7.1E-12	1.4E-11	1.9E-11	2.8E-11	9.5E-12	3.4E-11	1.4E-11	2E-11	1E-10
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	6.0E-08	2.9E-08	5.8E-08	1.2E-07	7.4E-07	8.9E-07	5.0E-08	2.0E-07	2.1E-07	6E-08	2E-06
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	2.0E-08	3.1E-09	1.4E-08	2.7E-08	NA	NA	1.4E-10	6.4E-08	NA	2E-08	1E-07
TOTAL CARCINOGENIC RISK	8E-07	3E-08	7E-08	1E-07	7E-07	9E-07	5E-08	3E-07	2E-07	8E-07	2E-06

SUMMARY OF ESTIMATED AVERAGE HAZARD INDICES

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL HAZARD INDEX	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	2.7E-05	5.1E-13	2.6E-11	4.4E-12	1.5E-13	4.4E-14	1.0E-16	9.3E-12	NA	3E-05	4E-11
Beryllium and compounds	NA	3.3E-12	1.7E-10	2.8E-11	9.6E-13	2.5E-15	2.5E-14	3.7E-11	NA	NA	2E-10
Cadmium and compounds	NA	5.6E-06	2.9E-04	4.9E-05	9.1E-07	4.8E-06	2.3E-08	2.1E-04	NA	NA	6E-04
Chromium (VI) and compounds	NA	2.0E-09	1.0E-07	1.7E-08	3.2E-09	2.5E-09	5.0E-12	4.3E-08	NA	NA	2E-07
Lead and compounds	2.5E-04	1.4E-05	3.2E-04	5.5E-05	2.1E-09	5.1E-09	3.3E-08	7.9E-05	NA	2E-04	5E-04
Mercury (inorganic) and compounds	7.2E-05	1.6E-04	3.7E-03	6.4E-04	5.9E-04	2.8E-05	1.3E-05	9.4E-04	NA	7E-05	6E-03
Nickel and compounds	NA	1.2E-07	6.0E-06	1.0E-06	3.4E-06	1.7E-06	1.2E-11	2.2E-06	NA	NA	1E-05
TOTAL HAZARD INDEX	3E-04	2E-04	4E-03	7E-04	6E-04	3E-05	1E-05	1E-03	NA	3E-04	7E-03

NA = Not Applicable.

Table 4.2.5D

SUMMARY OF ESTIMATED AVERAGE CARCINOGENIC RISKS

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL RISK	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	2.7E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Arsenic and compounds	6.7E-09	3.2E-10	1.4E-09	2.8E-09	1.9E-10	1.6E-11	1.6E-14	3.5E-09	NA	7E-09	8E-09
Beryllium and compounds	1.3E-11	NA	NA	NA	NA	NA	NA	NA	NA	1E-11	NA
Cadmium and compounds	4.0E-07	NA	NA	NA	NA	NA	NA	NA	NA	4E-07	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	2.3E-12	7.5E-12	1.5E-11	3.0E-11	4.1E-11	5.9E-11	2.8E-15	6.4E-11	NA	2E-12	2E-10
Chlorophenols (treated as 2,4,6-trichlorophenol)	2.0E-12	6.3E-12	1.3E-11	2.5E-11	1.7E-12	1.7E-12	2.2E-14	9.5E-11	NA	2E-12	1E-10
Chromium (VI) and compounds	3.5E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Nickel and compounds	5.0E-08	NA	NA	NA	NA	NA	NA	NA	NA	5E-08	NA
Polychlorinated Biphenyls	2.8E-11	2.3E-12	1.0E-11	2.0E-11	2.7E-11	3.9E-11	8.7E-12	4.7E-11	1.9E-11	3E-11	2E-10
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	8.5E-08	4.1E-08	8.3E-08	1.6E-07	1.0E-06	1.3E-06	4.6E-08	2.8E-07	3.0E-07	8E-08	3E-06
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	2.8E-08	4.4E-09	1.9E-08	3.8E-08	NA	NA	1.3E-10	9.1E-08	NA	3E-08	2E-07
TOTAL CARCINOGENIC RISK	1E-06	5E-08	1E-07	2E-07	1E-06	1E-06	5E-08	4E-07	3E-07	1E-06	3E-06

SUMMARY OF ESTIMATED AVERAGE HAZARD INDICES

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL HAZARD INDEX	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	3.8E-05	7.3E-13	3.7E-11	6.3E-12	2.1E-13	6.2E-14	9.4E-17	1.3E-11	NA	4E-05	6E-11
Beryllium and compounds	NA	4.7E-12	2.4E-10	4.0E-11	1.4E-12	3.6E-15	2.3E-14	5.2E-11	NA	NA	3E-10
Cadmium and compounds	NA	8.0E-06	4.0E-04	6.9E-05	1.3E-06	6.8E-06	2.2E-08	3.0E-04	NA	NA	8E-04
Chromium (VI) and compounds	NA	2.8E-09	1.4E-07	2.4E-08	4.5E-09	3.6E-09	4.6E-12	6.1E-08	NA	NA	2E-07
Lead and compounds	3.5E-04	1.9E-05	4.6E-04	7.8E-05	3.0E-09	7.2E-09	3.1E-08	1.1E-04	NA	4E-04	7E-04
Mercury (inorganic) and compounds	1.0E-04	2.3E-04	5.3E-03	9.1E-04	8.3E-04	4.0E-05	1.2E-05	1.3E-03	NA	1E-04	9E-03
Nickel and compounds	NA	1.7E-07	8.4E-06	1.4E-06	4.9E-06	2.4E-06	1.1E-11	3.1E-06	NA	NA	2E-05
TOTAL HAZARD INDEX	5E-04	3E-04	6E-03	1E-03	8E-04	5E-05	1E-05	2E-03	NA	5E-04	1E-02

NA = Not Applicable.

Table 4.2.5E
SUMMARY OF ESTIMATED MAXIMUM CARCINOGENIC RISKS

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL RISK	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	1.2E-06	NA	NA	NA	NA	NA	NA	NA	NA	1E-06	NA
Arsenic and compounds	2.9E-08	1.4E-09	1.7E-08	8.5E-08	1.2E-08	7.2E-09	1.4E-13	1.6E-07	NA	3E-08	3E-07
Beryllium and compounds	5.5E-11	NA	NA	NA	NA	NA	NA	NA	NA	6E-11	NA
Cadmium and compounds	1.8E-06	NA	NA	NA	NA	NA	NA	NA	NA	2E-06	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	1.0E-11	3.3E-11	1.8E-10	9.3E-10	2.7E-09	2.6E-08	1.2E-14	1.5E-08	NA	1E-11	4E-08
Chlorophenols (treated as 2,4,6-trichlorophenol)	8.6E-12	2.8E-11	1.5E-10	7.8E-10	1.1E-10	7.7E-10	9.2E-14	3.3E-08	NA	9E-12	3E-08
Chromium (VI) and compounds	1.5E-06	NA	NA	NA	NA	NA	NA	NA	NA	2E-06	NA
Nickel and compounds	2.2E-07	NA	NA	NA	NA	NA	NA	NA	NA	2E-07	NA
Polychlorinated Biphenyls	1.2E-10	3.0E-11	3.7E-10	1.8E-09	5.3E-09	5.2E-08	4.8E-11	1.4E-08	3.9E-08	1E-10	1E-07
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	3.7E-07	5.4E-07	3.0E-06	1.5E-05	2.1E-04	1.7E-03	3.4E-07	8.3E-05	1.1E-03	4E-07	3E-03
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	1.2E-07	5.8E-08	7.0E-07	3.5E-06	NA	NA	8.5E-10	2.7E-05	NA	1E-07	3E-05
TOTAL CARCINOGENIC RISK	5E-06	6E-07	4E-06	2E-05	2E-04	2E-03	3E-07	1E-04	1E-03	5E-06	3E-03

SUMMARY OF ESTIMATED MAXIMUM HAZARD INDICES

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL HAZARD INDEX	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	1.6E-04	3.2E-12	4.5E-10	1.9E-10	1.4E-11	2.7E-11	8.2E-16	2.9E-09	NA	2E-04	4E-09
Beryllium and compounds	NA	2.0E-11	2.9E-09	1.2E-09	9.0E-11	1.6E-12	9.9E-14	3.1E-09	NA	NA	7E-09
Cadmium and compounds	NA	3.5E-05	4.9E-03	2.1E-03	8.5E-05	3.0E-03	3.4E-07	9.1E-02	NA	NA	1E-01
Chromium (VI) and compounds	NA	1.2E-08	1.7E-06	7.5E-07	3.0E-07	1.6E-06	1.0E-10	1.6E-05	NA	NA	2E-05
Lead and compounds	1.5E-03	8.5E-05	5.6E-03	2.4E-03	2.0E-07	3.2E-06	5.3E-07	1.1E-02	NA	2E-03	2E-02
Mercury (inorganic) and compounds	4.5E-04	9.9E-04	6.5E-02	2.8E-02	5.4E-02	1.8E-02	1.5E-04	1.4E-01	NA	4E-04	3E-01
Nickel and compounds	NA	7.3E-07	1.0E-04	4.4E-05	3.2E-04	1.0E-03	1.2E-10	7.3E-04	NA	NA	2E-03
TOTAL HAZARD INDEX	2E-03	1E-03	8E-02	3E-02	5E-02	2E-02	2E-04	2E-01	NA	2E-03	4E-01

NA = Not Applicable.

Table 4.2.5F

SUMMARY OF ESTIMATED MAXIMUM CARCINOGENIC RISKS

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL RISK	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	1.7E-06	NA	NA	NA	NA	NA	NA	NA	NA	2E-06	NA
Arsenic and compounds	4.3E-08	2.1E-09	2.5E-08	1.3E-07	3.1E-08	1.8E-08	1.5E-13	2.1E-07	NA	4E-08	4E-07
Beryllium and compounds	8.2E-11	NA	NA	NA	NA	NA	NA	NA	NA	8E-11	NA
Cadmium and compounds	2.6E-06	NA	NA	NA	NA	NA	NA	NA	NA	3E-06	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	1.5E-11	4.8E-11	2.7E-10	1.4E-09	6.7E-09	6.6E-08	1.3E-14	1.9E-08	NA	2E-11	9E-08
Chlorophenols (treated as 2,4,6-trichlorophenol)	1.3E-11	4.1E-11	2.3E-10	1.1E-09	2.8E-10	1.9E-09	9.8E-14	4.3E-08	NA	1E-11	5E-08
Chromium (VI) and compounds	2.3E-06	NA	NA	NA	NA	NA	NA	NA	NA	2E-06	NA
Nickel and compounds	3.2E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Polychlorinated Biphenyls	1.8E-10	4.5E-11	5.4E-10	2.7E-09	1.3E-08	1.3E-07	5.1E-11	1.9E-08	8.8E-08	2E-10	3E-07
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	5.5E-07	7.9E-07	4.4E-06	2.2E-05	5.3E-04	4.2E-03	3.6E-07	1.1E-04	2.6E-03	5E-07	7E-03
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	1.8E-07	8.6E-08	1.0E-06	5.2E-06	NA	NA	9.0E-10	3.6E-05	NA	2E-07	4E-05
TOTAL CARCINOGENIC RISK	8E-06	9E-07	5E-06	3E-05	5E-04	4E-03	4E-07	1E-04	3E-03	8E-06	8E-03

SUMMARY OF ESTIMATED MAXIMUM HAZARD INDICES

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL HAZARD INDEX	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	2.4E-04	4.7E-12	6.6E-10	2.8E-10	3.5E-11	6.8E-11	8.6E-16	3.8E-09	NA	2E-04	5E-09
Beryllium and compounds	NA	3.0E-11	4.2E-09	1.8E-09	2.2E-10	3.9E-12	1.1E-13	4.1E-09	NA	NA	1E-08
Cadmium and compounds	NA	5.1E-05	7.2E-03	3.1E-03	2.1E-04	7.5E-03	3.6E-07	1.2E-01	NA	NA	1E-01
Chromium (VI) and compounds	NA	1.8E-08	2.6E-06	1.1E-06	7.5E-07	4.0E-06	1.1E-10	2.1E-05	NA	NA	3E-05
Lead and compounds	2.3E-03	1.3E-04	8.2E-03	3.5E-03	4.9E-07	8.0E-06	5.7E-07	1.4E-02	NA	2E-03	3E-02
Mercury (inorganic) and compounds	6.6E-04	1.5E-03	9.5E-02	4.1E-02	1.4E-01	4.4E-02	1.6E-04	1.9E-01	NA	7E-04	5E-01
Nickel and compounds	NA	1.1E-06	1.5E-04	6.5E-05	8.0E-04	2.6E-03	1.3E-10	9.6E-04	NA	NA	5E-03
TOTAL HAZARD INDEX	3E-03	2E-03	1E-01	5E-02	1E-01	5E-02	2E-04	3E-01	NA	3E-03	7E-01

NA = Not Applicable.

Table 4.2.5G

SUMMARY OF ESTIMATED AVERAGE CARCINOGENIC RISKS

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL RISK	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	1.9E-07	NA	NA	NA	NA	NA	NA	NA	NA	2E-07	NA
Arsenic and compounds	4.7E-09	2.3E-10	9.8E-10	2.0E-09	4.8E-09	2.9E-09	1.7E-14	1.9E-08	NA	5E-09	3E-08
Beryllium and compounds	8.9E-12	NA	NA	NA	NA	NA	NA	NA	NA	9E-12	NA
Cadmium and compounds	2.8E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	1.6E-12	5.3E-12	1.1E-11	2.1E-11	1.0E-09	1.0E-08	3.1E-15	2.9E-09	NA	2E-12	1E-08
Chlorophenols (treated as 2,4,6-trichlorophenol)	1.4E-12	4.4E-12	9.0E-12	1.8E-11	4.4E-11	3.0E-10	2.4E-14	6.7E-09	NA	1E-12	7E-09
Chromium (VI) and compounds	2.5E-07	NA	NA	NA	NA	NA	NA	NA	NA	2E-07	NA
Nickel and compounds	3.6E-08	NA	NA	NA	NA	NA	NA	NA	NA	4E-08	NA
Polychlorinated Biphenyls	2.0E-11	1.6E-12	7.1E-12	1.4E-11	6.9E-10	6.8E-09	9.5E-12	2.4E-09	8.9E-10	2E-11	1E-08
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	6.0E-08	2.9E-08	5.8E-08	1.2E-07	2.7E-05	2.2E-04	5.0E-08	8.9E-06	2.3E-05	6E-08	3E-04
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	2.0E-08	3.1E-09	1.4E-08	2.7E-08	NA	NA	1.4E-10	4.7E-06	NA	2E-08	5E-06
TOTAL CARCINOGENIC RISK	8E-07	3E-08	7E-08	1E-07	3E-05	2E-04	5E-08	1E-05	2E-05	8E-07	3E-04

SUMMARY OF ESTIMATED AVERAGE HAZARD INDICES

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL HAZARD INDEX	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	2.7E-05	5.1E-13	2.6E-11	4.4E-12	5.4E-12	1.1E-11	1.0E-16	5.8E-10	NA	3E-05	6E-10
Beryllium and compounds	NA	3.3E-12	1.7E-10	2.8E-11	3.5E-11	6.2E-13	2.5E-14	4.5E-10	NA	NA	7E-10
Cadmium and compounds	NA	5.6E-06	2.9E-04	4.9E-05	3.3E-05	1.2E-03	2.3E-08	1.9E-02	NA	NA	2E-02
Chromium (VI) and compounds	NA	2.0E-09	1.0E-07	1.7E-08	1.2E-07	6.3E-07	5.0E-12	3.3E-06	NA	NA	4E-06
Lead and compounds	2.5E-04	1.4E-05	3.2E-04	5.5E-05	7.6E-08	1.3E-06	3.3E-08	1.9E-03	NA	2E-04	2E-03
Mercury (inorganic) and compounds	7.2E-05	1.6E-04	3.7E-03	6.4E-04	2.1E-02	7.0E-03	1.3E-05	2.6E-02	NA	7E-05	6E-02
Nickel and compounds	NA	1.2E-07	6.0E-06	1.0E-06	1.2E-04	4.1E-04	1.2E-11	1.5E-04	NA	NA	7E-04
TOTAL HAZARD INDEX	3E-04	2E-04	4E-03	7E-04	2E-02	9E-03	1E-05	5E-02	NA	3E-04	8E-02

NA = Not Applicable.

Table 4.2.5H

SUMMARY OF ESTIMATED AVERAGE CARCINOGENIC RISKS

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL RISK	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	2.7E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Arsenic and compounds	6.7E-09	3.2E-10	1.4E-09	2.8E-09	6.8E-09	4.0E-09	1.6E-14	2.7E-08	NA	7E-09	4E-08
Beryllium and compounds	1.3E-11	NA	NA	NA	NA	NA	NA	NA	NA	1E-11	NA
Cadmium and compounds	4.0E-07	NA	NA	NA	NA	NA	NA	NA	NA	4E-07	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	2.3E-12	7.5E-12	1.5E-11	3.0E-11	1.5E-09	1.5E-08	2.8E-15	4.1E-09	NA	2E-12	2E-08
Chlorophenols (treated as 2,4,6-trichlorophenol)	2.0E-12	6.3E-12	1.3E-11	2.5E-11	6.2E-11	4.3E-10	2.2E-14	9.5E-09	NA	2E-12	1E-08
Chromium (VI) and compounds	3.5E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Nickel and compounds	5.0E-08	NA	NA	NA	NA	NA	NA	NA	NA	5E-08	NA
Polychlorinated Biphenyls	2.8E-11	2.3E-12	1.0E-11	2.0E-11	9.8E-10	9.7E-09	8.7E-12	3.5E-09	1.3E-09	3E-11	2E-08
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	8.5E-08	4.1E-08	8.3E-08	1.6E-07	3.8E-05	3.1E-04	4.6E-08	1.3E-05	3.3E-05	8E-08	4E-04
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	2.8E-08	4.4E-09	1.9E-08	3.8E-08	NA	NA	1.3E-10	6.7E-06	NA	3E-08	7E-06
TOTAL CARCINOGENIC RISK	1E-06	5E-08	1E-07	2E-07	4E-05	3E-04	5E-08	2E-05	3E-05	1E-06	4E-04

SUMMARY OF ESTIMATED AVERAGE HAZARD INDICES

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL HAZARD INDEX	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	3.8E-05	7.3E-13	3.7E-11	6.3E-12	7.7E-12	1.5E-11	9.4E-17	8.2E-10	NA	4E-05	9E-10
Beryllium and compounds	NA	4.7E-12	2.4E-10	4.0E-11	4.9E-11	8.8E-13	2.3E-14	6.4E-10	NA	NA	1E-09
Cadmium and compounds	NA	8.0E-06	4.0E-04	6.9E-05	4.7E-05	1.7E-03	2.2E-08	2.7E-02	NA	NA	3E-02
Chromium (VI) and compounds	NA	2.8E-09	1.4E-07	2.4E-08	1.6E-07	8.9E-07	4.6E-12	4.6E-06	NA	NA	6E-06
Lead and compounds	3.5E-04	1.9E-05	4.6E-04	7.8E-05	1.1E-07	1.8E-06	3.1E-08	2.7E-03	NA	4E-04	3E-03
Mercury (inorganic) and compounds	1.0E-04	2.3E-04	5.3E-03	9.1E-04	3.0E-02	9.9E-03	1.2E-05	3.7E-02	NA	1E-04	8E-02
Nickel and compounds	NA	1.7E-07	8.4E-06	1.4E-06	1.8E-04	5.9E-04	1.1E-11	2.1E-04	NA	NA	1E-03
TOTAL HAZARD INDEX	5E-04	3E-04	6E-03	1E-03	3E-02	1E-02	1E-05	7E-02	NA	5E-04	1E-01

NA = Not Applicable.

Table 4.2.5I

SUMMARY OF ESTIMATED MAXIMUM CARCINOGENIC RISKS

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL RISK	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	1.2E-06	NA	NA	NA	NA	NA	NA	NA	NA	1E-06	NA
Arsenic and compounds	2.9E-08	1.4E-09	1.7E-08	8.5E-08	3.4E-10	2.9E-11	9.7E-12	2.4E-08	NA	3E-08	1E-07
Beryllium and compounds	5.5E-11	NA	NA	NA	NA	NA	NA	NA	NA	6E-11	NA
Cadmium and compounds	1.8E-06	NA	NA	NA	NA	NA	NA	NA	NA	2E-06	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	1.0E-11	3.3E-11	1.8E-10	9.3E-10	7.4E-11	1.1E-10	8.4E-13	3.5E-10	NA	1E-11	2E-09
Chlorophenols (treated as 2,4,6-trichlorophenol)	8.6E-12	2.8E-11	1.5E-10	7.8E-10	3.1E-12	3.1E-12	6.4E-12	4.3E-10	NA	9E-12	1E-09
Chromium (VI) and compounds	1.5E-06	NA	NA	NA	NA	NA	NA	NA	NA	2E-06	NA
Nickel and compounds	2.2E-07	NA	NA	NA	NA	NA	NA	NA	NA	2E-07	NA
Polychlorinated Biphenyls	1.2E-10	3.0E-11	3.7E-10	1.8E-09	1.5E-10	2.1E-10	3.4E-09	5.9E-10	3.5E-09	1E-10	1E-08
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	3.7E-07	5.4E-07	3.0E-06	1.5E-05	5.8E-06	6.8E-06	2.4E-05	4.6E-06	3.3E-05	4E-07	9E-05
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	1.2E-07	5.8E-08	7.0E-07	3.5E-06	NA	NA	5.9E-08	1.1E-06	NA	1E-07	5E-06
TOTAL CARCINOGENIC RISK	5E-06	6E-07	4E-06	2E-05	6E-06	7E-06	2E-05	6E-06	3E-05	5E-06	1E-04

SUMMARY OF ESTIMATED MAXIMUM HAZARD INDICES

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL HAZARD INDEX	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	1.6E-04	3.2E-12	4.5E-10	1.9E-10	3.9E-13	1.1E-13	5.7E-14	7.2E-11	NA	2E-04	7E-10
Beryllium and compounds	NA	2.0E-11	2.9E-09	1.2E-09	2.5E-12	6.4E-15	6.9E-12	3.5E-10	NA	NA	4E-09
Cadmium and compounds	NA	3.5E-05	4.9E-03	2.1E-03	2.3E-06	1.2E-05	2.4E-05	1.3E-03	NA	NA	8E-03
Chromium (VI) and compounds	NA	1.2E-08	1.7E-06	7.5E-07	8.3E-09	6.4E-09	7.0E-09	3.1E-07	NA	NA	3E-06
Lead and compounds	1.5E-03	8.5E-05	5.6E-03	2.4E-03	5.4E-09	1.3E-08	3.7E-05	7.2E-04	NA	2E-03	9E-03
Mercury (inorganic) and compounds	4.5E-04	9.9E-04	6.5E-02	2.8E-02	1.5E-03	7.2E-05	1.0E-02	8.5E-03	NA	4E-04	1E-01
Nickel and compounds	NA	7.3E-07	1.0E-04	4.4E-05	8.9E-06	4.2E-06	8.3E-09	1.7E-05	NA	NA	2E-04
TOTAL HAZARD INDEX	2E-03	1E-03	8E-02	3E-02	2E-03	9E-05	1E-02	1E-02	NA	2E-03	1E-01

NA = Not Applicable.

Table 4.2.5J

SUMMARY OF ESTIMATED MAXIMUM CARCINOGENIC RISKS

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL RISK	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	1.7E-06	NA	NA	NA	NA	NA	NA	NA	NA	2E-06	NA
Arsenic and compounds	4.3E-08	2.1E-09	2.5E-08	1.3E-07	8.5E-10	7.3E-11	1.0E-11	3.1E-08	NA	4E-08	2E-07
Beryllium and compounds	8.2E-11	NA	NA	NA	NA	NA	NA	NA	NA	8E-11	NA
Cadmium and compounds	2.6E-06	NA	NA	NA	NA	NA	NA	NA	NA	3E-06	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	1.5E-11	4.8E-11	2.7E-10	1.4E-09	1.9E-10	2.7E-10	8.9E-13	4.6E-10	NA	2E-11	3E-09
Chlorophenols (treated as 2,4,6-trichlorophenol)	1.3E-11	4.1E-11	2.3E-10	1.1E-09	7.8E-12	7.8E-12	6.8E-12	5.7E-10	NA	1E-11	2E-09
Chromium (VI) and compounds	2.3E-06	NA	NA	NA	NA	NA	NA	NA	NA	2E-06	NA
Nickel and compounds	3.2E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Polychlorinated Biphenyls	1.8E-10	4.5E-11	5.4E-10	2.7E-09	3.7E-10	5.3E-10	3.6E-09	7.8E-10	4.9E-09	2E-10	1E-08
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	5.5E-07	7.9E-07	4.4E-06	2.2E-05	1.5E-05	1.7E-05	2.5E-05	6.1E-06	5.3E-05	5E-07	1E-04
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	1.8E-07	8.6E-08	1.0E-06	5.2E-06	NA	NA	6.3E-08	1.5E-06	NA	2E-07	8E-06
TOTAL CARCINOGENIC RISK	8E-06	9E-07	5E-06	3E-05	1E-05	2E-05	3E-05	8E-06	5E-05	8E-06	2E-04

SUMMARY OF ESTIMATED MAXIMUM HAZARD INDICES

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL HAZARD INDEX	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	2.4E-04	4.7E-12	6.6E-10	2.8E-10	9.7E-13	2.8E-13	6.0E-14	9.4E-11	NA	2E-04	1E-09
Beryllium and compounds	NA	3.0E-11	4.2E-09	1.8E-09	6.2E-12	1.6E-14	7.3E-12	4.6E-10	NA	NA	7E-09
Cadmium and compounds	NA	5.1E-05	7.2E-03	3.1E-03	5.9E-06	3.0E-05	2.5E-05	1.7E-03	NA	NA	1E-02
Chromium (VI) and compounds	NA	1.8E-08	2.6E-06	1.1E-06	2.1E-08	1.6E-08	7.4E-09	4.1E-07	NA	NA	4E-06
Lead and compounds	2.3E-03	1.3E-04	8.2E-03	3.5E-03	1.4E-08	3.2E-08	3.9E-05	9.5E-04	NA	2E-03	1E-02
Mercury (inorganic) and compounds	6.6E-04	1.5E-03	9.5E-02	4.1E-02	3.8E-03	1.8E-04	1.1E-02	1.1E-02	NA	7E-04	2E-01
Nickel and compounds	NA	1.1E-06	1.5E-04	6.5E-05	2.2E-05	1.1E-05	8.8E-09	2.2E-05	NA	NA	3E-04
TOTAL HAZARD INDEX	3E-03	2E-03	1E-01	5E-02	4E-03	2E-04	1E-02	1E-02	NA	3E-03	2E-01

NA = Not Applicable.

Table 4.2.5K

SUMMARY OF ESTIMATED AVERAGE CARCINOGENIC RISKS

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL RISK	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	1.9E-07	NA	NA	NA	NA	NA	NA	NA	NA	2E-07	NA
Arsenic and compounds	4.7E-09	2.3E-10	9.8E-10	2.0E-09	1.3E-10	1.2E-11	1.2E-12	2.5E-09	NA	5E-09	6E-09
Beryllium and compounds	8.9E-12	NA	NA	NA	NA	NA	NA	NA	NA	9E-12	NA
Cadmium and compounds	2.8E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	1.6E-12	5.3E-12	1.1E-11	2.1E-11	2.9E-11	4.2E-11	2.1E-13	4.5E-11	NA	2E-12	2E-10
Chlorophenols (treated as 2,4,6-trichlorophenol)	1.4E-12	4.4E-12	9.0E-12	1.8E-11	1.2E-12	1.2E-12	1.6E-12	6.7E-11	NA	1E-12	1E-10
Chromium (VI) and compounds	2.5E-07	NA	NA	NA	NA	NA	NA	NA	NA	2E-07	NA
Nickel and compounds	3.6E-08	NA	NA	NA	NA	NA	NA	NA	NA	4E-08	NA
Polychlorinated Biphenyls	2.0E-11	1.6E-12	7.1E-12	1.4E-11	1.9E-11	2.8E-11	6.6E-10	3.4E-11	3.9E-11	2E-11	8E-10
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	6.0E-08	2.9E-08	5.8E-08	1.2E-07	7.4E-07	8.9E-07	3.4E-06	2.0E-07	3.5E-07	6E-08	6E-06
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	2.0E-08	3.1E-09	1.4E-08	2.7E-08	NA	NA	9.7E-09	6.4E-08	NA	2E-08	1E-07
TOTAL CARCINOGENIC RISK	8E-07	3E-08	7E-08	1E-07	7E-07	9E-07	3E-06	3E-07	4E-07	8E-07	6E-06

SUMMARY OF ESTIMATED AVERAGE HAZARD INDICES

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL HAZARD INDEX	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	2.7E-05	5.1E-13	2.6E-11	4.4E-12	1.5E-13	4.4E-14	7.1E-15	9.3E-12	NA	3E-05	4E-11
Beryllium and compounds	NA	3.3E-12	1.7E-10	2.8E-11	9.6E-13	2.5E-15	1.7E-12	3.7E-11	NA	NA	2E-10
Cadmium and compounds	NA	5.6E-06	2.9E-04	4.9E-05	9.1E-07	4.8E-06	1.6E-06	2.1E-04	NA	NA	6E-04
Chromium (VI) and compounds	NA	2.0E-09	1.0E-07	1.7E-08	3.2E-09	2.5E-09	3.5E-10	4.3E-08	NA	NA	2E-07
Lead and compounds	2.5E-04	1.4E-05	3.2E-04	5.5E-05	2.1E-09	5.1E-09	2.3E-06	7.9E-05	NA	2E-04	5E-04
Mercury (inorganic) and compounds	7.2E-05	1.6E-04	3.7E-03	6.4E-04	5.9E-04	2.8E-05	9.0E-04	9.4E-04	NA	7E-05	7E-03
Nickel and compounds	NA	1.2E-07	6.0E-06	1.0E-06	3.4E-06	1.7E-06	8.0E-10	2.2E-06	NA	NA	1E-05
TOTAL HAZARD INDEX	3E-04	2E-04	4E-03	7E-04	6E-04	3E-05	9E-04	1E-03	NA	3E-04	8E-03

NA = Not Applicable.

Table 4.2.5L

SUMMARY OF ESTIMATED AVERAGE CARCINOGENIC RISKS

CHEMICAL EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL RISK	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Aldehydes (treated as formaldehyde	2.7E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Arsenic and compounds	6.7E-09	3.2E-10	1.4E-09	2.8E-09	1.9E-10	1.6E-11	1.1E-12	3.5E-09	NA	7E-09	8E-09
Beryllium and compounds	1.3E-11	NA	NA	NA	NA	NA	NA	NA	NA	1E-11	NA
Cadmium and compounds	4.0E-07	NA	NA	NA	NA	NA	NA	NA	NA	4E-07	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	2.3E-12	7.5E-12	1.5E-11	3.0E-11	4.1E-11	5.9E-11	2.0E-13	6.4E-11	NA	2E-12	2E-10
Chlorophenols (treated as 2,4,6-trichlorophenol)	2.0E-12	6.3E-12	1.3E-11	2.5E-11	1.7E-12	1.7E-12	1.5E-12	9.5E-11	NA	2E-12	1E-10
Chromium (VI) and compounds	3.5E-07	NA	NA	NA	NA	NA	NA	NA	NA	3E-07	NA
Nickel and compounds	5.0E-08	NA	NA	NA	NA	NA	NA	NA	NA	5E-08	NA
Polychlorinated Biphenyls	2.8E-11	2.3E-12	1.0E-11	2.0E-11	2.7E-11	3.9E-11	6.1E-10	4.7E-11	4.3E-11	3E-11	8E-10
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	8.5E-08	4.1E-08	8.3E-08	1.6E-07	1.0E-06	1.3E-06	3.2E-06	2.8E-07	4.2E-07	8E-08	6E-06
Polycyclic Aromatic Hydrocarbons (treated as benzo[a]pyrene)	2.8E-08	4.4E-09	1.9E-08	3.8E-08	NA	NA	9.0E-09	9.1E-08	NA	3E-08	2E-07
TOTAL CARCINOGENIC RISK	1E-06	5E-08	1E-07	2E-07	1E-06	1E-06	3E-06	4E-07	4E-07	1E-06	7E-06

SUMMARY OF ESTIMATED AVERAGE HAZARD INDICES

CHEMICAL EXHIBITING POTENTIAL NON-CARCINOGENIC EFFECTS	INHALATION PATHWAY	INGESTION PATHWAYS								TOTAL HAZARD INDEX	
		ING. VIA INHALATION	DUST	SOIL	BEEF	DAIRY MILK	FISH	LOCALLY GROWN PRODUCE	HUMAN BREAST-MILK	INHALATION ROUTE	INGESTION ROUTES
Antimony and compounds	3.8E-05	7.3E-13	3.7E-11	6.3E-12	2.1E-13	6.2E-14	6.5E-15	1.3E-11	NA	4E-05	6E-11
Beryllium and compounds	NA	4.7E-12	2.4E-10	4.0E-11	1.4E-12	3.6E-15	1.6E-12	5.2E-11	NA	NA	3E-10
Cadmium and compounds	NA	8.0E-06	4.0E-04	6.9E-05	1.3E-06	6.8E-06	1.5E-06	3.0E-04	NA	NA	8E-04
Chromium (VI) and compounds	NA	2.8E-09	1.4E-07	2.4E-08	4.5E-09	3.6E-09	3.2E-10	6.1E-08	NA	NA	2E-07
Lead and compounds	3.5E-04	1.9E-05	4.6E-04	7.8E-05	3.0E-09	7.2E-09	2.1E-06	1.1E-04	NA	4E-04	7E-04
Mercury (inorganic) and compounds	1.0E-04	2.3E-04	5.3E-03	9.1E-04	8.3E-04	4.0E-05	8.3E-04	1.3E-03	NA	1E-04	9E-03
Nickel and compounds	NA	1.7E-07	8.4E-06	1.4E-06	4.9E-06	2.4E-06	7.4E-10	3.1E-06	NA	NA	2E-05
TOTAL HAZARD INDEX	5E-04	3E-04	6E-03	1E-03	8E-04	5E-05	8E-04	2E-03	NA	5E-04	1E-02

NA = Not Applicable.

Table 4.2.50

COMPARISON OF PREDICTED AIR CONCENTRATIONS
WITH NATIONAL AMBIENT AIR QUALITY STANDARDS

Chemical	Maximum Predicted Air Concentration (ug/m^3)[a]	Primary National Ambient Air Quality Standard (ug/m^3)	
Sulfur dioxide			
Riverview	2.0	80	(Annual)
Riverbend	3.0		
Riverview	18.3	365	(24-hour)
Riverbend	24.4		
Nitrogen Oxides			
Riverview	3.5	100	(Annual)
Riverbend	5.1		
Carbon Monoxide			
Riverview	26.7	10,000	(8-hour)
Riverbend	39.4		
Riverview	72.3	40,000	(1-hour)
Riverbend	94.1		
Particulate Matter			
Riverview	0.8	50	(Annual)
Riverbend	1.2		
Riverview	7.2	150	(24-hour)
Riverbend	9.7		

[a]The maximum concentrations were predicted based on the following maximum generic air concentrations predicted for the Riverview and Riverbend sites, respectively, for a 1 g/sec emission rate for one boiler unit: 0.51 and 0.75 ug/m^3 annual average; 4.57 and 6.1 ug/m^3 24-hour average; 8.34 and 12.3 ug/m^3 8-hour average; 22.6 and 29.4 ug/m^3 1-hour average.

Table 4.2.5P

COMPARISON OF BACKROUND AMBIENT AIR CONCENTRATIONS WITH MAXIMUM PREDICTED AIR CONCENTRATIONS FOR CHEMICALS EMITTED FROM THE PROPOSED WINONA RESOURCE RECOVERY FACILITY

Compound	Maximum Predicted Annual Average Air Concentrations (ug/m3)		Backround Ambient Air Concentration Range (ug/m3) (a)
	Riverview Drive Site	Riverbend II Site	
Aldehydes (treated as formaldehyde)	1.73E-02	2.55E-02	3.4 - 18.0
Antimony and compounds	6.36E-04	9.36E-04	NA
Arsenic and compounds	1.12E-04	1.65E-04	0.0029 - 0.0041
Beryllium and compounds	3.09E-07	4.54E-07	0.13 - 0.24
Cadmium and compounds	1.00E-03	1.47E-03	0.001 - 0.002
Carbon Monoxide	1.63E+00	2.40E+00	NA
Chlorobenzenes (treated as 1,4-dichlorobenzene)	1.08E-05	1.59E-05	NA
Chlorophenols (treated as 2,4,6-trichlorophenol)	2.55E-05	3.75E-05	NA
Chromium (VI) and compounds	1.11E-03	1.64E-03	0.52 - 0.64
Hydrogen Chloride	2.45E+00	3.60E+00	NA
Lead and compounds	2.76E-02	4.06E-02	0.11 - 0.58
Mercury (inorganic) and compounds	3.57E-03	5.25E-03	NA
Nickel and compounds	7.83E-04	1.15E-03	0.001 - 0.017
Nitrogen Oxides	3.47E+00	5.10E+00	8 - 39
Particulate Matter	8.09E-01	1.19E+00	20 - 100
Polychlorinated Biphenyls (PCBs)	6.39E-06	9.39E-06	NA
PCDDs/PCDFs (expressed as 2,3,7,8-TCDD equivalents)	2.53E-08	3.72E-08	NA
Polycyclic Aromatic Hydrocarbons (treated as benzo(a)pyrene)	5.17E-03	7.60E-03	0.13 - 0.93
Sulfur Oxides (treated as SO2)	2.04E+00	3.00E+00	2.62 - 23.6

(a) See Section V for discussion and sources.

NA = Not Available.

4.2.6. ECOLOGICAL RISK ASSESSMENT

4.2.6.1 OBJECTIVE

The construction and operation of the proposed incinerator at Winona may impact fish, wildlife, and plants by directly removing available habitat and by increasing exposure to chemical pollutants in the system. The objective of this ecological risk assessment is to assess these two types of impacts by identifying the receptors potentially impacted (either habitat or fish, wildlife and plants) and estimating the loss.

Although absolute conclusions regarding the potential environmental impacts of the proposed incinerator can not be made, several conclusions about the potential for endangerment of fish, wildlife, and their habitat in the Winona area can be drawn with reasonable scientific certainty.

- Construction of the proposed incinerator at either site will result in the loss of wildlife habitat. However, construction at the Riverbend II site will result in the loss of four acres of wetland habitat whereas no wetlands exist at the Riverview construction site. Because wetlands are diminishing rapidly on a national scale and because these systems are of high value to fish and wildlife, construction at the Riverbend II site is estimated to have a greater impact on the environment (based on habitat loss) than construction at the Riverview site.

- The predicted concentrations of chemicals in the surface water are substantially below those associated with any toxic effects in fish, and therefore significant impacts on resident fish species are not likely .

- The predicted concentrations of contaminants in the animal diet of ducks and eagles also are substantially (5 to 11 orders of magnitude) below the levels known to cause toxic responses in these or similar species and adverse effects via the animal diet are not likely. Exposure via plant food, water, air, through the skin, and while preening will contribute to intake but most likely not to levels associated with adverse effects.

Conclusions can not be drawn regarding potential impacts to threatened and special concern plants of the area. However, soil concentrations of mercury and cadmium may be at sufficiently high levels to be toxic to some plant species.

4.2.6.2 DIRECT HABITAT LOSS FROM INCINERATOR CONSTRUCTION

The assessment of direct habitat loss as a result of construction requires an identification of the amount and type of habitat potentially impacted and the value of that habitat to fish and wildlife. Two 6-acre sites have been proposed as possible locations for the incinerator. The Riverview site is located in the northern portion of the city in a relatively developed area. According to EAW, construction at this site would eliminate 6 acres of brush/grassland habitat. The Riverbend II site is located in the southeastern portion of the city in a relatively undeveloped area. Construction at this site would eliminate 2 acres of brush/grassland habitat and 4 acres of wetland habitat. At either site, construction would result in the creation of 4.2 acres of impervious surface and 1.8 acres of landscaping.

The type of vegetation comprising the habitat at the Riverview site has not been completely described, but it is likely that the area is covered by a variety of shrubs including red and green dogwood (Cornus spp.), poison ivy (Rhus radicans), button bush (Cephalanthus occidentalis), and some patches of silver maple (Acer saccharinum) and cottonwood (Populus deltoides). Grasses at the site likely include big and little bluestem (Andropogon spp).

The shrub/grasslands at the Riverbend II site are likely to be similar to those at the Riverview site. The wetlands at the site are comprised of herbaceous marshes. Typical species of this type of habitat in the Winona area include rice cutgrass (Leersea sp.), a variety of sedges (Cyperus spp.), cattail (Typha latifolia), Phragmites, wild millet (Milium effusum), smartweed (Polygonum occidentalis), river bulrush (Scirpus sp.), and wild rice (Zizania aquatica).

Each of these types of habitat is valuable to wildlife. For example, shrub/grassland areas typically provide nesting and cover sites for a variety of small mammals, including field mice (Peromyscus spp.), shrews (Blarina brevicauda), moles (Scalopus aquaticus), chipmunk (Tamias striatus), ground squirrels (Cetullus spp.), rabbits (Sylvilagus aquaticus), and woodchucks (Marmota monax). Where the site borders the water, muskrat (Ondatra zibethicus), racoon (Procyon lotor) and mink (Mustela vison) may occur. The small mammal populations of mice, chipmunks, and ground squirrels are likely food sources for many raptor species in the area, including red-tailed hawk (Buteo jamaicensis), marsh hawk (Circus cyaneus), and a variety of owls (Otus asio, Bubo virginianus, Strix spp.). The shrub/grasslands also may provide habitat for a variety of resident and migratory song birds. Further, although the Riverview site is located in a relatively developed area, its proximity to the Upper Mississippi Wildlife and Fish Refuge ensures its use by some of the bird species on the Refuge.

Wetlands such as those at the Riverbend II site are highly productive and diverse systems and constitute habitat of high value to fish and wildlife species. Freshwater wetlands are essential to maintaining many fish populations; in fact, many freshwater fish can be considered wetland dependent because these species feed in wetlands or on wetland dependent food and use wetlands as nursery or spawning grounds (Tiner 1984). For example, almost all important recreational fish in northern areas [e.g., northern pike (Esox lucius), carp (Cyprinus carpio), smallmouth bass (Micropterus dolemieui), bluegill (Lepomis machrochirus)] use wetlands to spawn.

In addition to their value to fish, wetlands are valuable to birds and provide year-round habitat for resident birds, as well as breeding and feeding grounds and over-wintering areas for migratory waterfowl, passerine and wading birds, and raptors. Wetlands also provide cover, forage, resting and rearing habitat for a variety of furbearers (mink, racoon, muskrat, beaver (Castor canadensis), fox (Urocynon sp.)), small mammals (rabbits, mice, shrews), reptiles and amphibians.

Wetlands also serve a valuable secondary function by filtering sediments and pollutants from adjacent upland areas. This protects adjacent open water areas from excessive pollutant and sediments loads, and thereby maintains the value of the fishery and aquatic habitat. Construction of the incinerator at either of the proposed sites will result in the direct loss of valuable wildlife habitat, and potentially may result in secondary impacts to adjacent aquatic areas. Construction at the Riverview site will result in the loss of 4 acres of shrub/grassland habitat, and construction at the Riverbend II site will result in the loss of 2 acres of shrub/grassland and 4 acres of marsh

wetlands. In addition, the open water areas adjacent to the Riverbend II site may be secondarily impacted by the wetland loss via increased pollutant and sediment loads from surrounding upland areas.

Wetlands are diminishing rapidly on a national scale. For this reason, and because wetlands are highly productive systems of extreme value to fish and wildlife species, loss of wetland habitat is generally regarded as having greater impact on the environment than losses of other types of habitat. Therefore, construction at the Riverbend II site and the elimination of 4 acres of wetland habitat would have a greater impact on the environment than construction at the Riverview site.

4.2.6.3. CHEMICAL-INDUCED EFFECTS ON FISH, WILDLIFE, AND PLANTS

Assessment of impacts following exposure of fish, wildlife and plants to chemical pollutants requires identification of potential receptors, assessment of receptor-specific toxicity and exposure, and interpretation of toxicity and exposure to generate estimates of risk. Because natural systems are comprised of individual organisms, local populations, and communities, risks to each of these components should be assessed. However, because of the large number of species and communities present in natural systems, such ecosystem-wide assessments are very complex, and appropriate assessment methodologies have not yet been developed. In addition, dose-response data are generally limited to individual- and population-level responses. Therefore, ecological assessments are usually limited to the individual and population level and are often qualitative in nature.

In the following sections we asses potential impacts to fish, wildlife and plants at the individual and population level. In Section XI.A, the receptors potentially affected by chemicals emitted from the incinerator at Winona are identified. In Section XI.B TWP, the available toxicity data for each chemical is discussed. Potential exposure routes are identified and exposure is quantified in Section XI.C. TWP. Finally, in Section XI.D TWP, the toxicity information is combined with the exposure to provide an estimate of risk.

4.2.6.4 RECEPTOR CHARACTERIZATION

The area surrounding the Winona area is ecologically diverse due to its location in a sheltered river floodplain and its north-south expanse along bird migratory routes. The area is characterized by shallow and open water habitats, a variety of wetlands, forested hardwood stands, bluff communities, and shrub and grassland habitats. Because of this diversity of habitat, the fish, wildlife and plant species of the area are diverse and numerous. However, because of this diversity, it is not feasible to assess impacts to all receptors in the Winona area. Therefore, we have selected representative species in both aquatic and terrestrial habitats as indicators of potential impacts in those systems. Resident fish of the Mississippi River around Winona are identified as receptors for aquatic systems, and resident waterfowl are selected as representative species for the terrestrial systems. A third category of receptors, endangered and threatened species, also is selected for more detailed evaluation. Below, we identify the particular species selected as representatives within each category.

4.2.6.4.1. Resident Waterfowl

The wetlands of the Winona area provide ideal habitat for waterfowl. During winter migration, the area host a large variety of waterfowl including canvasback ducks (Aythya valisineria), scaup (Aythya affinis), ring-necked

ducks (Athya collaris), blue- and green-winged teal (Anas discors and Anas carolinensis, respecitvely), mallards (Anas platyrynchos), wood ducks (Aix sponsa), black ducks (Anas rubripes), shovelers (Spatula clypeata), Canada geese (Branta canadensis), and tundra swans (Cygnus columbianus). Most of these species remain in the area for up to two months before continuing their southward migration. However, wood ducks, mallards, blue-winged teal, black duck, shovelers, and Canada geese also are year-round residents of the Winona area. Mallards, blue-winged teal, and wood ducks comprise over 90% of the resident waterfowl population (USFWS 1987).

The resident duck species use the wetlands in the area extensively; mallards and blue-winged teal primarily use the marshes and wood ducks use the smaller pools and wooded areas. The ducks rely primarily on the seeds and leaves of wetland plants for food, but also consume animal life (insects, crustaceans, snails), particularly during the spring and summer months when insect larvae are abundant. Of these species, blue-winged teal consume the highest proportion of animal food, with 30% of the diet comprised of nonvegetative matter (Terres 1980). Approximately 10 to 20% of the mallard's diet is animal matter (Martin et al. 1961) as is 10% of the wood duck's diet (Terres 1980).

4.2.6.4.2. Resident Fish Species In the Winona Area

Because the river around Winona contains so many different habitats, the area supports a varied fish fauna, including a number of sport and commercial fish species. According to information provided by the Minnesota Department of Natural Resources (DNR 1979) on fish harvest for pool 5 near Winona, crappie (Pomoxis spp.) and sunfish (Lepomis spp.) are two of the major sport and commercial fish. Other game fish include largemouth and smallmouth bass (Micropterus salmoides and Micropterus dolemieui), white bass (Morone chrysops), walleye (Stizostedian vitreum), northern pike (Esox lucius), perch (Morono americana and Perca flavescens), and sauger (Stizostedian canadense). Carp (Cyprinus carpio) and channel catfish (Ictalurus punctatus) are other species that commonly occur in the area.

4.2.6.4.3. Endangered and Threatened Species

The Winona area hosts a large number of rare species classified by the state or Federal government as either endangered, threatened, or of special concern. The Minnesota Natural Heritage Program provided a listing of the designated plant and animal species that occur within a 5 mile radius of Winona. These are presented in Table 4.2.2a along with the designated status and a description of the general types of habitat in which they occur. Because of their position at the top of the food chain, bald eagles (Haliaeetus leucocephalus) and peregrine falcons (Falco peregrinus) may be particularly susceptible to toxic effects from chemicals that tend to bioconcentrate and biomagnify through the food chain. Bald eagles rely largely on fish as a food source, and in the Winona area, carp form a large percentage of the diet (USFWS 1987). Peregrine falcons consume a variety of birds including ducks, grebes (Podiceps spp.), snipes (Capella gallinago), woodcock (Philohela minor), herons (Ardeidae), coots (Fulicia americana), and rails (Rallus sp.). In the Winona area, peregrine falcons feed largely on domestic pigeons (Columbia livia), although the other species listed above are sure to comprise some portion of the diet (USFWS 1987).

4.2.6.5 HAZARD ASSESSMENT

Chemical compounds released from the incinerator at Winona may be toxic to

plants and animals exposed to these substances via air, water, soil, sediment or food. Unfortunately, the available toxicity information is not adequate to assess potential toxic effects of all chemicals for each receptor via each of these pathways. Therefore, this hazard assessment is necessarily limited to a discussion of the toxic effects of a subset of chemicals for a subset of receptors and pathways.

The chemicals of concern in this hazard assessment are the same chemicals evaluated for potential impacts to human health. We have structured our hazard assessment around the potential toxicity of the indicator chemicals to the selected receptors identified in the previous section. In particular, the aquatic toxicity assessment focusses on the effects on fish, with emphasis on those fish species resident to Winona. The terrestrial toxicity assessment focusses on the effects on birds, in particular, the effects on resident ducks, peregrine falcons and bald eagles. Potential toxicity of the indicator chemicals to the other terrestrial animal receptors which were identified (blue racer and timber rattle snake) are not discussed because adequate data on the toxicity of the indicator chemicals to these or similar species are not available. The final receptors which were identified are the threatened and special concern plants of the Winona area. Although toxicity information specific to these species is not available, the toxicity of the indicator chemicals to plants in general will be discussed.

4.2.6.5.1. Aquatic Toxicity - Fish

The potential toxicity to fish of the chemicals of concern in surface water can be assessed using ambient water quality criteria (AWQC) developed by the USEPA. These criteria are developed to be protective of 95% of all aquatic species. Therefore, not only are fish protected, but aquatic invertebrates and plants are protected as well. The criteria can therefore be regarded as ambient levels that are safe for fish as well as fish food and habitat. Although the criteria are not specific to the aquatic species at Winona, they should be sufficiently protective of those species.

The ambient water quality criteria for each of the chemicals of concern are presented in Table 4.2.2b. For chemicals for which no AWQC exist, toxic levels identified in the literature for fish species common to Winona are listed in Table 4.2.2c. Below, the toxicity of all chemicals of concern is discussed briefly. Emphasis is placed on the AWQC and toxicity information pertinent to fish species resident of Winona.

Antimony. Few data are available to assess the toxicity of antimony to freshwater organisms, and no AWQC has been developed. However, the available data base for antimony indicates that plants may be more sensitive than fish or invertebrate species. No toxicity tests have been conducted using fish species common to the Winona area. But in acute tests with fathead minnows (Pimephales promelas), a 96-hour median lethal concentration (LC_{50}) of 21,900 ug/liter was reported (USEPA 1980a). In early life stage tests with the same species, a chronic toxicity value of 1,600 ug/liter was reported (USEPA 1980a).

Arsenic. The toxicity of arsenic to freshwater aquatic organisms is similar for both the trivalent and pentavalent forms. Although the exact mechanism of arsenic toxicity is not known, the main pathological effect appears to be enzyme inhibition. USEPA (1984a) has established for arsenic (III) a chronic criterion of 190 ug/liter and a 1-hour average concentration criterion of 360 ug/liter not to be exceeded more than once every 3 years. No criteria for arsenic (V) have been established because of insufficient data. In

acute assays involving species common to Winona, a 96-hour LC_{50} of 15,400 ug/liter was reported for bluegills (Lepomis machrochirus) (USEPA 1984a).

Beryllium. No AWQC has been developed for beryllium because adequate data are not available. Some of the data indicate that beryllium toxicity is inversely related to water hardness, but this relationship has not been quantitatively defined. In acute lethality tests with bluegills in hard (400 mg/l as $CaCO_3$) and soft (20 mg/liter) waters, 96-hour LC_{50} values of 12,000 and 1,300 ug/liter, respectively, were reported (USEPA 1980b). The acute median lethal toxicity of beryllium to bluegills in the Winona area, where the average water hardness is 200 mg/l (as $CaCO_3$), is likely to be between these two values. No chronic toxicity data are available for any fish species.

Cadmium. Cadmium is a cumulative poison to aquatic organisms because excretion of this metal is virtually nonexistent. Studies reporting cadmium toxicity to aquatic organisms demonstrate an inverse relationship between cadmium toxicity and water hardness. USEPA (1984b) recommended a 4-day average concentration criterion for cadmium not to exceed the value given by $e^{(0.7852(\ln \text{hardness}))-3.490)}$ and a 1-hour average concentration criterion not to exceed the value given by $e^{(1.128(\ln(\text{hardness}))-3.828)}$ more than once every 3 years. Using an average water hardness of 200 mg/liter (as $CaCO_3$) at Winona, the corresponding 4-day and 1-hour concentrations are 2.0 and 8.6 ug/liter, respectively.

In acute and chronic assays conducted at a similar water hardness and using fish species common to Winona, a 96-hour LC_{50} value of 21,100 ug/liter was reported for bluegills, and a chronic value of 143 ug/liter was reported for channel catfish (Ictalurus puntatus) (USEPA 1984b).

Chromium. For freshwater aquatic organisms, chromium (VI) appears to be more toxic than chromium (III). Impaired growth, increased locomotor activity, and enzyme inhibition have been reported in fish following chronic exposure to chromium. USEPA (1984c) has established a continuous concentration criterion for chromium (VI) of 11 ug/liter and a 1-hour average criterion of 16 ug/liter not to be exceeded more than once every three years. A species mean acute value of 133,000 ug/liter has been reported for bluegills, a Winona species (USEPA 1984c).

1,4- Dichlorobenzene. Data on the toxicity of 1,4-dichlorobenzene to freshwater organisms is limited, and no AWQC has been developed for this chemical. In acute lethality tests with bluegills, a 96-h LC_{50} of 4,280 ug/liter has been reported (USEPA 1980d).

Lead. The primary mechanism of acute toxicity of lead to freshwater organisms is unknown. Lead toxicity decreases with increasing water hardness. USEPA (1985) has established 4-day and 1-hour average concentration criteria for lead not to be exceeded by the values given by $e^{(1.266(\ln(\text{hardness}))-4.661)}$ and $e^{(1.266(\ln(\text{hardness}))-1.416)}$, respectively, more than once every three years. Using the average water hardness at Winona of 200 mg/liter, the 4-day and 1-hour criteria correspond to 7.7 and 200 ug/liter, respectively. No toxicity data on Winona species at a similar hardness were located in the literature searched.

Mercury. Mercury is acutely and chronically toxic to freshwater fish. Signs of acute mercury poisoning in fish include flaring of gill covers, increased frequency of respiratory movements, loss of equilibrium, and sluggishness. Chronic poisoning can cause emaciation, brain lesions, cataracts, inability to capture food, and abnormal motor coordination. USEPA

(1984d) has established 4-day and 1-hour concentration criteria of 0.012 and 2.4 ug/liter. In acute lethality assays on species common to the Winona area, a 10-day LC_{50} of 0.3 ug/liter has been reported for channel catfish, and an 8-day LC_{50} of 5.3 ug/liter has been reported for largemouth bass (Micropoterus salmoides) (Eisler 1987a).

Nickel. The adverse effects of nickel in aquatic organisms include alteration of cell membranes, formation of precipitates on gills, hematological effects and reproductive impairment. Toxicity of nickel to freshwater organisms decreases with increasing water hardness. USEPA (1986) has proposed 4-day and 1-hour concentration criteria not to exceed the value given by $e^{(0.8460(\ln(\text{hardness}))+1.1645)}$ and $e^{(0.8460(\ln(\text{hardness}))+3.3612)}$, respectively more than once every 3 years. Using the average water hardness of 200 mg/liter at Winona, the 4-day and 1-hour criteria correspond to 280 and 2,500 ug/liter, respectively. No toxicity data on Winona species at a similar hardness were located in the literature searched.

PAHs. Information on the toxicity of PAHs to aquatic life is extremely limited and the available data are not sufficient to develop an AWQC. However, it is known that carcinogenic PAHs can produce cancer like-growths, teratogenesis, and mutagenesis in aquatic life. Schultz and Schultz (1982) reported liver neoplasms in two species of minnows (Poeiciliopsis spp.) exposed 6 hours per week for 5 weeks to an aqueous suspension of 5,00 ug/liter of 7,12-dimethylbenz(a)pyrene.

PCBs. In fish, PCBs have caused death, behavioral abnormalities, increased locomotor activity, decreased success in capturing food, neurochemical alterations, disrupted osmoregulation, and liver and thyroid effects. Reproductive effects also have been linked to PCB exposure. USEPA (1980e) has established 4-day and 1-hour concentration criterion for PCBs in freshwater of 0.014 and 2.0 ug/liter not to be exceeded more than once every 3 years. These criteria are protective of fish but were designed specifically to protect mink (Mustela vison) that eat fish from PCB-contaminated waters. In 96-hour acute assays using largemouth bass and redear sunfish (Lepomis microlophus) exposed to Capacitor 21 (a PCB mixture), LC_{50} values of 2.3 and 19 ug/liter, respectively, were reported (USEPA 1980e).

PCDD/PCDFs. No AWQC have been developed for PCDD/PCDFs, but the available data indicate that these substances are extremely toxic to aquatic life at very low concentrations. Several toxicity test with fish species common to Winona have been conducted. Egg development was retarded and growth of yolk sac fry was reduced in northern pike (Esox lucius) exposed for 96 hours to 0.1 ng/liter of 2,3,7,8-TCDD and observed for 163 days (USEPA 1984e). Channel catfish exposed to 2.4 to 4.2 ng/liter of 2,3,7,8-TCDD for 20 days exhibited fin necrosis, erratic swimming, hemorrhaging from the anus, and death (Eisler 1986). Carp exposed for 71 days to 0.1 ng/liter of 2,3,7,8-TCDD suffered erosion and blackening of the fins, skin lesions, degeneration of the bones, tumorous lesions in the caudal fin, necrosis of liver cells, and a variety of other toxic effects (USEPA 1984e).

2,4,6-Trichlorophenol. Data on the freshwater toxicity of 2,4,6-trichlorophenol are limited and an AWQC has not been developed for this or related compounds. In an acute lethality assay with bluegills, a 96-hour LC_{50} of 320 ug/liter has been reported (USEPA 1980c).

4.2.6.5.2. Terrestrial Toxicity-Birds

Criteria similar to AWQC have not been developed for the protection of

terrestrial species. Consequently, critical toxicity values to evaluate hazards to terrestrial species must be derived using data available in the literature. We conducted a literature search to identify the toxic effects of the indicator chemicals on birds, with particular emphasis on the birds common to the Winona area. No toxicity information was found on the avian toxicity of nitrogen oxides, sulfur oxides, carbon monoxide, antimony, 1,4-dichlorobenzene, and 2,4,6-trichlorophenol. Toxicity information for all other indicator chemicals was available to varying degrees.

Below, we discuss the toxicity of the indicator chemicals to birds, and identify toxicity values that may be used in an assessment of potential risks to resident bird species at Winona. These toxicity values are listed in Table 4.2.2d, and in most cases represent the highest dietary concentration of a chemical not associated with an adverse effect in birds, or the lowest dietary concentration reported to cause an effect.

The following guidelines were used to select the critical toxicity values. Chronic values were chosen over acute toxicity values when available. Acute toxicity values were used only if no chronic data were available for any species. If a chronic no-observed-effect level (NOEL) and a chronic lowest-observed-effect level (LOEL) were available, the NOEL was chosen as the critical toxicity value. The NOEL selected was the highest NOEL reported. If LOELs were used, the value selected was the lowest LOEL reported. Effort was made to obtain toxicity information for bird species of Winona, or for similar species. Therefore, if chronic information was available for both ducks and raptor species (represented by falcons and eagles), critical toxicity values were identified for both types of birds. If a chronic value was available for only one of these bird types, it was used as a representative value for both types of bird if it was not larger than the highest NOEL or lowest LOEL for any other species. If it was larger, one of the latter values were used as critical toxicity value for the species for which specific toxicity data was not available. If only an LC_{50} was available, the value selected was the lowest LC_{50} reported in the literature.

Arsenic. Studies of arsenic avian toxicity are limited primarily to studies of lethal exposures. Heath et al. (1972) and Hill et al. (1975) studied the effects of arsenic (III) on several species of birds. In these experiments, mallard ducks, bobwhite quail (Colinus virginianus), Japanese quail (Coturnix japonicus), and ring-necked pheasants (Phasianus colichicus) were fed arsenic in the diet for 5 days and then given an arsenic-free diet for 3 days. LC_{50} values based on these exposures ranged from 480 ppm in the diet of bobwhite quail to greater than 5,000 ppm in the diet of mallards. The order of species sensitivity was bobwhite quail > Japanese quail > ringed-neck pheasant > mallard. The LC_{50} for mallards is used as a critical toxicity value for ducks and the lowest LC_{50} (for bobwhites) is used as a value for raptors.

Cadmium. Cadmium can cause death and a variety of sublethal effects in birds exposed via the diet. Sublethal effects include growth retardation, anemia, and testicular damage. Several studies are available on the dietary toxicity of cadmium to ducks. In mallard ducklings fed dietary cadmium from day 1 of age for 12 weeks, hematological effects were observed at 8 weeks and mild to severe kidney lesions developed at 12 weeks (Cain et al. 1983). However, no hematological effects were observed in adult mallards exposed to 20 or 200 ppm cadmium in the diet for 90 days (White and Finley 1978), although kidney lesions, testicular degeneration, and complete cessation of spermatogenic activity was observed in adult mallards exposed to 200 ppm in the diet for 90 days (White et al. 1978). In other duck species, no effects on growth or kidneys were observed in wood ducks (Aix sponsa) exposed from age 1

week to age 7 weeks to 1, 10, or 100 ppm cadmium in the diet (Mayack et al. 1981). Altered avoidance behavior in the form of hyperresponsiveness was observed in young black ducks (Anus rubripes) born to parents exposed to 4 ppm cadmium in the diet for 4 months prior to birth of their young (Heinz and Haseltine 1983).

No data were located on cadmium toxicity in raptor species, such as falcons or eagles, nor were effects identified in other bird species which occurred at dietary concentrations lower than those observed to cause toxic effects in ducks. The LOEL in ducks was 4 ppm in the diet. This level resulted in altered avoidance behavior in young black ducks.

Chromium. Few data are available on the toxic effects of chromium in birds. Adult black ducks fed diets containing 10 or 50 ppm chromium (III) for five months were normal in survival, reproduction, and blood chemistry (Haseltine et al. 1985). However, ducklings from treated groups that were fed chromium at the original parental doses experienced altered growth and reduced survival. No effect on avoidance response was observed in young black ducklings fed chromium at 20 or 100 ppm in the diet for seven days that were born to parents fed these same doses (Heinz and Haseltine 1981). No data on the toxicity of chromium to raptor species were located. The 10 ppm dietary LOEL in ducks was the lowest LOEL for birds indicated in the available literature. Because no toxicity data on chromium (VI) were available, the toxicity of chromium (III) is used as representative of chromium (VI) toxicity.

Lead. The majority of information on lead toxicity in birds is on body burdens in waterfowl that have ingested spent lead shot and died. However, limited dose-response information is available for a few species. Neurological effects were observed within 24 hours of dosing in mallard ducks that had ingested and absorbed lead shot for a total intake of 423.8 mg/kg bw (Mautino and Bell 1987). These effects decreased 8 days after dosing. Inhibition of delta-ALAD was apparent 1 week after dosing. Assuming a mallard weighs approximately 1.2 kg (calculated from Terres 1980) and consumes an amount of food equivalent to 10% of its body weight each day, the dosage of 423.8 mg/kg bw is equivalent to an approximate lead concentration in the food of 4,600 ppm. In American kestrels (Falco sparverius) fed 10 or 50 ppm lead in the diet for 7 months, no effects were noted with respect to survival, egg laying, initiation of incubation, or egg shell thickness (Pattee 1984). In 1-day-old American kestrels fed 125 or 625 mg/kg bw lead for 10 days, growth was seriously depressed by day 6, and hematocrit values were significantly depressed by day 10 (Hoffman et al. 1985). Forty percent of the birds receiving 625 mg/kg lead died within 6 days. No effects were observed in kestrels exposed to 25 mg/kg bw. Assuming complete absorption of the administered dose, and that a kestrel weighs 0.11 kg (calculated from Terres 1980) and consumes an amount of food equivalent to 10% of its body weight, the 25 mg/kg bw dosage corresponds to an approximate lead concentration in food of 280 ppm. Therefore, the 50 ppm level identified in the Pattee (1984) study is the highest NOEL identified for birds based on the studies reviewed.

Mercury. Most of the data on the toxicity of mercury to birds in on the effects of methylmercury, which is a much more toxic form to birds. In toxicity studies with Coturnix (a type of quail), violent neurological dysfunction was observed beginning at 2 and ending at 4 hours following oral treatment with inorganic mercury (Eisler 1987a). An LD_{50} of 26 to 54 mg/kg bw was reported in this species (Eisler 1987a). No death occurred in this species when exposed to 32 ppm mercury in the diet from hatching for 9 weeks. In Japanese quail, an LD_{50} of 31.1 mg/kg bw was reported (Eisler 1987a). No other appropriate data were located on the toxicity of inorganic mercury to birds.

The 32 ppm NOEL reported for Coturnix was the only chronic value NOEL for inorganic mercury identified in the literature.

Nickel. Few data are available on the toxic effects of nickel in birds. In a feeding study with mallard ducklings fed 0, 200, 800, or 1200 ppm dietary nickel from day 1 to day 90 of age, neurological effects were observed in the highest dose group within 14 days of dosing (Cain and Pafford 1981). The weights of the ducks in the highest dose group were significantly decreased at 28 days of age, and the weight/length ratio of females in the 800 ppm group were significantly different from controls at days 30 and 60. A NOEL of 200 ppm can be identified for this study. No information was available on nickel toxicity in raptor species or in other species at a concentration lower than the duck NOEL.

PAHs. Data on the toxicity of PAHs to birds are limited. Mallards fed for 7 days diets containing various PAHs at a total concentration of 4,000 ppm exhibited increased liver weights and blood flow to the liver when compared to controls (Patton and Deiter 1980). This was the only LOEL identified in the literature.

PCBs. PCBs can cause death and a variety of sublethal effects in birds exposed to the chemical via the diet. Sublethal effects following chronic exposures include enzyme induction, porphyria, altered vitamin A metabolism, alteration of the thyroid and cardiac, behavioral, and hormonal effects. There is significant variability in the species sensitivity to the reproductive effects of PCBs. No reproductive effects were observed in mallards exposed for 12 weeks during egg laying to 150 ppm Aroclor 1252 (Haseltine and Prouty 1980), or in mallards exposed to 25 ppm Aroclor 1254 for at least a month before egg laying (Custer and Heinz 1980). However, mourning doves (Zenaida macroura) exposed to 10 ppm Aroclor 1254 for 28 days in the food experienced delayed reproduction and a decrease in the number of eggs laid (Koval et al. 1987). American kestrel exposure to 10 ppm Aroclor 1254 in food potentiated increased egg shell thinning and subsequent breakage of eggs caused by DDE (Lincer 1972). No reproductive effects were observed in screech owls (Otus asio) exposed for 8 weeks before the onset of egg laying to 3 ppm Aroclor 1248 in the diet (McLane and Hughes 1980). Chickens appear to be the most sensitive species to the reproductive effect of PCBs, with effects noted at dietary levels of 8 to 10 ppm. A NOEL of 150 ppm is identified for ducks based on the Haseltine and Prouty (1980) study in mallards. A NOEL of 3 ppm is identified for raptor species based on the McLane and Hughes (1980) study in screech owls.

These levels may not be sufficiently protective of other terrestrial species, however. Mink (Mustela vison) are particularly susceptible to the reproductive effects of PCBs. Mink fed approximately 0.2 mg/day PCBs in the form of residues in Great Lake carp failed to reproduce (Hornshaw et al. 1983), and mink fed contaminated beef from cows fed Aroclor 1254 developed reproductive complications at residue levels as low as 0.64 ppm (Platanow and Karstad 1973). Dietary concentrations only slightly higher than the latter value can be lethal to mink. A dietary LC_{50} of 6.7 ppm has been reported for Aroclor 1254 (Ringer 1983).

PCDD/PCDFs. PCDD/PCDFs pose potential hazards to terrestrial wildlife because they accumulate in the food chain and are toxic at low levels. Top predators are particularly susceptible to dioxin accumulation because of potentially high dioxin levels in their prey. Lower et al. (1987) reported high dioxin concentrations (857 ppt) in blue racer snakes (Coluber constrictor) collected from a dioxin hazardous waste site at Times Beach, Missouri. Snakes are a top carnivore and feed on small mammals, frogs, insects, and other

snakes. Likewise, other terrestrial species that are completely or partially carnivorous (e.g., ducks, eagles) will be exposed to dioxin levels higher than those in the surrounding media.

PCDD exposure in birds has been most prominently linked with "chick edema" disease, which is characterized by increased pericardial fluid, interstitial edema of the myocardium, and pulmonary edema. Levels as low as 10 ug/kg of 2,3,7,8-TCDD were sufficient to cause this response in 3-day-old chicks exposed for 21 days by oral intubation (Schwetz et al. 1973). Assuming a 3 to 21 day-old chick weighs an average of 0.12 kg (calculated from Schwetz et al. 1973) and consumes 10% of its weight in food, the 10 ug/kg dosage corresponds to a dietary concentration of 0.1 ppm. 2,3,7,8-TCDD also has been shown to be teratogenic in chickens at very low concentration in eggs.

Very little information is available on toxicity in wild species. Gilbertson (1983) reported symptoms characteristic of chick edema in herring gulls (Larus argentatus) in the Great Lakes area. Although no cause and effect relationship could be established, a decrease in edema symptoms in herring gulls from 1971 to 1981 accompanied a decrease in 2,3,7,8-TCDD in eggs. In another study in the Great Lakes area, reproductive failure of Foster's terns (Sterna fosteri) was associated with significant differences in the egg concentrations of 2,3,7,8-TCDD as well as other contaminants that have similar mechanisms of toxicity (Harris et al. 1985). Quantitative dose-response data in wild species are limited to LD_{50}s. LD_{50} values computed 37 days after a single oral dose of 2,3,7,8-TCDD varied from 15 ug/kg bw in Northern bobwhite quail to more than 810 ug/kg bw in ringed turtle doves (Hudson et al. 1984). Mallards were intermediate in sensitivity with an LD_{50} of 108 ug/kg bw. The LD_{50} information will not be used as a critical toxicity value, but instead the LOEL of 0.1 ppm calculated for chickens is selected.

4.2.4.6.3. Toxicity to Plants

No information is available on the toxicity of the indicator chemicals to the threatened and special concern species or similar species. However, information is available for toxicity of some indicator chemicals to some plant species. This information is presented below.

Carbon monoxide. Carbon monoxide at a continuous concentration of 24 ppm (28,000 ug/m^3) accelerated the rate of seedling death in wild mustard (Brassica kaber), cocklebur (Xanthuim pennsylvanicum), soybean (Glycine max), giant ragweed (Ambrosia trifida) and pigweed (Amaranthus retroflexus) (Chakrabarti 1979). In addition, the lifespan for seedlings of wild mustard, pigweed and soybean was reduced.

Metals. Metal toxicity in plants operates via several mechanisms. Metals can change the permeability of plant cell membranes (cadmium, mercury lead), can compete for sites with essential metabolites (arsenic), can occupy sites for essential groups such as phosphate and nitrate (arsenic), can react with thiol groups (mercury, lead), and disrupt a variety of other plant processes. Kabata-Pendias and Pendias (1984) reviewed the literature on metal phytotoxicity and identified the total concentrations of selected metals in surface soils that were phytotoxically excessive. These concentrations include the following (as ppm dry weight): arsenic, 15; cadmium, 3; chromium, 75; lead, 100; mercury, 0.3; nickel, 100.

Nitrogen Dioxide. Nitrogen dioxide inhibited apparent photosynthesis of alfalfa at concentrations above 0.6 ppm (1,000 ug/m^3) (Hill and Bennent 1970). Continuous exposure of naval orange trees to 0.5 ppm (900 ug/m^3) and 1 ppm

(1,800 ug/m^3) of nitrogen dioxide for 30 days caused severe defoliation and chlorosis (Thompson et al. 1970). Chakrabarti (1979) reported that exposure to 24 ppm (45,000 ug/m^3) nitrogen dioxide inhibited germination in wild mustard, soybean, giant ragweed, and pigweed. In addition, nitrogen dioxide accelerated seedling death in all species and reduced seedling lifespan in wild mustard, pigweed and soybean.

PCBs. PCBs do not appear to be very toxic to terrestrial plants, probably because of the extensive sorption of PCBs to soils. However, adverse effects were reported in soybean plants exposed to 1,000 mg/kg Aroclor 1254 in soil (Weber and Mrozek 1979). A PCB concentration of 20 ppm (269 ug/m^3) Aroclor 1221 inhibited photosystem II in spinach chloroplasts (Sinclair et al. 1977).

Sulfur dioxide. The treatment of 1 g/m^3 sulfur dioxide for 2 hours at an interval of 7 days caused partial chlorosis in some leaves of Hordeum vulgare (Coibanu and Coibanu 1973). Additionally, photosynthesis was inhibited and the cytoplasm and organelles of exposed plants were completely destroyed. Sulfur dioxide at a concentration of 24 ppm (63,000 ug/m^3) inhibited germination and induced rotting of seeds in wild mustard, soybean, giant ragweed and pigweed (Chakrabarti 1979). In comparative studies with nitrogen dioxide and carbon monoxide, sulfur dioxide was found to be more toxic to plants, inducing more severe toxic responses at similar exposure concentrations (Chakrabarti 1979).

4.2.6.6. EXPOSURE ASSESSMENT

In this Section, we identify exposure pathways and quantify exposure for fish, birds and plants. The exposure estimates are based on the modeling results presented in Section IX TWP ICF Clement. Only estimated maximum exposure concentrations are used in this exposure assessment.

4.2.6.6.1. Exposure to Fish

Fish may be exposed to chemical contaminants released from the incinerator at Winona via contact with contaminated water and sediment and ingestion of contaminated food. However, toxicity data are not available to assess exposure via each of these pathways, and therefore this exposure assessment is limited to an assessment of exposure to contaminated surface water. The maximum surface water concentrations presented in Section IX TWP Clements are the estimated exposure concentrations for fish.

4.2.6.6.2. Exposure to Birds

There are a number of pathways by which birds may be exposed directly or indirectly to chemicals released from the incinerator. Direct pathways are those in which animals directly contact or ingest contaminated media. Indirect pathways are those in which animals consume other, previously contaminated animals.

Direct pathways for bird at Winona include ingestion of contaminants in sediments or water, direct contact with contaminated soil or sediments, and inhalation of airborne contaminants. Unfortunately, estimates of exposure via these pathways are not easily quantified using available data. Intake from ingestion of contaminated water could be estimated, but this pathway is not believed to contribute significantly to overall intake relative to intake via food for most contaminants. Therefore, exposure to birds via any of the direct pathways will not be quantified.

Birds may be exposed to chemicals indirectly by ingesting contaminated

food. Exposure via this pathway is greatest in those species which consume food items which tend to bioaccumulate or bioconcentrate contaminants. For example, many aquatic invertebrates and fish are known to bioaccumulate some heavy metals and organic chemicals. Therefore this pathway may be significant for ducks which rely on aquatic invertebrates for a portion of the dietary intake and bald eagles which, in Winona, rely almost exclusively on fish as a food source. Intake estimates from food will be made for both ducks and eagles. Peregrine falcons also may be exposed to contaminants via food. However, in Winona the primary food source for these birds is pigeons and the data available are not sufficient to estimate uptake and accumulation of contaminants in pigeons. Therefore, falcon exposure will not be quantified.

Ducks. As discussed in Section TWP ICF Clements on receptor characterization, the resident duck species of Winona rely on animal food (i.e., insects, crustaceans, and snails) for some portion of their dietary intake, although the three species vary somewhat in the percentage of animal food in the d, and wood ducks consume approximately 10% animal food. For the purposes of this exposure assessment, the diet composition of mallards will be used as an average value for the three resident duck species.

Dietary intakes are estimated by calculating the amount of food ingested each day and the concentration of chemical in the food. For the purposes of this exposure assessment it is assumed that contaminants accumulate only in the animal portion of the diet, although it is likely that some contaminants also accumulate in plants. To determine the amount of food consumed it is assumed that a mallard consumes daily an amount of food equal to 10% of its body weight. Using an average weight of 1.2 kg for male and female mallards (calculated from Terres 1980), a daily intake of 0.12 kg is determined, and of this 0.024 kg is animal food. The concentration of chemical in the food is estimated using bioconcnetration factors (BCFs) for aquatic invertebrates derived from the literature. These values are listed in Table 4.2.6e. Concentrations in the animal food are derived by multiplying the BCF by the surface water concentration. The amount (mg) of chemical ingested from animal food is determined by multiplying the concentration in the food by 0.024. This chemical intake is then divided by the total amount of food ingested daily (0.12 kg) to determine the concentration of chemical in the entire diet (plant and animal). The estimated concentrations in the animal food and the whole diet for the Riverbend II and Riverview sites are presented in Tables 4.2.2f and 4.2.6g, respectively.

Bald Eagles. Because of the rich supply of fish in the Winona area, bald eagles living in the area rely almost exclusively on fish for food, and therefore for the purposes of this exposure assessment, it is assumed that 100% of the eagle's diet is fish. The dietary concentration of contaminants for bald eagles is calculated by multiplying the surface water concentration by BCFs for fish obtained from the literature. The BCFs used in these calculations are presented in Table 4.2.6h. The resultant dietary concentrations for the Riverbend II and Riverview sites are presented in Tables 4.2.6i and 4.2.6j, respectively. BCFs for whole body bioconcnetration were used because birds eat the whole fish and not just the muscle, as do humans. The BCF for PCBs was derived following short-term exposure in fathead minnows. Some of the available data indicate that steady state may not be reached after short-term exposures, and that BCFs at steady state may be higher. Therefore, the BCF of 100,000 may underestimate accumulation of PCBs in aquatic invertebrates.

4.2.6.6.3 Exposure to Plants

Plants may be exposed to contaminants in air, soil and water. However, because toxicity information was available only for pollutants in air and soil, only exposure via these pathways will be quantified. The maximum estimated air and soil concentrations presented in Section IX TWP ICF Clement will be used as exposure levels for plants.

4.2.6.7. RISK CHARACTERIZATION

In the following sections we discuss the possible risks to fish, birds and plants resulting from exposure to chemicals potentially released from the incinerator at Winona.

4.2.6.7.1 Risks to Fish

Potential risks to fish at Winona are estimated by comparing the estimated surface water concentrations with the AWQC or the selected toxicity value for each chemical of concern. Chemicals at concentrations that exceed the AWQC may pose a risk to the fish as well as other aquatic life near Winona. For the five indicator chemicals for which no AWQC have been developed, estimated surface water concentrations are compared to measured or estimated chronic toxicity values obtained from the literature. For antimony, PAHs and PCDD/PCDFs, estimated surface water concentrations are compared to measured chronic values. Chemicals at concentrations that equal or exceed these LOELs may pose an increased risk to the fish of the area. For the two remaining chemicals, only LC_{50} values were available. Surface water concentrations are compared to a value $1/100^{th}$ of the reported LC_{50} value. Kenaga (1983) evaluated available acute and chronic aquatic toxicity data to derive a relationship between acute LC_{50} values and chronic maximum allowable toxicant concentrations (MATC) for aquatic species. He found that for approximately 86% of all species examined (252 total), the LC_{50} value was 100 times greater than the MATC. Therefore, in this risk assessment we have divided the LC_{50} value by 100 to derive a MATC. Chemicals at concentrations which exceed the estimated MATC may pose an increased risk to fish in the Winona area.

Tables 4.2.6k and 4.2.6l present a comparison of the estimated surface water concentrations at the Riverbend II and Riverview sites, respectively, with the AWQC or selected toxicity value. As the Tables show, air emissions from either site do not result in surface water concentrations in excess of the acute or chronic AWQC for any chemical. In fact, all estimated surface water concentrations are between 3 and 8 orders of magnitude below the AWQC. Estimated concentrations for the chemicals without AWQC also are well below the selected MATC or LOEL value. Because of these very low estimated surface water concentrations relative to toxic concentrations, it is unlikely that the incinerator emissions would pose a significant threat to fish in the Winona area. There are, however, several uncertainties associated with concentration estimates and toxicity values which may over- or under-estimate risk.

4.2.6.7.2. Risks to Birds

Risks to birds are estimated by comparing the estimated concentrations of chemicals in food to the dietary concentrations identified as critical toxicity values in Section XI.B TWP ICF Clement. Estimated dietary concentrations that are below a NOEL for a chemical are not likely to pose a risk to birds. Dietary concentrations that are near, equal or greater than a LOEL for a chemical may pose a risk to the birds. NOELs or LOELs are available for all chemicals of concern except arsenic, for which a dietary LC_{50} is available. In evaluating the potential effects of pesticides, USEPA analyzed a subset of available dose-response data and suggested that if the estimated dose is less

than one-fifth of the LC_{50} for nonendangered species and one-tenth of the LC_{50} for endangered species, no acute hazard can be presumed (Urban and Cook 1986). We adopt this rule for this risk assessment, and compare the estimated dietary concentration of arsenic to one-fifth of the LC_{50} value for ducks and one-tenth of the LC_{50} value for eagles. This approach does not consider chronic toxicity however.

Tables 4.2.6m and 4.2.6n compare the selected toxicity values with the estimated dietary concentrations for ducks at the Riverbend II and Riverview sites, respectively. Tables 4.2.6o and 4.2.6p compare these values for bald eagles at the Riverbend II and the Riverview sites, respectively. In all cases, the estimated dietary concentration is well below (5 to 11 orders of magnitude) the toxic concentration. Therefore emissions from the incinerator at either proposed location may not adversely affect birds in the Winona area. These conclusions are not absolute however. Many assumptions were made to estimate toxicity and exposure which may lead to under- or over-estimates of risk. For example, only potential impacts to ducks and raptors were assessed and these species were assumed to be representative of other bird species in the Winona area, although these other species may be more or less sensitive than ducks or raptors. Also, it was assumed that ducks' sole contaminant source is via aquatic invertebrates. However, ducks may ingest contaminants in plant food or water and while preening, and may inhale contaminants or absorb them across the skin. These pathways all contribute to exposure, and although it is unlikely that exposure via these pathways would increase overall exposure by 5 to 11 orders of magnitude to toxic levels, estimated exposure would be higher. There are also many uncertainties associated with the toxicity values used which may contribute to over- or underestimates of risk. Nevertheless, the estimates of low risk for birds are viewed as scientifically reasonable, although not absolute estimates of risk.

4.2.6.7.3. Risks to Threatened and Special Concern Plants

Estimates of risks to threatened and special concern plants in the Winona area are limited because no toxicity data are available for these or similar species. Available information on other plant species can be used for a general estimate of potential risks, but the species in the Winona area may be more or less sensitive so that may risks may be over- or under-estimated. Nevertheless, a comparison of the maximum estimated annual average ambient air concentrations of nitrogen dioxide, sulfur dioxide, and PCBs and the estimated maximum 8-hour average carbon monoxide concentrations with the lowest concentrations of these chemicals associated with an adverse effect in plants indicates that potential toxic effects from estimated air contamination from either proposed incinerator may not occur. The estimated air concentrations in each case are well below (3 to 9 orders of magnitude) those associated with adverse effects in other plant species.

Comparison of estimated soil concentrations of arsenic, chromium, lead, and nickel with soil levels of these metals identified as phytotoxic also indicates potentially small risk. Estimated soil concentrations for these metals resulting from emissions at either proposed incinerator location are between 1 and 3 orders of magnitude lower than identified toxic levels for some plants. However, the estimated maximum soil concentrations of mercury at the Riverview Drive site (6.09 ppm) and the Riverbend II site (8.96 ppm) exceed the soil level identified as phytotoxic (0.3 ppm) in at least some plants. Also, the estimated maximum soil concentrations of cadmium at the Riverview Drive site (1.71 ppm) and the Riverbend II site (2.52 ppm) approach the soil level identified as phytotoxic in some plants. Therefore, soil concentrations resulting from the emission of mercury and cadmium from either proposed site

may result in increased risks to threatened and special concern plants in the Winona area as well as other plants. Again, however the uncertainties identified above limit the certainty of this conclusion.

TABLE 4.2.6A

ENDANGERED, THREATENED AND SPECIAL CONCERN THAT OCCUR WITHIN A FIVE MILE RADIUS OF WINONA

SPECIES	STATUS	HABITAT PREFERENCE
ANIMALS		
Peregrine falcon (*Falco peregrinus*)	FE, SE	Uses cliff faces for nesting sites
Bald eagle (*Haliaeetus leucocephalus*)	FT, ST	Forest and open water habitats
Blue racer (*Coluber constrictor*) and open woods	SSC	Prefers upland habitats, including bluff prairies, forested hillsides,
Timber rattlesnake (*Crotalus horridus*)	SSC	Deciduous forests and bottomlands; bluff areas
PLANTS		
Hill's thistle (*Cirsium hillii*)	SSC	Dry, sandy or gravely soils in prairies, savannas and open woods
White ladys' slipper (*Cyperpedium candidum*)	SSC	Mesic prairies
Clustered broom-rape (*Orobanche fasciculata*)	SSC	Dry, gravel prairies and sand dunes
Ginseng (*Panax quinquefolium*)	SSC	Hardwood forests and loamy soil
Hair-like beak-rush (*Rhynchospora capillacea*)	ST	Groundwater-maintained habitats
Lance-leaved violet (*Viola lanceolata*)	SSC	Wet, sandy shores, wetland margins, and peaty meadows

FE = federally endangered; SE = state endangered; FT = federally threatened; ST = state threatened; SSC = state special concern

TABLE 4.2.6B

AMBIENT WATER QUALITY CRITERIA FOR THE ASSESSMENT
OF IMPACTS TO FISH OF SURFACE WATER CONTAMINANTS AT WINONA

CHEMICAL	AMBIENT WATER QUALITY CRITERION (ug/liter)		REFERENCE
	Acute	Chronic	
Antimony	--	--	
Arsenic (III)	360	190	USEPA (1984a)
Beryllium	--	--	
Cadmium	8.6+	2.0+	USEPA (1984b)
Chromium (VI)	16	13	USEPA (1984c)
1,4-dichlorobenzene	--	--	
Lead	200+	7.7+	USEPA (1985)
Mercury	2.4	0.012	USEPA (1984d)
Nickel	2,500+	280+	USEPA (1986)
PAHs	--	--	
PCBs	2.0	0.014	USEPA (1980e)
PCDD/PCDFs	--	--	
2,4,6-Trichlorophenol	--	--	

-- = not available
+ = hardness dependent criterion; a value of 200 mg/liter (as $CaCO_3$) was used to determine criterion

TABLE 4.2.6C

OTHER TOXICITY VALUES FOR THE ASSESSMENT OF IMPACTS TO FISH OF SURFACE WATER CONTAMINANTS AT WINONA

CHEMICAL	CONCENTRATION (ug/l)	SPECIES	EFFECT	REFERENCE
Antimony	1,600	Fathead minnow	Chronic value	USEPA (1980a)
Beryllium	1,300[a]	Bluegill	96 h LC_{50}	USEPA (1980b)
1,4-Dichlorobenzene	4,280	Bluegill	96 h LC_{50}	USEPA (1980d)
PAHs	5,000	Minnow (2 species)	Liver neoplasms following 6 weeks of exposure to 7,12-dimethylbenz(a)pyrene	Schultz and Schultz (1980)
PCDD/ PCDFs	0.0001	Carp	skin tumors, lesions, liver necrosis and other effects following 71 days exposure to 2,3,7,8-TCDD	USEPA (1984e)
2,4,6-Trichlorophenol	320	Bluegill	96 h LC_{50}	USEPA (1980c)

[a] Test was conducted at a water hardness of 20 mg/l (as $CaCO_3$). The water hardness at Winona is 200 mg/liter. Data suggest that beryllium toxicity decreases with increasing water hardness. Therefore it is likely that an LC50 determined at a water hardness of 200 mg/liter would be higher.

TABLE 4.2.6D

CRITICAL TOXICITY VALUES FOR THE ASSESSMENT OF IMPACTS TO BIRDS IN THE WINONA AREA

Chemical	Diet conc. (ppm)	Exposure duration	Species	Effect	Source
Arsenic	5,000 480	5 d 5 d	Mallards Bobwhite quail	LC_{50} LC_{50}	Heath et al. (1972), Hill et al. (1975)
Cadmium	4	4 mo	Black ducks	LOEL, behavior alterations	Heinz and Haseltine (1983)
Chromium	10	5 mo	Black ducks	LOEL, reproduction	Haseltine et al. (1985)
Lead	50	7 mo	American kestrels	NOEL, reproduction	Pattee (1984)
Mercury	32	9 wk	Coturnix	No death observed	Eisler (1987a)
Nickel	200	90 d	Mallards	NOEL, neurological effects	Cain and Pafford (1981)
PAHs	4,000	7 d	Mallards	LOEL, liver effects	Patton and Dieter (1980)
PCBs	150	12 wk	Mallards	NOEL, reproduction	Haseltine and Prouty (1980)
	3	8 wk	Screech owls	NOEL, reproduction	McLane and Hughes (1980)
PCDD/ PCDFs	0.1[a]	18 d	Chickens	LOEL, chick edema	Schwetz et al. (1973)

[a] Calculated value; see text

TABLE 4.2.6E

BIOCONCENTRATION FACTORS FOR AQUATIC INVERTEBRATES

CHEMICAL	BCF	BASIS	REFERENCE
Arsenic	17	whole body bioconcentration by snails (Helisoma campanulatum) for 28 days of exposure	USEPA (1984a)
Cadmium	4,190	28-week whole body bioconcentration by caddis fly (Hydropsyche botteni) nymphs	USEPA (1984b)
Chromium	--	no information available	
Lead	2366	whole body bioconcentration by mayfly (Ephemerella grandis) nymphs	USEPA (1985)
Mercury	2500	7-day bioconcentration in amphipods (Gammarus sp.)	USEPA (1984d)
Nickel	192	estimated whole body bioconcentration in cladocerans (Daphnia sp.) exposed for 3.75 days	USEPA (1986)
PAHs	82,231	3-day bioconcentration in snails (Physa sp.)	Eisler (1987b)
PCBs	6,200	whole body bioconcentration in amphipods (Gammarus pseudolimnaes) exposed for 21 days to Aroclor 1254	USEPA (1980e)
PCDD/PCDFs	3,731	32-day bioconcentration in snails (Helimsoma sp.) exposed to 2,3,7,8-TCDD	Eisler (1985)

TABLE 4.2.6F

ESTIMATED CHEMICAL CONCENTRATIONS IN AQUATIC INVERTEBRATES AND THE WHOLE DIET OF DUCKS FOR THE RIVERBEND II SITE

CHEMICAL	BCF	CONCENTRATIONa		
		WATER (mg/liter)	ANIMAL FOOD (ppm)	WHOLE DIET (ppm)
Arsenic	17	1.14E-08	1.9E-07	3.9E-08
Cadmium	4,190	9.53E-08	4.0E-04	8.0E-05
Lead	2,366	2.28E-06	5.4E-03	1.1E-03
Mercury	2,500	3.52E-07	8.8E-04	1.8E-04
Nickel	192	7.53E-08	1.4E-05	2.9E-06
PAHs	82,231	5.26E-07	4.3E-02	8.7E-03
PCBs	6,200	6.68E-10	4.1E-06	8.3E-07
PCDD/PCDFs	3,731	2.52E-12	9.4E-09	1.9E-09

[a] Estimated concentrations are for the maximum case only.

TABLE 4.2.6G

ESTIMATED CHEMICAL CONCENTRATIONS IN AQUATIC INVERTEBRATES AND THE WHOLE DIET OF DUCKS FOR THE RIVERVIEW SITE

CHEMICAL	BCF	CONCENTRATIONa		
		WATER (mg/liter)	ANIMAL FOOD (ppm)	WHOLE DIET (ppm)
Arsenic	17	1.07E-08	1.8E-07	3.6E-08
Cadmium	4,190	9.00E-08	3.8E-04	7.5E-05
Lead	2,366	2.15E-06	5.1E-03	1.0E-03
Mercury	2,500	3.33E-07	8.3E-04	1.7E-04
Nickel	192	7.11E-08	1.4E-05	2.7E-06
PAHs	82,231	4.97E-07	4.1E-02	8.2E-03
PCBs	6,200	6.31E-10	3.9E-06	7.8E-07
PCDD/PCDFs	3,731	2.38E-12	8.9E-09	1.8E-09

[a] Estimated concentrations are for the maximum case only.

TABLE 4.2.6H

BIOCONCENTRATION FACTORS FOR FISH

CHEMICAL	BCF	BASIS	REFERENCE
Arsenic	4	28-day whole body bioconcentration of arsenic (III) by bluegills (Lepomis machrochirus)	USEPA (1984a)
Cadmium	540	140-day whole body bioconcentration by rainbow trout (Salmo gairdneri)	USEPA (1984b)
Chromium (VI)	1	whole body bioconcentration of chromium (VI) by rainbow trout (Salmo gairdneri) exposed for 30 days	USEPA (1984c)
Lead	45	Whole body bioconcentration by bluegills (Lepomis machrochirus)	USEPA (1985)
Mercury	5,000	287-day whole body bioconcentraion by fathead minnows (Pimephales promelas)	USEPA (1984e)
Nickel	106	whole body bioconcentration by rainbow trout (Salmo gairdneri) exposed for 30 days	USEPA (1986)
PAHs	920	10-day whole body bioconcentration of B(a)P by rainbow trout (Salmo gairdneri)	Gerhart and Carlson (1978)
PCBs	100,000	32-day whole body bioconcentration in fathead minnows	Vieth and Kosian (1983)
PCDD/PCDFs	86,000	whole body bioconcentration of TCDD by rainbow trout (Salmo gairdneri) calculated for 382 days	Petty et al. (1986)

TABLE 4.2.6I

ESTIMATED CHEMICAL CONCENTRATION IN THE
FISH DIET OF BALD EAGLES FOR THE RIVERBEND II SITE

CHEMICAL	BCF	CONCENTRATION[a]	
		WATER (mg/liter)	FISH (ppm)
Arsenic	4	1.14E-08	4.6E-08
Cadmium	540	9.53E-08	5.1E-05
Chromium	1	9.71E-09	9.7E-09
Lead	45	2.28E-06	1.0E-04
Mercury	5,000	3.52E-07	1.8E-03
Nickel	106	7.53E-08	8.0E-06
PAHs	920	5.26E-07	4.8E-04
PCBs	100,000	6.68E-10	6.7E-05
PCDD/PCDFs	86,000	2.52E-12	2.2E-07

[a] Estimated concentrations are for the maximum case only.

TABLE 4.2.6J

ESTIMATED CHEMICAL CONCENTRATION IN THE
FISH DIET OF BALD EAGLES FOR THE RIVERVIEW SITE

CHEMICAL	BCF	CONCENTRATION[a]	
		WATER (mg/liter)	FISH (ppm)
Arsenic	4	1.07E-08	4.3E-08
Cadmium	540	9.00E-08	4.9E-05
Chromium	1	9.17E-09	9.2E-09
Lead	45	2.15E-06	9.7E-05
Mercury	5,000	3.33E-07	1.7E-03
Nickel	106	7.11E-08	7.5E-06
PAHs	920	4.97E-07	4.6E-04
PCBs	100,000	6.31E-10	6.3E-05
PCDD/PCDFs	86,000	2.38E-12	2.0E-07

[a] Estimated concentrations are for the maximum case only.

TABLE 4.2.6K

COMPARISON OF ESTIMATED SURFACE WATER CONCENTRATIONS
AT THE RIVERBEND II SITE WITH AWQC OR OTHER TOXICITY VALUES

CHEMICAL	CONCENTRATION (ug/liter)[a] WATER	ACUTE AWQC	CHRONIC AWQC	EXCEEDS AWQC?
Arsenic	1.14E-05	360	190	No
Cadmium	9.53E-05	8.6	2.0	No
Chromium	9.71E-06	16	13	No
Lead	2.28E-03	200	7.7	No
Mercury	3.52E-04	2.4	0.012	No
Nickel	7.53E-05	2,500	280	No
PCBs	6.68E-07	2.0	0.014	No

CHEMICAL	CONCENTRATION (ug/liter)[a] WATER	OTHER TOXICITY VALUE	EXCEEDS VALUE?
Antimony	6.44E-05	[b] 1,600	No
Beryllium	3.34E-08	[c] 13 (1,300)	No
1,4-Dichloro-benzene	1.17E-06	[c] 43 (4,280)	No
PAHs	5.26E-04	[b] 5,000	No
PCDD/PCDFs	2.52E-09	[b] 0.0001	No
2,4,6-Trichloro-phenol	2.76E-06	[c] 3.2 (320)	No

[a] Estimated concentrations are for the maximum case only.

[b] Value presented is the LOEL. See text for source.

[c] Value presented is 1/100th of the LC_{50} value in parentheses. See text for explanation of derivation and source.

TABLE 4.2.6L

COMPARISON OF ESTIMATED SURFACE WATER CONCENTRATIONS
AT THE RIVERVIEW SITE WITH AWQC OR OTHER TOXICITY VALUES

CHEMICAL	CONCENTRATION (ug/liter)[a]			
	WATER	ACUTE AWQC	CHRONIC AWQC	EXCEEDS AWQC?
Arsenic	1.07E-05	360	190	No
Cadmium	9.00E-05	8.6	2.0	No
Chromium	9.17E-06	16	13	No
Lead	2.15E-03	200	7.7	No
Mercury	3.33E-04	2.4	0.012	No
Nickel	7.11E-05	2,500	280	No
PCBs	6.31E-07	2.0	0.014	No

CHEMICAL	CONCENTRATION (ug/liter)[a]		
	WATER	OTHER TOXICITY VALUE	EXCEEDS VALUE?
Antimony	6.08E-05	[b] 1,600	No
Beryllium	3.16E-08	[c] 13 (1,300)	No
1,4-Dichloro-benzene	1.11E-06	[c] 43 (4,280)	No
PAHs	4.97E-04	[b] 5,000	No
PCDD/PCDFs	2.38E-09	[b] .0001	No
2,4,6-Trichloro-phenol	2.61E-06	[c] 3.2 (320)	No

a Estimated concentrations are for the maximum case only.

b Value presented is the LOEL. See text for source.

c Value presented is 1/100th of the LC_{50} value in parentheses. See text for explanation of derivation and source.

TABLE 4.2.6M

COMPARISON OF ESTIMATED DIETARY CONCENTRATIONS IN DUCKS
FOR THE RIVERBEND II SITE WITH SELECTED CRITICAL TOXICITY VALUES

CHEMICAL	CONCENTRATION (ppm)		DIET EXCEED TOXICITY VALUE?
	TOXICITY VALUE[a]	ESTIMATED IN DIET[b]	
Arsenic	[c]1,000	3.9E-08	No
Cadmium	4 (LOEL)	8.0E-05	No
Lead	50 (NOEL)	1.1E-03	No
Mercury	32 (NOEL)	1.8E-04	No
Nickel	200 (NOEL)	2.9E-06	No
PAHs	4,000 (LOEL)	8.7E-03	No
PCBs	150 (NOEL)	8.3E-07	No
PCDD/PCDFs	0.1 (LOEL)	1.9E-09	No

[a] See Section XI.2.2 for derivation of values. The type of toxicity value is indicated in parentheses.

[b] Estimated concentrations are for the maximum case only.

[c] This value is one-fifth of the LC50 for arsenic in mallards. See text for explanation.

TABLE 4.2.6N

COMPARISON OF ESTIMATED DIETARY CONCENTRATIONS IN DUCKS
FOR THE RIVERVIEW SITE WITH SELECTED CRITICAL TOXICITY VALUES

CHEMICAL	CONCENTRATION (ppm)		DIET EXCEED TOXICITY VALUE?
	TOXICITY VALUE[a]	ESTIMATED IN DIET[b]	
Arsenic	[c]1,000	3.6E-08	No
Cadmium	4 (LOEL)	7.5E-05	No
Lead	50 (NOEL)	1.0E-03	No
Mercury	32 (NOEL)	1.7E-04	No
Nickel	200 (NOEL)	2.7E-06	No
PAHs	4,000 (LOEL)	8.2E-03	No
PCBs	150 (NOEL)	7.8E-07	No
PCDD/PCDFs	0.1 (LOEL)	1.8E-09	No

[a] See Section XI.2.2 for derivation of values. The type of toxicity value is indicated in parentheses.

[b] Estimated concentrations are for the maximum case only.

[c] This value is one-fifth of the LC50 for arsenic in mallards. See text for explanation.

TABLE 4.2.60

COMPARISON OF ESTIMATED DIETARY CONCENTRATIONS IN BALD EAGLES AT THE RIVERBEND II SITE WITH SELECTED CRITICAL TOXICITY VALUES

CHEMICAL	CONCENTRATION (ppm)		DIET EXCEED TOXICITY VALUE?
	TOXICITY VALUE[a]	ESTIMATED IN DIET[b]	
Arsenic	[c] 48	4.6E-08	No
Cadmium	4 (LOEL)	5.1E-05	No
Chromium	10 (LOEL)	9.7E-09	No
Lead	50 (NOEL)	1.0E-04	No
Mercury	32 (NOEL)	1.8E-03	No
Nickel	200 (NOEL)	8.0E-06	No
PAHs	4,000 (LOEL)	4.8E-04	No
PCBs	3 (NOEL)	6.7E-05	No
PCDD/PCDFs	0.1 (LOEL)	2.2E-07	No

[a] See Section XI.2.2 for derivation of values. The type of toxicity value is indicated in parentheses.

[b] Estimated concentrations are for the maximum case only.

[c] This value is one-tenth of the LC_{50} for arsenic in bobwhite quail. See text for explanation of derivation.

TABLE 4.2.6P

COMPARISON OF ESTIMATED DIETARY CONCENTRATIONS IN BALD EAGLES AT THE RIVERVIEW SITE WITH SELECTED CRITICAL TOXICITY VALUES

CHEMICAL	CONCENTRATION (ppm)		DIET EXCEED TOXICITY VALUE?
	TOXICITY VALUE[a]	ESTIMATED IN DIET[b]	
Arsenic	[c] 48	4.3E-08	No
Cadmium	4 (LOEL)	4.9E-05	No
Chromium	10 (LOEL)	9.2E-09	No
Lead	50 (NOEL)	9.7E-05	No
Mercury	32 (NOEL)	1.7E-03	No
Nickel	200 (NOEL)	7.5E-06	No
PAHs	4,000 (LOEL)	4.6E-04	No
PCBs	3 (NOEL)	6.3E-05	No
PCDD/PCDFs	0.1 (LOEL)	2.0E-07	No

[a] See Section XI.2.2 for derivation of values. The type of toxicity value is indicated in parentheses.

[b] Estimated concentrations are for the maximum case only.

[c] This value is one-tenth of the LC_{50} for arsenic in bobwhite quail. See text for explanation of derivation.

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4.3 SUPPLEMENTARY HEALTH RISK ASSESSMENT

4.3.1 SUMMARY

Four age groups are modeled in both the urban and rural family populations; adults, adolescents, toddlers, and infants. Only adults are modeled in the sportsfisherman population. Infant doses apply for one year, toddler for five years, adolescents for 12 years and adults for 52 years.

4.3.1.1 PCDD/PCDF

The results of this analysis indicate that the highest cancer risk due to PCDD/PCDF emission was associated with the sportsfishermen receptor group. This cancer rate was determined to be 87.0×10^{-5} if this facility emitted 3.0 ng/m^3 PCDD/PCDF. The cancer risk for the other two receptor populations were 4.8×10^{-5} for the self-sufficient rural individual, and 1.1×10^{-5} for a citizen of Winona. The ingestion of fish exposure pathway dominates this assessment, it accounts for over 91% of the cancer risk.

4.3.1.2 PCB

An analysis of the data indicates that the highest cancer risk due to PCB

emissions was associated with the sportsfishermen receptor group. This cancer risk rate was 1.15×10^{-7}, if this facility emitted 12.9 ng/m^3 PCB. The cancer risk for the other two receptor populations were 8.92×10^{-9} for the self-sufficient rural individual, and 1.85×10^{-9} for citizens of Winona. It is evident that the ingestion of fish exposure pathway dominates this assessment, it accounts for over 82% of the cancer risk. With respect to non-cancer health effects, the highest daily does calculated, i.e., to sportsfishermen was only 0.05% of a conservatively estimated chronic reference dose. It is therefore unlikely that any non-cancer health effects due to PCB emissions from the facility would occur in the surrounding populace.

4.3.1.3 LEAD

Conclusions from an analysis of the lead data indicate that much higher lead emissions would have to be generated for this facility to pose significant health risks for the public from lead exposure. The health risk assessment for lead indicates that the health risk due to lead emission was for the sportsfishermen receptor group. However, the estimated dose for this group was only 10% of the USEPA chronic reference dose. Therefore, it is unlikely that adverse health effects from exposure to lead emissions from the proposed facility would occur in any populace.

4.3.1.4 ARSENIC

From an interpretation of the data, conclusions can be made that the greatest cancer risk due to arsenic exposure was associated with the sportsfishermen receptor group. The cancer risk rate was 1.9×10^{-6}, if this facility emitted 472 ng/m^3 arsenic. The cancer risk for the other two receptor populations were 8.5×10^{-8} for the self-sufficient rural individual, and 4.8×10^{-8} for a citizen of Winona.

With respect to the non-cancer health risk, none of the receptor groups should be a risk. The highest arsenic dose was calculated for the sportsfishermen group. This esimated daily does is only 4.4% of our recommended chronic reference dose.

4.3.2 INTRODUCTION

This capsulized presentation of the Health Risk Assessment for PCDDs/PCDFs, PCBs, lead and arsenic is based on the technical work paper developed by J.P. Stevens and Associates. The purpose of the technical work paper was to evaluate the potential health risks posed to the residents living in or around Winona, Minnesota, from resulting air emissions associated with the proposed Resource Recovery Facility and serves as a supplimentary Draft EIS.

Staff became aware of the problems of using laboratory derived dioxin bioaccumulation data during our investigation of the dioxin problem on the Rainy River. Our contacts with EPA, Environmental Research Laboratory-Duluth led us to understand that the real-life dynamics of fish exposure to dioxin came through the food web and from sediments, not just from the water column as most other classical laboratory bioaccumulation experiments were conducted. Staff felt we needed to address the concepts central to bioaccumulation from sediments and food both at the Rainey River and coincidentally for the risk assessment for the Winona incinerator.

During the review of ICF's paper, staff decided to prepare a supplimentary HRA of limited scope due to economic consideration. This

complimentary review was designed to answer the questions as to how the designated chemicals would partition through the environment relative to the fugaticy model and biomagnification. PCDDs/PCDFs, PCBs, lead and arsenic were determined to be the most significant chemicals corresponding to foodchain impacts. and were specified by staff for review in providing more resolution in conjunction with the pathways identified by staff. These chemicals and concepts were addressed in the model used by J.B. stevens and Assoc.

As outlined in the original Health Risk Assessment (HRA) document for the proposed Resource Recovery Facility in Winona, Minnesota (Clement Associates, Inc., 1987), there are four components of any health risk assessment: hazard identification, exposure assessment, dose-response assessment and risk characterization. Since this study is intended as a supplementary document to the original HRA, not all of the information contained in each HRA component will be reiterated.

4.3.2.1 HAZARD IDENTIFICATION

The Health Risk Assessment for the proposed Resource Recovery Facility in Winona, Minnesota (Clement Associates, Inc., 1987) contains the complete description of both proposed facilities, as well as the surrounding areas. This document, therefore will not further delineate this information. In a meeting held on January 5, 1988, an agreement was reached between J.B. Stevens and Assoicates and the MPCA staff that the health risk associated with emissions of four substances would be evaluated in this technical work paper (TWP): dioxins and furans (PCDD/PCDF), polychlorinated biphenyls (PCB), lead (Pb), and arsenic (As). The emission rate for each of these chemical substances and the air dispersion modeling data were provided to J.B. Stevens & Associates by the MPCA staff. Three receptor populations were also identified at that inital meeting - "typical" urban citizens of Winona, sportsfishermen, and hypothetical self-sufficient rural families.

4.3.2.2 Exposure Assessment

The objective of this technical volume is to translate the estimated ambient air level of the chemical into public health risks to the above designated receptors around this facility. For the most part, our own exposure data and assumptions are utilized. However, new input assumptions (as agreed upon by the MPCA staff and J.B. Stevens & Associates) were utilized, as well as input data from the original HRA, when applicable.

4.3.2.3 DOSE-RESPONSE ASSESSMENT

The dose-response parameters, i.e., reference doses and potency slopes, that were documented by Clement Associates, Inc. (1987) are also used in this assessment.

Information relating the emissions to impacts on fish were newly evaluated, however, with the assistance and input data from the Water Quality Division, MPCA; the Minnesota Department of Health; and the Minnesota Department of Natural Resources.

4.3.2.4 RISK CHARACTERIZATION

The risk characterization procedures followed by Clement Associates, Inc. (1987) were followed in this assessment with the exception that total doses (i.e., from all routes of exposure) rather than route- specific doses (as per the the original HRA) were calculated and equated to health risk.

Three exposure populations were evaluated in this project: urban citizens of Winona; self sufficient rural families in the Winona area, who consume most locally-grown foodstuff; and sportsfisherman who live in Winona and who consume approximately one meal per week consisting of fish caught in the Mississippi River near Winona.

4.3.2.4.1 Typical Winona Urban Resident

The first receptor population or typical urban citizen of Winona is comprised of individuals residing in the City of Winona. Adults, adolescents, toddlers and infants are assumed to be subpopulations in this receptor group. A total exposure dose for each of these subpopulations is initially calculated so that a time-weighted average could be made for an individual's 70-year lifetime. The 70-year convention is used in order that the U.S. EPA unit cancer risk values and our chronic reference dose are applicable.

The following routes of exposure are considered relevant pathways of human exposure for the residents of Winona:

(a) Inhalation of contaminated air

(b) Ingestion of contaminated soil/dust

(c) Ingestion of contaminated foodstuffs (garden vegetables and fruit, agricultural products, and fish)

(d) Dermal contact with contaminated soil/dust

(e) Infant ingestion of breastmilk

It is important to stress that typical (or most probable) behavior pattern are modeled with this receptor population. Probable case exposure assumptions should provide a more accurate estimate of health risk (as compared to worst case behavior patterns).

For this assessment, adult individuals, as well as adolescents (6-17 years of age), toddlers (1-5 years of age) and infants (0-1 year of age), are modeled for chemical exposure. Toddlers would be expected to be exposed to most of the same pathways as adults, as well as through PICA behavior. Infants are assumed to be exposed to chemical emission substances only via ingestion of breastmilk and inhalation of contaminated air. Adolescents are modeled indentically to adults.

4.3.2.4.2 Self-sufficient Rural Population

The second receptor population is composed of self-sufficient rural families who live in the vicinity of Winona. This population also is assumed to be comprised of adults, adolescents, toddlers, and infants. Again, the approach that is used in assessing this receptor population is to calculate an average daily dose to an individual in each subpopulation group. This receptor population is unique from the urban population (receptor group I) mainly with respect to the sources and quantities of agricultural foodstuffs ingested.

As with the urban population in Winona, emissions from the proposed Resource Recovery Facility are also expected to dissipate in the ambient air over agricultural areas near Winona. Therefore, exposure doses to rural individuals from inhalation of airborne contaminants, direct and indirect

ingestion of soil/dust, dermal contact of soil/dust, ingestion of home grown produce and agricultural foodstuffs, and infant ingestion of breastmilk are modeled. The self-sufficient rural families are unique in that they are assumed to grow their own produce. These individuals are also assumed to exclusively ingest their own milk, beef, pork, lamb, chicken and eggs. It is, therefore, important to stress that this receptor group is atypical for most farm families, yet represents for this assessment worst case rural population dose estimates.

4.3.2.4.3 Sportsfishermen

The last receptor population is sportsfishermen who are assumed to catch and eat fish in the Mississippi River backwaters near Winona. This population is assumed to be adults only (both male and female). These individuals are also assumed to live in the City of Winona. Since fish have been shown in a previous HRA to represent the highest single exposure does of any pathway for organochlorine compounds (Stevens and Gerbec, 1986), these individuals many represent the maximally exposed group in this assessment as well.

Sportsfishermen, also Winona residents, have been modeled as a third receptor population because of the larger amounts of fish they are assumed to catch and eat from the Mississippi River and backwaters near Winona.

As mentioned previously, arsenic emitted from the proposed Resource Recovery Facility deposits onto surface waters and partitions through the water column. Arsenic can then accumulate in the tissue of fish that reside in these waters. Thus, ingestion of fish represents a major exposure pathway to sportsfishermen. These individuals, assumed to be adults, are expected to consume fish caught exclusively in the Winona, MN area.

Since the sportsfishermen reside in the urban areas around this facility, they are also expected to be exposed similarly to chemicals, by all of the other routes listed for the urban population (except infant ingestion of breastmilk). Thus, the sportsfishermen receptor group represents the worst case exposure conditions with respect to one single pathway, ingestion of fish. All other exposure routes are probable case, similarly to the urban population. It must be recognized that the dietary patterns of the sportsfisherman receptor postulated in this model may in fact be exceeded by certain individuals.

4.3.2.5 ASSUMPTION USED IN MODEL

Certain generalized assumption are made with respect to PCDD/PCDF, PCBs, lead and arsenic exposure to there receptor population. Therefore, the estimated dose for each population (and the risk associated with that dose) should only be considered representation of the general behavior pattern outlined. It is acknowledged that variability of behavior does occur in each population-variability that could lead to increased risk or decreased risk on an individual basis.

4.3.3 SUPPLEMENTARY HEALTH RISK ASSESSMENT FOR PCDD/PCDF

4.3.3.1 INTRODUCTION

This supplementary Health Risk Assessment for Lead for the Winona Resource Recovery Facility Draft EIS is based on a TWP which was prepared by Jeff Stevens and Associated in conjunction with MPCA and MDOH staff. This assessment evaluated the potential health risk posed to the residents living in or around Winona, Mn. resulting from air emissions of PCDD/PCDF from the proposed facility.

Again, three exposure pathways were evaluated; urban citizens of Winona, self sufficient rural families who consume locally-grown foodstuff and sportsfishermen who live in Winona and consume approximately one meal per week consisting of fish caught in the Minnesota River near Winona. Additionally, the pathways modeled for human exposure were as follows; inhalation of contaminated air; ingestion of contaminated soil/dust; injestion of contaminated foodstuffs (garden produce; fish and agricultural products); dermal contact with contaminated soil/dust; and infant ingestion of breastmilk.

4.3.3.2 PROCEDURES FOR HEALTH RISK ASSESSMENT

As outlined in the original Health Risk Assessment (HRA) document for the proposed Resource Recovery Facility in Winona, Minnesota (Clement Associated Inc., 1987), there are four components of any health risk assessment; hazard identification, exposure assessment, dose-response assessment and risk characterization. Again since this study is intended as a supplementary document to the original HRA, this information will not be reinterated. Please refer to ICF/Clement, TWP, sections VIII for a complete discussion of the Activity and Population Analysis, VI for Hazard Identification, and IX for the Exposure Assessment.

4.3.3.3 HEALTH BASED STANDARDS/REGULATIONS FOR PCDD/PCDF

The U.S. EPA has classified 2,3,7,8-TCDD as a probable human carcinogen. Because the cancer incidence is systemic, i.e., not at the site of chemical contact, all routes of exposure are applicable to calculate total dose. The Carcinogen Assessment Group has established a 10^{-5} unit cancer risk dose of 6.4×10^{-2} pg/kg/d for 2,3,7,8-TCDD (U.S. EPA, 1985).

4.3.3.4 EMISSION RATE OF PCDD/PCDF FROM THE PROPOSED RESOURCE RECOVERY FACILITY IN WINONA, MINNESOTA.

A pre-determined emission rate of 3.0 ng/M^3 of PCDD/PCDF was used in this health risk assessment. This emission rate of 3.0 ng/M^3 has been utilized in past projects as a permit limitation and it is likely that the MPCA would not consider a higher emission limitation for the Winona County RRF. Health risk from a given contaminant will vary on a basis directly proportionate to the emission rate. For this reason, an appropriate emission rate can be back-calculated, given a specific health risk. Conversely, a health risk can be estimated using any given emission factor.

4.3.3.5 AMBIENT AIR

The ambient air concentrations of all contaminants were calculated by Black and Veatch, using the Industrial Source Complex Long Term (ISCLT) air dispersion model (Nelson, 1987.) Both locations of the proposed Resouce Recovery Facility were modeled. Both sites yielded identical results for the

maximal annual average and maximal summer average PCDD/PCDF concentrations in ambient air. The annual average ambient PCDD/PCDF air concentrations, 0.011 pg/M^3, (worst case) and 0.002 pg/M^3 (probable case), are used in all of the exposure pathways modeled in the exposure assessment section of this report except for garden produce/agricultural crops, where the summer months' average PCDD/PCDF concentrations in air are used. These summer months' average ambient air concentrations are 0.016 pg/M^3 (worst case) and 0.003 pg/M^3 (probable case).

It is important to stress that typical (or most probable) behavior patterns are modeled with this receptor population. Probable case exposure assumptions should provide a more accurate estimate of health risk (as compared to worst case behavior patterns).

4.3.3.6 RISK CHARACTERIZATION

4.3.3.6.1 Human Exposure to PCDD/PCDF

In order to calculate the lifetime cancer risks for PCDD/PCDF emissions in this study, a time-weighted average method is used initially to calculate the average daily dose of PCDD/PCDF (D) over a 70-year lifetime of an individual.

Four age groups are modeled in both the urban and rural family populations; adults, adolescents, toddlers and infants. Only adults are modeled in the sportsfishermen population. Infant doses apply for 1 year; toddlers for 5 years; adolescents for 12 years and adults for 52 years.

The doses calculated from all of these routes of exposure were summed to arrive at a total daily PCDD/PCDF exposure dose. Total exposure doses were initially determined for infants, toddlers, adolescents and adults in each of the first two exposure populations. Sportsfishermen were assumed to be adults only. Daily doses in these various age groups were then used to calculate an average daily dose over a 70-year lifetime for each receptor population. These lifetime daily doses were determined using a time-weighted averaging equation wherein infants were modeled for one year, toddlers for five years, adolescents for 12 years and adults for 52 years.

Each lifetime daily dose estimate was compared to the U.S. EPA unit cancer risk dose for 2,3,7,8-TCDD (for quantifying cancer effects).

Total population doses are presented in Tables 4.3.3A, 4.3.3B, and 4.3.3C.

Table 4.3.3A: Summary of Human Doses of PCDD/PCDF to Urban Families in Winona

Route of Exposure	PCDD/PCDF Dose (pg/kg/d) Adults	Adolescents	Toddlers	Infants
Inhalation of Air	6.8 E-4	7.4 E-4	8.4 E-4	9.4 E-4
Ingestion of Soil/Dust	0.9 E-4	1.5 E-4	64.0 E-4	0.0
Ingestion of Garden Produce	22.8 E-4	35.5 E-4	51.2 E-4	0.0
Ingestion of Milk	4.2 E-4	6.7 E-4	23.5 E-4	0.0
Ingestion of Beef	25.0 E-4	28.0 E-4	17.0 E-4	0.0
Ingestion of Other Agricultural Products	0.7 E-4	1.2 E-4	0.1 E-4	0.0
Ingestion of Fish	555.5 E-4	864.2 E-4	926.0 E-4	0.0
Dermal Contact with Soil/Dust	0.1 E-4	0.1 E-4	0.1 E-4	0.0
Ingestion of Breastmilk	0.0	0.0	0.0	24.4 E-4
Total Dose	0.062	0.95	0.109	0.003

The total dose to adults across all routes of exposure for adults is 0.062 pg/kg/d, for adolescents, 0.95 pg/kg/d, for toddlers, 0.109 pg/kg/day and for infants 0.003 pg/kg/d.

Table 4.3.3B: Summary of Human Doses of PCDD/PCDF to Self-sufficient Rural Families living near Winona

Route of Exposure	PCDD/PCDF Dose (pg/kg/d) Adults	Adolescents	Toddlers	Infants
Inhalation of Air	2.2 E-4	1.3 E-4	1.6 E-4	1.7 E-4
Ingestion of Soil/Dust	0.3 E-4	0.4 E-4	11.3 E-4	0.0
Ingestion of Garden Produce	26.7 E-4	41.5 E-4	53.8 E-4	0.0
Ingestion of Milk	1160 E-4	1740 E-4	2320 E-4	0.0
Ingestion of Beef	900.0 E-4	1410.0 E-4	530.0 E-4	0.0
Ingestion of Other Agricultural Products	67.1 E-4	104.5 E-4	3.6 E-4	0.0
Ingestion of Fish	555.5 E-4	864.2 E-4	926.0 E-4	0.0
Dermal Contact with Soil/Dust	0.0 E-4	0.0 E-4	0.0 E-4	0.0
Ingestion of Breastmilk	0.0	0.0		111.4 E-4
Total Dose	0.271	0.416	0.385	0.011

Total dose for rural adults across all routes of exposure is 0.271 pg/kg/d, for adolescents, 0.416 pg/kg/d, for toddlers, 0.385 pg/kg/d and for infants, 0.011 pg/kg/d.

Table 4.3.3C: Summary of Human Doses of PCDD/PCDF to Sportsfishermen in Winona

Route of Exposure	PCDD/PCDF Dose (pg/kg/d)
Inhalation of Air	6.8 E-4
Ingestion of Soil/Dust	0.9 E-4
Ingestion of Garden Produce	22.8 E-4
Ingestion of Milk	4.2 E-4
Ingestion of Beef	25.0 E-4
Ingestion of Other Agricultural Products	0.7 E-4
Ingestion of Fish	5.55
Dermal Contact with Soil/Dust	0.1 E-4
Total Dose	5.55

Total doses for the sportsfishermen is 5.55 pg/kg/d.

Table 4.3.3D summarizes the average lifetime exposure doses of PCDD/PCDF to receptor Populations

Table 4.3.3D: Average Lifetime Exposure Doses of PCDD/PCDF to Receptor Populations

Population	Chronic Exposure Dose (pg/kg/d)
Urban Individual	0.070
Rural Individual	0.307
Sportsfisherman	5.55

4.3.3.6.1.1 Fish Pathway

Because of the significance of PCDD/PCDF relative to fish and the importance of this pathway to humans, a more in depth discussion of this component of the supplementary Health Risk Assessment will be presented here. Partitioning and persistence of PCDD/PCDF in bodies of surface water, e.g. the Mississippi River and backwater 'pools' near Winona, are important components of this health risk assessment because fish represent a major potential source of PCDD/PCDF exposure to several receptor populations. Therefore, we made a significant effort to obtain the most recent information available with respect to PCDD/PCDF modeling in aquatic ecosystems (published information, as well as personal communications).

Due to their insolubility in water, the vast majority of PCDD/PCDF that

enter a surface water system partition to suspended solids, sediment and incorporate into biota (Karickhoff and Morris, 1983; U.S. EPA Duluth, 1987). Thus, these natural organic matter 'sinks' represent the major environmental reservoirs for these chemicals in an aqueous system. Under steady-state conditions, the PCDD/PCDF concentrations in the major reservoirs are predicted to be proportional to each other, provided the chemical levels are expressed as a function of organic carbon mass in each 'sink' (Karickhoff and Morris, 1983; Lake, Rubinstein and Pavignano, 1984). Recent laboratory studies have confirmed this hypothesis (U.S. EPA Duluth, 1987). Therefore, equilibrium constants (termed bioavailability indexes by the cited authors) can be determined that describe this partitioning (the fugacity model approach, Clark et al., 1988).

It is additionally important, however, to recognize that the equilibrium levels of PCDD/PCDF in the various biological 'sinks' are not only dictated by physicochemical properties of these chemicals but also by factors such as foodchain doses and the metabolic degradation rates in each biota. For many organic, insoluble chemicals such as PCDD/PCDF, these factors have been proposed to explain observed chemical accumulation in biota above fugacity levels of sediment (Connolly and Pedersen, 1988).

River Backwater Modeling

The approach that is used in this investigation to model PCDD/PCDF behavior in the Mississippi River backwater pools near Winona consists of four steps:

(a) a calculation of the annual PCDD/PCDF input into a 'representative' pool from the air,

(b) a calculation of the steady-state input concentration of PCDD/PCDF into the bottom sediment,

(c) a calculation of the steady-state input concentration of organic matter into the bottom sediment, and

(d) expression of the steady-state PCDD/PCDF level in the bottom sediment in terms of organic carbon content.

The fraction of water volume settling per year is obtained by the phosphorus retention method developed by Dillon and colleagues (Dillon and Rigler, 1974; Kirchner and Dillon, 1975). This method assumes the back water areas behave as lakes. The method requires the additional following data which was supplied to J. B. Stevens & Associates by the U.S. Army Corps of Engineers and the MN Dept. of Natural Resources: annual average water inflow/outflow rate (5 cfs). This method yields a fractional water volume settling rate of 1.00. In other words, on an annual basis 100% of all suspended solids settle into the sediment. (see Appendix A). (TWP) J.B. Steven & Associates

This calculation thus predicts that 100% of the annual PCDD/PCDF input concentration also will settle into the bottom sediment. Since the average depth in the back waters is estimated to be 0.67M, an annual sediment input of 1044 pg/M^2 can be calculated.

If assumptions are made that PCDD/PCDF do not degrade in the sediment, and that at least 0.2 centimeters of sediment is laid down (Korschgen, et. al., 1987) and mixed each year then the average annual PCDD/PCDF concentration in sediment over this period of time can be calculated as:

$$\text{PCDD/PCDF Concentration in Sediment (pg/g oc)} = \frac{\text{PCDD/PCDF Input Concentration (pg/M}^2\text{)}}{\text{Organic Carbon Mass (g/M}^2\text{)}} \quad (5)$$

Where the organic carbon mass/M^2 is calculated assuming 0.2 cm mixing depth, bottom sediment is composed of 50% water, and that the organic matter content of bottom sediment is 2% (MN DNR, 1988). Under these assumptions a square meter of bottom sediment contains 54 g OC.

Therefore, the bottom sediment annual average PCDD/PCDF concentration is:

$$\frac{1044 \text{ pg/M}^2}{54 \text{ g/M}^2} = 19.3 \text{ pg/g OC.}$$

However, as recently noted by Connolly and Pedersen (1988) field studies have shown that the fugacity model approach underestimates organochlorine chemical concentrations in fish species, especially those chemicals with Kow values greater than 4.0. The data of Connolly and Pedersen indicate that chemical biomagnification occurs, the magnitude of which is dependent on the K_{ow} of the chemical and the trophic level of the aquatic animal. The K_{ow} for 2,3,7,8-TCDD is 4.24×10^6 (Marple, et al., 1986). This molecule was used as an surrogate for PCDD/PCDF because its K_{ow} lies in the intermediate range of all PCDD/PCDF values, this molecule is the most toxic of all PCDD/PCDF, and our emissions rates are expressed in TCDD equivalents. TCDD biomagnification factors of 59 and 23 were calculated for large fish (Walleye, Northern Pike, catfish) and small fish (panfish), respectively (see TWP J.B. Stevens calculations).

To convert these values into specific fish species, one only needs to know the percent lipid in the fish and where the fish resides in the aqueous system (i.e., in the backwater pools or in the river channel). Such information was provided to J. B. Stevens & Associates by the staff of the MPCA (Water Quality Division) and by the Minnesota Department of Health. The sport fish lipid content of 0.84% is the value used in the model for purposes of assessing risk associated with fish consumption. Below is a summary table of these data.

Table 4.3.3E: Lipid Content in Common Minnesota Fish

Common Fish Name	Median % Lipid in Fillets	Location
Mixed Sport fish[a]	0.84	Pools
Sunfish	0.40	Pools
Carp	9.46	Pools
Channel catfish	5.22	Channel
Flathead catfish	4.74	Channel
Walleye	0.70	Pools
Northern Pike	0.50	Pools

a. Pan fish composed primarily of sunfish, walleye, pike, black crappie

and sauger (see Appendix C).

Table 4.3.3F below describes the estimated PCDD/PCDF levels in various fish species due to facility emissions as calculated in this document.

Table 4.3.3F: Worst Case PCDD/PCDF Concentration in Various Fish Species

Fish Species	PCDD/PCDF Concentration (pg/g)
Mixed sport fish	14.9
Sunfish	7.1
Carp	168.0
Channel catfish	1.7
Flathead catfish	1.5
Walleye	31.9
Northern Pike	22.8

4.3.3.6.2 Estimated Cancer Risk from PCDD/PCDF Exposure

Based on the information provided in Table 32, the human population with the highest lifetime daily exposure dose is sportsfishermen. This dose, 3.83 pg/kg/d, equates to a 6.0 $X10^{-4}$ cancer risk.

The other populations would be expected to experience the following cancer risk levels: urban individuals - 1.1 $x10^{-5}$; rural individuals - 4.80 $x10^{-5}$.

4.3.3.6.3 Estimated Non-Cancer Health Risk from PCDD/PCDF Exposure

In the TWP, the hazard evaluation indicates that human exposure to 2,3,7,8-TCDD, whether acute or chronic results in chloracne, altered liver function, hematological problems and altered blood chemistry, prophyria cutanea tarda, hyperpigmentation and hirsutism (Taylor, 1979; Walker and Martin, 1979; Crow, 1981; Hay, 1981). This document addresses carcinogenic impacts because of the potency of this chemical and its potential for foodchain impacts

4.3.3.6.4 Uncertainties in Above Estimates

The cancer risk estimates calculated above have been derived using a series of assumptions about human exposure to PCDD/PCDF and about the dose-response relationships used to quantify the health hazard from this chemical group. Because of these assumptions and estimates, uncertainty is introduced into these final estimates of risks. These uncertainties, however, usually are conservative by design, and thus the final risk estimates are most certainly inflated.

The major uncertainties in this project arise in two areas - estimations of PCDD/PCDF exposure to the receptors, and the downward extrapolation model used to quantify PCDD/PCDF cancer risk at low exposure doses.

Because of the necessary lack of detailed information about the actual population exposures to PCDD/PCDF emitted from the proposed Resource Recovery in Winona (i.e., behavior patterns and PCDD/PCDF environmental fate), many

exposure assessment assumptions had to be made in this assessment. The major assumptions in this exposure assessment include:

(a) Assumption that PCDD/PCDF exposure to the populace is continuous for 70 years (neglects time spent by the receptors out of the impact zone, assumes 70-year life of facility or similar replacement facility)

(b) Assumption that receptor groups reside in the area of highest ambient contaminations (urban citizens and sportsfishermen)

(c) Assumption that all receptors catch and eat fish from backwater areas in the highest impact zone and that these fish are a mixture of species as listed in Appendix C

(d) Assumptions related to the amounts and sources of ingested agricultural foodstuffs by animals and humans

(e) Assumption that TCDD environmental fate data (i.e., K_{ow}) represent behavior for all PCDD/PCDF.

(f) Assumption that TCDD pharmacokinetics in the pig, lamb, and chicken are similar in magnitude to TCDD pharmacokinetics in the cow.

(g) Assumption that the facility will emit PCDD/PCDF at a rate of 3.0 ng/M^3 throughout the period modeled.

(h) Assumptions relating to the concentration of organic carbon in sediment. Assumptions regarding sediment accretion rates in navigation pool six.

(i) Assumptions relating to the bioavailability of PCDD/PCDF.

4.3.3.6.5 Variability of Estimated PCDD/PCDF Cancer Rate Due to Exposure

In order to realize the variability that exists due to the above exposure assessment assumptions, other scenarios (besides the assumptions used in the text of this document) were evaluated. As shown in Table 4.3.3g the exposure assessment assumptions can be subdivided into two general categories--behavior patterns (e.g., how much an individual eats, from where the food originates, etc.) and air modeling [e.g., whether the receptors (or the foodstuffs, etc) are in the 'hot spot' or are located in an area other than the 'hot spot'].

Table 4.3.3G: Variability of PCDD/PCDF Cancer Risk in Winona Health Risk Assessment

Receptor Population	Assumed Behavior Patterns	Air Modeling Assumptions	Estimated PCDD/PCDF Cancer Risk
Urban Citizen	worst case	worst case	1.1×10^{-5}
	worst case	probable case	0.4×10^{-5}
	probable case	worst case	1.1×10^{-5}
	probable case	probable case	0.4×10^{-5}
Rural Citizen	worst case	worst case	12.2×10^{-5}
	worst case	probable case	4.8×10^{-5}
	probable case	worst case	2.7×10^{-5}
	probable case	probable case	0.5×10^{-5}
Sportsfisherman	worst case	worst case	87.0×10^{-5}
	worst case	probable case	30.5×10^{-5}
	probable case	worst case	87.0×10^{-5}
	probable case	probable case	30.5×10^{-5}

____ indicates the scenarios described in the text

a. Worst case: 100% of all pathways in impact zone; probable case: not all of exposure from impact zone

b. Worst case: 'hot spot' approach; probable case: average isopleth approach

Each of the three receptor populations was modeled in four separated scenarios ranging from worst case to probable case. As can be seen with the urban citizen receptor population, the estimated risk seem to be totally dependent upon the air modeling assumptions. This phenomenon occurred because one exposure route, ingestion of fish, was so predominant in this assessment (see Table 4.3.3h). Thus, placing the fish in the 'hot spot' or under an average air isopleth dictates the ultimate risk to this population. This same phenomenon is also true for sportsfishermen, only their level of risk is approximately two orders of magnitude greater than the urban citizen because of their higher intake of locally- caught fish. The other routes of PCDD/PCDF exposure are simply insignificant in comparison.

With respect to the rural citizen receptor population, there is evidence

of influence of the other exposure pathways. The reason for this is that this modeled population is considered self-sufficient, thereby maximizing the agricultural foodchain influence on their total dose. Such an assumption was not made for either the urban individual or the sportsfisherman.

Because of the uncertainty in dose-response modeling at low exposure dose levels, the U.S. EPA has developed and promotes the use of one particular downward extrapolation protocol (U.S. EPA, 1986a). This protocol (the linearized, multi-stage model) is highly conservative in that humans are viewed as the most sensitive animal species to carcinogens, and because biological defense systems (e.g., immune system) are essentially neglected.

Thus, the cancer risk estimates presented in this paper should not be regarded as accurate representations of health risks. Rather, these risks are calculated for use on a comparative basis by risk managers in the decision making process.

4.3.3.6.6 Modeling of Additional Emission Rate

In response to a request by the MPCA staff, other emission rates of PCDD/PCDF from this proposed facility were also modeled (Table 4.3.3H).

Table 4.3.3H: Emission Rates vs. Cancer Risks

Facility Emission		PCDD/PCDF Cancer Risk: Urban Resident	Rural Resident	Sportsfishermen
3.0	ng/M^3 (a)	1.1×10^{-5}	4.8×10^{-5}	87.0×10^{-5}
5.2	ng/M^3 (b)	1.9×10^{-5}	8.3×10^{-5}	150.8×10^{-5}
0.1	ng/M^3 (c)	0.04×10^{-5}	0.2×10^{-5}	2.9×10^{-5}
2.7	ng/M^3	1.00×10^{-5}	4.3×10^{-5}	78.3×10^{-5}
0.6	ng/M^3	0.2×10^{-5}	1.0×10^{-5}	17.4×10^{-5}
0.03	ng/M^3	0.01×10^{-5}	0.07×10^{-5}	1.0×10^{-5}

_____ = operative risk level for permit; other risk estimates vary in consequence.

(a) Emission rate modeled in text
(b) Upper 95% confidence interval value for stock emissions
(c) Feasible PCDD emission with BACT.

As shown in this table, the 3.0 ng/M^3 emission of PCDD/PCDF used for the calculations in this text leads to cancer risk rates of 0.8×10^{-5} for urban residents, 4.1×10^{-5} for rural residents and 60.0×10^{-5} for sportsfishermen. It should be again stressed that these risks are associated only with the behavioral and air modeling assumptions outlined earlier. An emission of 5.2 ng/M^3 was also evaluated. This is the upper 95% confidence interval for PCDD/PCDF stack emissions for other MSW facilities currently in operation with

similar pollution control equipment as specified in the proposed resource Recovery Facility in Winona. This emission leads to cancer risk rates of 1.4 $x10^{-5}$ for urban citizens, 7.1 $x10^{-5}$ for rural citizens and 103.8 $x10^{-5}$ for sportsfishermen. A PCDD/PCDF emission of 0.1 ng/M^3 (feasible unit with BACT) yields cancer risks of 0.03 $x10^{-5}$ for urban residents, 0.14 $x10^{-5}$ for rural residents and 2.0 $x10^{-5}$ for sportsfishermen.

4.3.3.6.7 Conclusions

The doses calculated from all of these routes of exposure were summed to arrive at a total daily PCDD/PCDF exposure dose. Total exposure doses were initally determined for infants, toddlers, adolescents and adults in each of the first two exposure populations. Sportsfishermen were assumed to be adults only. Daily doses in these various age groupds were then used to calculate an average daily dose ver a 70-year lifetime for each receptor population. These lifetime daily doses were determined using a time-weighted averaging equation wherein infacts were modeled for one year, toddlers for five years, adolescents for 12 years and adults for 52 years.

Each lifetime daily dose estimate was compared to the U.S. EPA unit cancer risk dose for 2, 3, 7, 8-TCDD (for quantifying cancer effects). When these comparisons were made, it was determined that the highest cancer risk due to PCDD/PCDF emissions was associated with the sportsfishermen receptor group. This cancer risk rate was determined to be 87.0 $x10^{-5}$, if this facility emitted 3.0 ng/m^3 PCDD/PCDF. The cancer risk for the other two receptor populations were 4.8 $x10^{-5}$ for the self-sufficient rural individual, and 1.1 $x10^{-5}$ for a citizen of Winona. It should be pointed out, however, that these risks are predominately due to one exposure route, ingestion of locally caught fish (see below), and therefore are highly dependent on this particular behavior assumption. For example, the cancer risk associated with the urban population can be expressed on a percentage basis according to exposure pathway:

Lastly, a 1.0 $x10^{-5}$ cancer risk level was selected for each receptor population and the corresponding emission rate from this facility was "back-calculated". This was done in order to determine what emission rate would have to be met in order to meet the Minnesota Department of Health "acceptable" risk level. As can be seen in Table 4.3.3H, an emission rate of 3.75 ng/M^3 corresponds to a 1.0 $x10^{-5}$ cancer risk for the urban population, while an emission rate of 0.73 ng/M^3 corresponds to an acceptable risk level for rural populations around this facility. Because of their much greater risk level, a 1.0 $x10^{-5}$ cancer risk rate for sportsfishermen can only be met with a much lower allowable emission rate, 0.05 ng/M^3.

The cancer risk associated with the urban population can be expressed on a percentage basis according to exposure pathway. Table 4.3.3I represents the percentage of total cancer risk to urban citizens through each exposure pathway. As is evident, the ingestion of fish exposure pathway dominates this assessment; it accounts for over 91% of the cancer risk.

Table 4.3.3I: Percentage of Total Cancer Risk to Urban Citizen

Exposure Pathway	Percentage of Total Cancer Risk
Inhalation of Air	0.7
Ingestion of Soil/Dust	0.1
Ingestion of Garden Produce	3.8
Ingestion of Milk	0.2
Ingestion of Beef	4.1
Ingestion of Other Agricultural Products	0.1
Ingestion of Fish	91.0
Dermal Contact with Soil/Dust	0.9
	100.0

As is evident, the ingestion of fish exposure pathway dominates this assessment; it accounts for over 91% of the cancer risk.

4.3.4 SUPPLEMENTARY HEALTH RISK ASSESSMENT FOR PCBS

4.3.4.1. INTRODUCTION

This supplementary Health Risk Assessment for PCB for the Winona Resource Recovery Facility Draft EIS, is based on a Technical Work Paper (TWP) prepared by Jeff Stevens and Associates in conjunction with MPCA and MDH staff. This assessment evaluated the potential health risk posed to the residents living in or around Winona, Wn. resulting from air emissions of PCBs from the proposed facility.

Again, three exposure pathways were evaluated; urban citizens of Winona, self sufficient rural families who consume locally-grown foodstuff and sportsfishermen who live in Winona and consume approximately one meal per week consisting of fish caught in the Minnesota River near Winona. Additionally the pathways modeled for human exposure were as follows; inhalation of contaminated air; ingestion of contaminated soil/dust; injestion of contaminated foodstuffs (garden produce; fish and agricultural products); dermal contact with contaminated soil/dust; and infant ingestion of breastmilk.

4.3.4.2 PROCEDURES FOR HEALTH RISK ASSESSMENT

As outlined in the original Health Risk Assessment (HRA) document for the proposed Resource Recovery Facility in Winona, Minnesota (Clement Associates Inc., 1987), there are four components of any health risk assessment; hazard identification, exposure assessment, dose-response assessment and risk characterization. Again since this study is intended as a supplementary document to the original HRA, this information will not be reinterated. Please refer to ICF/Clement, TWP, sections VIII for complete discussion of the Activity and Population Analysis, VI for Hazard Identification and IX for the Exposure Assessment.

4.3.4.3 HEALTH BASED STANDARDS/REGULATIONS FOR PCBS

Environmental fate data for all of the PCB isomers is not currently available. Therefore, in this assessment, Arochlor 1254 data was often used to represent chemical behavior of the PCB emissions from this proposed facility. This assumption adds some degree of uncertainty to the final estimated risk values. However, since Archlor 1254 is the most abiquitous PCB mixture in use, the assumption that the PCB emissions from this facility are composed of the same isomer distribution as 1254 affects a view that Arochlor 1254 will be the primary PCB constituent present in the waste stream and that the destruction efficieny of all isomers is equals.

The U.S. EPA (1987a) has classified PCB as a probable human carcinogen. The daily intake does that corresponds to a 10^{-5} lifetime cancer risk is 1.3 ng/kg. Because the cancer incidence is systemic, i.e., not at the site of chemical contact, all routes of exposure to PCB are applicable to calculate total dose.

Occupational exposure limits recommended by the ACGIH (1987) for Arochlor 1254 are:

$$\begin{aligned} \text{TLV} &= 0.5\ \text{mg/M}^3 \\ \text{STEL} &= 1.0\ \text{mg/M}^3 \end{aligned}$$

The NIOSH (1977) criterion is 1.0 mg/M^3 for an exposure of 10 hr/d or 40 hr/wk. This value is set to protect workers from all non-cancer effects.

Based on our review of the toxicology literatue on PCB, we recommend a chronic reference dose of 30.0 ng/kg/d to protect individuals from the non-cancer adverse health effects associated with PCB exposure.

4.3.4.4 AMBIENT AIR

The ambient air concentrations of all contaminants will calculated by Black and Veatch, using the Industrial Source Complex Long Term (ISCLT) air dispersion model (Nelso, 1987). Both locations of the proposed Resouce Recovery Facility were modeled. Both sites yielded identical results for the maximal annual average and maximal summer average PCB concentrations in ambient air. The annual average ambient PCB air concentrations, 0.046 pg/M^3 (worst case) and 0.008 pg/M^3 (probable case), are used in all of the exposure pathways modeled in the exposure assessment section of this report except for garden produce/agricultural crops, where the summer months' average PCB concentrations in air are used. These summer months' average ambient air concentrations are 0.069 pg/M^3 (worst case) and 0.012 pg/M^3 (probable case).

4.3.4.5 EMISSION RATE OF PCB FROM THE PROPOSED RESOURCE RECOVERY FACILITY IN WINONA, MINNESOTA

A pre-determined emission rate of 121 ng/sec (12.9 ng/M^3) of PCB was used in this health risk assessment. This emission rate was agreed upon by J. B. Stevens & Associates and the staff of the MPCA (Air Quality Division) and represents the upper 95% confidence interval, as calculated from data of mass burn incinerators that are currently in operation and are similar in size and design to the proposed Resource Recovery facility in Winona.

4.3.4.6 RISK CHARACTERIZATION

4.3.4.6.1 Human Exposure to PCB

Each lifetime daily dose estimate was compared to the U.S. Environmental Protection Agency (EPA) unit cancer risk dose for PCB (for quantifying cancer effects) and to our estimated chronic reference dose (to quantify non-cancer toxic effects).

The doses calculated from all of these routes of exposure were summed to arrive at a total daily PCB exposure dose. Total exposure doses were initially determined for infants, toddlers, adolescents and adults in each of the first two exposure populations. Sportsfishermen were assumed to be adults only. Daily doses in these various age groups were then used to calculate an average daily dose over a 70-year lifetime for each receptor population. These lifetime daily doses were determined using a time-weighted averaging equation wherein infants were modeled for one year, toddlers for five years, adolescents for 12 years and adults for 52 years.

A summary of PCB exposure to urban families, rural families, and to the sportsfishermen is presented in Tables 4.3.4A, 4.3.4B, and 4.3.4C, respectively.

Table 4.3.4A: Summary of Human Doses of PCB to Urban Families in Winona

Route of Exposure	PCB Dose (pg/kg/d) Adults	Adolescents	Toddlers	Infants
Inhalation of Air	3.2 E-3	3.4 E-3	3.9 E-3	4.4 E-3
Ingestion of Soil/Dust	0.6 E-4	1.0 E-4	44.4 E-3	0.0
Ingestion of Garden Produce	15.8 E-3	24.5 E-3	34.9 E-3	0.0
Ingestion of Milk	1.6 E-3	2.6 E-3	9.4 E-3	0.0
Ingestion of Beef	9.7 E-3	15.1 E-3	7.1 E-3	0.0
Ingestion of Other Agricultural Products	0.5 E-3	0.8 E-3	0.1 E-3	0.0
Ingestion of Fish	10.45	16.25	12.05	0.0
Dermal Contact with Soil/Dust	0.0 E-3	0.0 E-3	0.0 E-3	0.0
Ingestion of Breastmilk	0.0	0.0	0.0	1.42
Total Dose	10.48	16.30	12.15	1.42

The total human doses of PCB to urban families of Winona, for adults across all pathways is 10.48 pg/kg/d, for adolescents 16.30 pg/k/d, for toddlers 12.15 pg/kg/d and for infants 1.42 pg/kg/d.

Table 4.3.4B: Summary of Human Doses of PCB to Self-sufficient Rural Families living near Winona

Route of Exposure	PCB Dose (pg/kg/d) Adults	Adolescents	Toddlers	Infants
Inhalation of Air	5.7 E-3	3.4 E-3	3.9 E-3	4.4 E-3
Ingestion of Soil/Dust	0.2 E-3	0.3 E-3	0.8 E-3	0.0
Ingestion of Garden Produce	16.1 E-3	35.9 E-3	46.5 E-3	0.0
Ingestion of Milk	468.6 E-3	728.9 E-3	937.1 E-3	0.0
Ingestion of Beef	357.1 E-3	555.6 E-3	142.9 E-3	0.0
Ingestion of Other Agricultural Prod.	49.5 E-3	76.2 E-3	2.2 E-3	0.0
Ingestion of Fish	0.15	0.23	0.25	0.0
Dermal Contact with Soil/Dust	0.0 E-3	0.0 E-3	0.0 E-3	0.0
Ingestion of Breastmilk	0.0	0.0	0.0	0.142
Total Dose	1.047	1.630	1.383	0.146

The total human doses of PCB to self-sufficient rural families living near Winona for adults across all routes of exposure is 1.047 pg/kg/d, for adolescents, 1.630 pg/kg/d, for toddlers, 1.383 pg/kg/d and for infants, 0.146 pg/kg/d.

Table 4.3.4C: Summary of Human Doses of PCB to Sportsfishermen in Winona

Route of Exposure	PCB Dose (pg/kg/d)
Inhalation of Air	3.2 E-3
Ingestion of Soil/Dust	0.6 E-3
Ingestion of Garden Produce	15.8 E-3
Ingestion of Milk	1.6 E-3
Ingestion of Beef	9.7 E-3
Ingestion of Other Agricultural Products	0.5 E-3
Ingestion of Fish	14.96
Dermal Contact with Soil/Dust	0.0 E-3
Total Dose	14.99

The total human dose of PCB to sportsfishermen in Winbona, MN is 14.99 pg/kg/d

In order to calculate the chronic health risks from PCB emissions in this study, a time-weighted average method is used initially to calculate an average daily dose of a PCB (D) over a 70-year lifetime of an individual.

Table 4.3.4D represents the average lifetime exposure dose to receptor population.

Table 4.3.4D: Average Lifetime Exposure Doses of PCB to Receptor Populations

Population	Chronic Exposure Dose (pg/kg/d)
Urban Individual	0.24
Rural Individual	1.16
Sportsfisherman	14.99

4.3.4.6.1.1 Fish pathway

Partitioning and persistence of PCB in bodies of surface water, e.g. the Mississippi River and backwater 'pools' near Winona are important components of this health risk assessment because fish represent a major potential source of PCB exposure to several receptor populations. Therefore, we made a significant effort to obtain the most recent information available with respect to PCB modeling in aquatic ecosystems (published information, as well as personal communications). Because PCB represents a major potential source of exposure to several receptor populations as well as the persistence and partitioning of PCB in bodies of surface water, a more indepth discussion will be presented

here.

Due to their insolubility in water, the vast majority of PCB that enter a surface water system partition to suspended solids, sediment and incorporate into biota (Karickhoff and Morris, 1983; US EPA Duluth, 1987). Thus, these natural organic matter 'sinks' represent the major environmental reservoirs for these chemicals in an aqueous system. Under steady-state conditions, the PCB concentrations in the major reservoirs are predicted to be proportional to each other, provided the chemical levels are expressed as a function of organic carbon mass in each 'sink' (Karickhoff and Morris, 1983; Lake, Rubinstein and Pavignano, 1984). Recent laboratory studies have confirmed this hypothesis (US EPA Duluth, 1987). Therefore, equilibrium constants (termed bioavailability indexes by the cited authors) can be determined that describe this partitioning (the fugacity model approach; Clark et al., 1988).

It is additionally important, however, to recognize that the equilibrium levels of PCB in the various biological 'sinks' are over factors such as foodchain doses and the metabolic degradation rates in each biota. For many organic, insoluble chemicals such as PCB, these factors have been proposed to explain observed chemical accumulation in biota above fugacity levels of sediment (Connolly and Pedersen, 1988).

River Backwater Modeling

The approach that is used in this investigation to model PCB behavior in the Mississippi River backwater pools near Winona, MN consists of four steps.

(a) a calculation of the annual PCB input into a 'representative' pool from the air,

(b) a calculation of the steady-state input concentration of PCB into the bottom sediment,

(c) a calculation of the steady-state input concentration of organic matter into the bottom sediment, and

(d) expression of the steady-state PCB level in the bottom sediment in terms of organic carbon content.

The annual average PCB input into the 'representative' surface pool is calculated using the Industrial Source Complex (ISC) long term air dispersion model which is consistent with the modeling procedures used in the other sections of this report. The assumption inherent in this approach is that fish reside in areas over which the highest ambient air PCB levels are predicted. The following equation is used to calculate PCB input to the backwater pools with the air dispersion modeling method.

		a	b	c	
PCB Input to System = (pg/yr)	Ambient Air PCB Concentration (pg/M3)	Particle Deposition Rate (M/yr)	Surface Area of System (M2)		(2)

a. The worst case ambient air level of PCB over the surface water near Winona was previously modeled to be 0.046 pg/M^3 (Section 4.2.1)

b. The particle deposition rate used in this analysis is 94,608 M/yr

(0.3 cm/sec).

c. The surface area of the representative backwater pool is 1.05×10^7 M^2 (MN Dept. of Natural Resources, 1988).

This method yields an annual PCB input level of 4.57×10^{10} pg/yr.

Assuming uniform dispersion of the PCB input into the aquatic system and a water volume of 7.04×10^6 M^3 (1.05×10^7 M^2 x0.67 M average depth), an annual PCB input concentration of 6750 pg/M^3 can be calculated for backwater pool from the following equation:

$$\text{Annual PCB Input Concentration } (pg/M^3/yr) = \frac{\text{PCB Input to System (pg/yr)}}{\text{Volume of System (M3)}} \quad (3)$$

The steady-state PCB input concentration to bottom sediment of this representative pool can be calculated with the following equation:

$$\text{PCB Input Concentration to Sediment } (pg/M^3) = \frac{\text{Annual PCB Input Concentration to Pool } (pg/M3/yr)}{\text{Fraction of Water Volume Settling per Year } (yr^{-1})} \quad (4)$$

The fraction of water volume settling per year is obtained by the phosphorus retention method developed by Dillon and colleagues (Dillon and Rigler, 1974; Kirchner and Dillon, 1975). This method assumes the backwater areas behave as lakes. The method requires the additional following data which was supplied to J. B. Stevens & Associates by the U.S. Army Corps of Engineers and the MN Dept. of Natural Resources: annual average water inflow/outflow rate (5 cfs). This method yields a fractional water volume settling rate of 1.00. In other words, on an annual basis 100% of all suspended solids settle into the sediment (see Appendix A).

This calculation thus predicts that 100% of the annual PCB input concentration also will settle into the bottom sediment. Since the average depth in the backwaters is estimated to be 0.67 M, an annual sediment input of 4523 pg/M^2 can be calculated.

If assumptions are made that PCB do not degrade in the sediment and that at least 0.2 cm of sediment is laid down and mixed each year (Korschgen et al., 1987), then the average annual PCB concentration in sediment over this period of time can be calculated as:

$$\frac{\text{PCB Concentration in Sediment}}{(pg/g\ oc)} = \frac{\text{PCB Input Concentration } (pg/M^2)}{\text{Organic Carbon Mass}} \quad (5)$$

Where the organic carbon mass/M^2 is calculated assuming 0.2 cm mixing depth, bottom sediment is composed of 50% water, and that the organic matter content of bottom sediment is 2% (MnDNR, 1988). Under these assumptions, a square meter of bottom sediment contains 54 g OC.

Therefore, the bottom sediment annual averag PCB concentrations is:

$4523 ng/M^2$

$$\frac{\text{4525 pg/M}^2}{\text{54 g/M}^2} = 83.8 \text{ pg/g OC}$$

The length of the river in the impact zone is estimated to be 8045 M (MN DNR, 1988). The average river velocity in the stretch of the river near Winona, is measured at 1.14 M/sec (MN DNR, 1988; as calculated from an average river flow of 694 M^3/sec). The average river depth in the impact zone is estimated to be 2.5 M (MN DNR, 1988).

Based on these data, the PCB concentration in the river due to plant emissions is 0.39 pg/M^3.

Assuming that the PCB associate totally with the organic matter in the river column, 0.27 g/M^3 [18000 mg/M^3 TSS x 0.15 (fraction OC in TSS)], a PCB concentration of 1.44 pg/g OC can be calculated.

Translation of the Organic Carbon Levels of PCB to Fish Levels

The bioavailability index (BI) approach initially described by Karickhoff and Morris (1983) and refined by Lake et al., 1984) was used to translate PCB concentrations in environmental reservoirs of organic matter to PCB concentration in fish. Theoretically, the above fugacity model predicts a BI value of 0.25.

Therefore, the fugacity PCB concentration in fish can be calculated by the following equation:

$$\begin{array}{c}\text{Fugacity PCB Concentration in Fish} \\ \text{(pg/g lipid)}\end{array} = \frac{\begin{array}{c}\text{PCB Concentration in Particulate Organic Carbon} \\ \text{(pg/g OC)}\end{array}}{\text{BI}} \qquad (10)$$

This calculation leads to a PCB concentrations in fish as listed in Table 4.3.4E.

Table 4.3.4E: Worst Case Fugacity Levels of PCB in Fish

Fish Location	PCB Concentration (pg/g)
Backwater Pools	335
River Channel	5.76

However, as recently noted by Connolly and Pedersen (1988) field studies have shown that this fugacity model approach underestimates organochlorine chemical concentrations in fish species, especially those chemicals with Kow values greater than 4.0. The data of Connolly and Pedersen indicate that chemical biomagnification occurs, the magnitude of which is dependent on the Kow of the chemical and the trophic level of the aquatic animal. The average Kow for a PCBs mixture similar to Arochlor 1254 was calculated to be 1.95×10^6 (Burkhead, 1984). Thus, PCB biomagnification factors of 23.1 and 12.4 were calculated for large fish (Walleye, Northern Pike) and small fish (panfish), respectively (see Appendix B, J.B. Stevens TWP, for calculations).

To convert these values into specific fish species, one only needs to

know the percent lipid in the fish and where the fish resides in the aqueous system (i.e., in the backwater pools or in the river channel). Such information was provided to J. B. Stevens & Associates by the staff of the MPCA (Water Quality Division) and by the Minnesota Department of Health. The sportfish lipid content of 0.84% is the value used in the model for purposes of assessing risk associated with fish consumption. Below is a summary table of these data.

Table 4.3.4F: Lipid Content in Common Minnesota Fish

Common Fish Name	Median % Lipid in Fillets	Location
Mixed Sport fish[a]	0.84	Pools
Sunfish	0.40	Pools
Carp	9.46	Pools
Channel catfish	5.22	Channel
Flathead catfish	4.74	Channel
Walleye	0.70	Pools
Northern Pike	0.50	Pools

a. Pan fish composed primarily of sunfish, walleye, pike, black croppie and sauger (see Appendix C)

Table 4.3.4G below describes the estimated PCB levels in various fish species due to facility emissions as calculated in this document.

Table 4.3.4G: Worst Case PCB Concentration in Various Fish Species

Fish Species	PCB Concentration (pg/g fish)
Mixed sport fish	34.9
Sunfish	16.6
Carp	393.0
Channel catfish	3.7
Flathead catfish	3.4
Walleye	54.2
Northern Pike	38.7

4.3.4.6.2 Estimated Cancer Risk from PCB Exposure

Based on the information provided in Table 30, the human population with the highest lifetime daily exposure dose is sportsfishermen. This dose, 14.99 pg/kg/d, equates to a 1.15×10^{-7} cancer risk.

The other populations would be expected to experience the following cancer risk levels: urban individuals -- 1.85×10^{-9}; rural individuals -- 8.92×10^{-9}.

4.3.4.6.3 Estimated Non-Cancer Health Risk from PCB Exposure

Based on our review of the literature on PCB and our estimation of 'safe' doses for PCB exposure to the general population, chronic non-cancer health effects, would not be expected to occur to the populace in the impact zone of the proposed Resource Recovery Facility.

The highest total PCB dose calculated in this assessment, 14.99 pg/kg/d, for sportsfishermen, is only 0.05% of our recommended chronic reference dose.

4.3.4.6.4 Uncertainties in Above Estimates

The health risk estimates calculated above have been derived using a series of assumptions about human exposure to PCB and about the dose-response relationships used to quantify the health hazard from this chemical group. Because of these assumptions and estimates, uncertainty is introduced into the final estimates of risk. These uncertainties, however, usually are conservative by design, and therefore the final risk estimates are most certainly inflated.

The major uncertainties in this project arise in two areas--estimations of PCB exposure to the receptors, and the downward extrapolation model used to quantify PCB cancer health risk at low exposure doses.

Because of the necessary lack of detailed information about the actual population exposures to PCB emitted from the proposed Resource Recovery in Winona (i.e., behavior patterns and PCB environmental fate), many exposure assessment assumptions had to be made in this assessment. The major assumptions in this exposure assessment include:

(a) Assumption that PCB exposure to the populace is continuous for 70 years (neglects time spent by receptors out of the impact zone, assumes 70-year life of facility or similar replacement facility)

(b) Assumption that receptor groups reside in the area of highest ambient concentrations (urban citizens and sportsfishermen)

(c) Assumption that all receptors catch and eat fish from backwater areas in the highest impact zone and that these fish are a mixture of species as listed in Appendix C

(d) Assumptions related to the amounts and sources of ingested agricultural foodstuffs by animals and humans

(e) Assumption that Arochlor 1254 environmental fate data (i.e., Kow) represent behavior of PCB emitted form facility

(f) Assumption that PCB pharmacokinetics in the pig, lamb and chicken are similar in magnitude to PCB pharmacokinctis in the cow.

(g) Assumption that the facility will emit PCB at a rate of 12.9 ng/m^3 throughout the period modeled.

(h) Assumption relating to the concentration of organic carbon in sediment. Assumption regarding sediment accretion rates in mavigation poool six

(i) Assumptions relating to the bioavailability of PCB.

4.3.4.6.5 Variability of Estimated PCB Cancer Rate due to Exposure

In order to realize the variability that exists due to the above exposure assessment assumptions, other scenarios (besides the assumptions used in the text of this document) were evaluated. As shown in Table 4.3.4H, the exposure assessment assumptions can be subdivided into two general categories--behavior patterns (e.g., how much an individual eats, from where the food originates, etc.) and air modeling [e.g., whether the receptors (or the foodstuffs, etc.) are in the 'hot spot' or are located in an area other than the 'hot spot'].

Table 4.3.4H: Variability of PCB Cancer Risk in Winona Health Risk Assessment

Receptor Population	Assumed Behavior Patterns	Air Modeling Assumptions	Estimated PCB Cancer Risk
Urban Citizens	worst case	worst case	1.89×10^{-9}
	worst case	probable case	5.92×10^{-9}
	probable case--worst case		1.85×10^{-9}
	probable case	probable case	5.92×10^{-9}
Rural Citizens	worst case	worst case	8.92×10^{-9}
	worst case	probalbe case	3.31×10^{-9}
	probable case--worst case		8.92×10^{-9}
	probable case	probable case	2.81×10^{-9}
Sportsfishermen	worst case	worst case	1.15×10^{-7}
	worst case	probable case	4.02×10^{-8}
	probable case	worst case	1.15×10^{-7}
	probable case	probable case	4.02×10^{-8}

____ indicates scenarios described in the text

a. Worst case: 100% of all pathways in impact zone; probable case: not all of exposure from impact

b. Worst case: 'hot spot' approach; probable case: average isopleth approach

Each of the three receptor populations was modeled in four separated scenarios ranging from worst case to probable case. As can be seen with the

urban citizen receptor population, the estimated risk seems to be totally dependent upon the air modeling assumptions. This phenomenon occurred because one exposure route, ingestion of fish, was so predominant in this assessment (see Table 4.3.4I). Thus, placing the fish in the 'hot spot' or under an average air isopleth dictates the ultimate risk to this population. This same phenomenon is also true for sportsfishermen, only their level of risk is two orders of magnitude greater than the urban citizen because of their higher intake of locally-caught fish. The other routes of PCB exposure are simply insignificant in comparison.

With respect to the rural citizen receptor population, there is evidence of influence of the other exposure pathways. The reason for this is that this modeled population is considered nearly self-sufficient, thereby maximizing the agricultural foodchain influence on their total dose. Such an assumption was not made for either the urban individual or the sportsfisherman.

Because of the uncertainty in dose-response modeling at low exposure dose levels, the U.S. EPA has developed and promotes the use of one particular downward extrapolation protocol (U.S. EPA, 1986b). This protocol (the linearized, multi-stage model) is highly conservative in that humans are viewed as the most sensitive animal species to carcinogens, and because biological defense systems (e.g., the immune system) are essentially neglected.

Thus, the cancer risk estimates presented in this paper should not be regarded as accurate representations of health risks. Rather, these risks are calculated for use on a comparative basis by risk managers in the decision making process.

4.3.4.6.6 Modeling of Additional Emission Rates

In response to a request by the MPCA staff, other emission rates of PCB from this proposed facility were also modeled (Table 4.3.4I).

Table 4.3.4I: Emission Rates vs Cancer Risk

	PCB CANCER RISK		
Facility Emission Rate	Urban Resident	Rural Resident	Sportsfisherman
12.9 ng/M^3 (a)	1.85×10^{-9}	8.92×10^{-9}	1.15×10^{-7}
69,700 ng/M^3	1.00×10^{-5}	4.82×10^{-5}	6.24×10^{-4}
14,500 ng/M^3	2.07×10^{-6}	1.00×10^{-5}	1.29×10^{-4}
1120 ng/M^3	1.61×10^{-7}	7.76×10^{-7}	1.00×10^{-5}

(a) Emission rate modeled in text

___ = operative risk level for permit: other risk estimates vary in consequence

As shown in this table, the 12.9 ng/M^3 emission rate used for the calculations in this text leads to cancer risk rates of 1.85×10^{-9} for urban residents, 8.92×10^{-9} for rural residents and 1.15×10^{-7} for sportsfishermen. It should be again stressed that these risks are associated only with the

behavioral and air modeling assumptions outlined earlier.

A 1.0 $x10^{-5}$ cancer risk level was selected for each receptor population and the corresponding emission rate was 'back-calculated'. This was done in order to determine what emission rate would have to be met in order for the facility to meet the Minnesota Department of Health 'acceptable' risk level. An emission rate of 69,700 ng/M^3 from this facility corresponded to a 1.0 x10-5 cancer risk for the urban population, while an emission rate of 14,500 ng/M^3 corresponded to a 10^{-5} risk for the rural population. However, because of their much greater risk level, a 1.0 $x10^{-5}$ cancer risk level for sportsfishermen corresponds to a much lower allowable emission rate, 1120 ng/M^3.

4.3.4.6.7 Conclusions

An analysis of the data indicate that the highest cancer risk due to PCB emissions was associated with the sportsfishermen receptor group. This cancer risk rate was 1.15 $x10^{-7}$, if this facility emitted 12.9 ng/m^3 PCB. The cancer risks for the other two receptor populations were 8.92 $x10^{-9}$ for the self-sufficient rural individual, and 1.85 $x10^{-9}$ for citizen of Winona. It should be pointed out, however, that these risks are predominatly due to one exposure route, ingestion of lcoally caught fish (see below), and therefore are highly dependent on this particular behavior assumption. For example, the cancer risk associated with the urban population can be expressed on a percentage basis according to exposure pathway:

URBAN CITIZENS

Exposure Pathway	Percentage of Total Cancer Risk
Inhalation of Air	2.043
Ingestion of Soil/Dust	0.330
Ingestion of Garden Produce	8.740
Ingestion of Milk	0.783
Ingestion of Beef	5.157
Ingestion of Other Agricultural Products	0.274
Ingestion of Fish	82.673
Dermal Contact with Soil/Dust	0.000
	100.000

As is evident, the ingestion of fish exposure pathway dominates this assessment; it accounts for over 82% of the cancer risk.

With respect to non-cancer health effects, the highest daily dose calculated, i.e., to sportsfishermen, was only 0.05% of a conservatively estimated chronic reference dose. It is therefore unlikely that any non-cancer health effects due to PCB emissions from the facility would occur in the surrounding populace.

4.3.5 SUPPLEMENTARY HEALTH RISK ASSESSMENT FOR LEAD

4.3.5.1 INTRODUCTION

This supplementary Health Risk Assessment for lead for the Winona Resource Recovery Facility Draft EIS is based on Technical Work Paper prepared by Jeff Stevens and Associates in conjunction with MPCA and MDOH staff. This assessment evaluates the potential health risk posed to the residents living in or around Winona resulting from air emissions of lead from the proposed facility.

Again, three exposure pathways were evaluated; urban citizens of Winona, self sufficient rural families who consume locally-grown foodstuff and sportsfishermen who live in Winona and consume approximately one meal per week consisting of fish caught in the Minnesota River near Winona. Additionally, the pathways modeled for human exposure were as follows; inhalation of contaminated air; ingestion of contaminated soil/dust; injestion of contaminated foodstuffs (garden produce; fish and agricultural products); dermal contact with contaminated soil/dust; and infant ingestion of breastmilk.

4.3.5.2 PROCEDURES FOR HEALTH RISK ASSESSMENT

As outlined in the original Health Risk Assessment (HRA) document for the proposed Resource Recovery Facility in Winona, Minnesota (Clement Associates Inc., 1987), there are four components of any health risk assessment; hazard identification, exposure assessment, dose-response assessment and risk characterization. Again since this study is intended as a supplementary document to the original HRA, this information will not be reinterated. Please refer to ICF/Clement, TWP, sections VIII for complete discussion of the Activity and Population Analysis, VI for Hazard Identification and IX for the Exposure Assessment.

4.3.5.3 HEALTH BASED STANDARDS/REGULATIONS FOR LEAD

The U.S. EPA (1980) has set ambient water quality criteria for the protection of humans from the chronic exposure effects of lead. This ambient water level is 50 ug/L. It has recently been proposed to lower this level to 20 mg/L (Fed. Reg. 50: 46969, 1988). The US EPA has also issued a health advisory for lead based on estimated NOAEL values in children and pregnant women. These NOAEL values are expressed as mg Pb/dl blood. In both cases blood Pb level of 15 mg/dl has been judged to be acceptable (U.S. EPA, 1985).

Conversion of the blood Pb level to Pb intake doese has been accomplished by the U.S. EPA in the health effects advisory. This conversion leads to an acceptable daily intake of 20 mg of Pb, which would apply to adults and children. Thus, this daily dose can be viewed as a reference dose.

For this assessment daily reference doses for all subpopulations must be calculated so that Hazard Indexes can be determined for infants, toddlers, adolescents and adults. This subpopulation doses are 4.0 mg/kg/d for infants, 1.4 mg/kg/d for toddlers, 0.4 mg/kg/d for adolescents and 0.3 mg/kg/d for adults.

4.3.5.4 EMISSION RATE OF LEAD FROM THE PROPOSED RESOURCE RECOVERY FACILITY IN WINONA

A pre-determined emission rate of 13.6 mg/sec (1.45 mg/M3) of lead was used in this health risk assessment. This emission rate was agreed upon by J. B. Stevens & Associates and the staff of the MPCA (Air Quality Division) and represents the upper 95% confidence interval, as calculated from data of mass burn incinerators that are currently in operation and are similar in size and design to the proposed Resource Recovery Facility in Winona.

4.3.5.5 AMBIENT AIR

The ambient air concentrations of all contaminants were calculated by Black and Veatch, using the Industrial Source Complex Long Term (ISCLT) air dispersion model (Nelson, 1987). Both locations of the proposed Resouce Recovery Facility were modeled. Both sites yielded identical results for the maximal annual average and maximal summer average arsenic concentrations in ambient air. The annual average ambient arsenic air concentrations, 1.67 pg/M3 (worst case) and 0.29 pg/M3 (probable case), are used in all of the exposure pathways modeled in the exposure assessment section of this report except for garden produce/agricultural crops, where the summer months' average arsenic concentrations in air of ng/M3 are used. These summer months' average ambient air concentrations are 2.51 pg/M3 (worst case) and 0.44 pg/M3 (probable case).

4.3.5.6 RISK CHARACTERIZATION

4.3.5.6.1 Human Exposure to Lead Exposure

In order to calculate the chronic health risks from lead, emissions in this study, both the average lifetime daily lead dose and each specific subpopulation lead doses need to be compared to their respective reference doses. A time-weighted average method was used to calculate an average daily dose of a lead over a 70-year lifetime of an individual. Four age groups are modeled in both the urban and rural family populations; adults, adolescents, toddlers, and infants. Only adults are modeled in the sportsfishermen population. Infant doses apply for one year; toddlers for five years; adolescents for 12 years and adults for 52 years.

The doses calculated from all of these routes of exposure were summed to arrive at a total daily lead exposure dose. Total exposure doses were initially determined for infants, toddlers, adolescents and adults in each of the first two exposure populations. Sportsfishermen were assumed to be adults only. Daily doses in these various age groups were then used to calculate an average daily dose over a 70-year lifetime for each receptor population. These lifetime daily doses are determined using a time-weighted averaging equation wherein infacts were modeled for one year, toddlers for five years, adolescents for 12 years and adults for 52 years.

Each lifetime daily dose estimate was compared to the U.S. EPA chronic exposure reference dose for lead (for quantifying non-cancer toxic effects).

Tables 4.3.5A, 4.3.5B, and 4.3.5C represent a summary of human doses of lead to urban families, rural families and sportsfishermen of Winona.

Table 4.3.5A: Summary of Human Doses of Lead to Urban Families in Winona

Route of Exposure	Lead Dose (ng/kg/d) Adults	Adolescents	Toddlers	Infants
Inhalation of Air	0.55	0.60	0.68	0.71
Ingestion of Soil/Dust	0.09	0.14	6.19	0.00
Ingestion of Garden Produce	0.81	1.26	1.79	0.00
Ingestion of Milk	0.00	0.00	0.02	0.00
Ingestion of Beef	0.00	0.00	0.00	0.00
Ingestion of Other Agricultural Products	0.00	0.01	0.00	0.00
Ingestion of Fish	0.01	0.02	0.02	0.00
Dermal Contact with Soil/Dust	0.02	0.04	0.04	0.00
Ingestion of Breastmilk	0.00	0.00	0.00	0.05
Total Dose	1.48	2.07	8.74	0.76

The total dose for urban adults over all routes of exposure is 1.48 ng/kg/day and 2.07 ng/kg/day, 8.74 ng/kg/day and 0.76 ng/kg/day for adolescents, toddlers and infants respectively.

Table 4.3.5B: Summary of Human Doses of Lead to Rural Families in Winona

Route of Exposure	Lead Dose (ng/kg/d) Adults	Adolescents	Toddlers	Infants
Inhalation of Air	0.10	0.10	0.12	0.13
Ingestion of Soil/Dust	0.02	0.03	1.09	0.00
Ingestion of Garden Produce	1.13	1.74	2.25	0.00
Ingestion of Milk	0.76	1.18	1.52	0.00
Ingestion of Beef	0.05	0.08	0.02	0.00
Ingestion of Other Agricultural Products	0.08	0.12	0.04	0.00
Ingestion of Fish	0.01	0.02	0.02	0.00
Dermal Contact with Soil/Dust	0.00	0.01	0.01	0.00
Ingestion of Breastmilk	0.00	0.00	0.00	0.07
Total Dose	2.15	3.28	5.07	0.20

The total dose for adults of rural families over all routes of exposure is 2.15 ng/kg/day and 3.28 ng/kg/day, 5.07 ng/kg/day and 0.20 ng/kg/day for adolescents, toddlers and infants respectively

Table 4.3.5C: Summary of Human Doses of Lead to Sportsfishermen in Winona

Route of Exposure	Lead Dose (ng/kg/d)
Inhalation of Air	0.55
Ingestion of Soil/Dust	0.09
Ingestion of Garden Produce	0.81
Ingestion of Milk	0.00
Ingestion of Beef	0.00
Ingestion of Other Agricultural Products	0.00
Ingestion of Fish	1.46
Dermal Contact with Soil/Dust	0.02
Total Dose	2.93

The total dose of lead to sportsfishermen is 2.93 ng/kg/day.

Table 4.3.5D lists the average lifetime exposure doses of lead to receptor populations.

Table 4.3.5D: Average Lifetime Exposure Doses of Lead to Receptor Populations

Population	Chronic Exposure Dose (ng/kg/d)
Urban Individual	2.09
Rural Individual	2.52
Sportsfisherman	2.93

Specific subpopulation daily lead doses are given below in Table 4.3.5E.

Table 4.3.5E: Subpopulation Daily Doses of Lead

Urban Familes	Daily Lead Doses Ng/kg/day
Adults	1.48
Adolescents	2.07
Toddlers	8.74
Infants	0.76
Self-sufficient Rural Families	
Adults	2.15
Adolescents	3.28
Toddlers	5.07
Infants	0.20
Sportfisherman	2.93

4.3.5.6.2 Estimated Cancer Risk from Lead Exposure

The EPA Air Quality Criteria Document for Lead reviews the major animal studies that have been conducted to determine the carcinogenic potential of lead (U.S. EPA, 1986) It is clear from this review that lead will induce renal tumors in mice and rats at toxic levels (1.0 and 0.1% lead in the diet). Most of the studies are not useful though, because they employ dose levels that significantly increase the rate of mortality of the animals (i.e., greater than the maximum tolerated dose). At the present time EPA does not recognize lead as carcinogen and this document thus addresses non-carcinogenic endpoints only.

4.3.5.6.3 Estimated Non-Cancer Health Risk from Lead Exposure

Based on our review of the scientific literature and applicable standard on lead, non-cancer health effects would not be expect to occur to any populace in the impact zone of the propose Resource Recovery Facility. Table 4.3.5F discusses the hazard indexes estimated for each subpopulation as the lifetime hazard index for each receptor group.

As defined by the U.S. EPA, the hazard index (HI)for a chemical is calculated for noncarcinogenic effectgs (subchronic as well as chronic) by the equation below:

$$HI = \frac{\text{Estimated Exposure Dose to Receptor (mg/kg/d)}}{\text{Reference Dose (mg/kg/day)}}$$

Hazard indexes greater than 1.00 indicate potential for adverse health effects. It should be again stressed that these risks are associated only with the behavioral and air modeling assumptions outlined earlier

Table 4.3.5F: Hazard Indexes for Lead Exposure

Receptors Group/Subpopulation	Hazard Index Subpopulation	Hazard Index Lifetime
Urban Citizens	----	0.007
Adult	0.005	----
Adolescent	0.005	----
Toddler	0.006	----
Infant	0.000	----
Rural Citizen	----	0.008
Adult	0.007	----
Adolescent	0.008	----
Toddler	0.004	----
Infant	0.000	----
Sportsfisherman	----	0.010

As is evident none of the hazard indexes calculated is greater than 1.00. The highesty value is 0.010, to the sportsfisherman. This is only 1.0% of the acceptable daily intake estimated for Pb as established by the U.S. EPA (U.S.

EPA, 1985) The most sensitive targets, e.g. toddlers have approximately one-half this highest calacuted dose.

4.3.5.6.4 Uncertainties in Estimates

The risk estimates calculated above have been derived using a series of assumptions about human exposure to lead and about the dose-response relationship used to quantify the health hazard from exposure to this metal. Because of these assumptions and estimates, uncertainty is introduced into this final estimate of risk. These uncertainties, however, usually are conservative by design, and thus the final risk estimate is most certainly inflated.

The major uncertainties in this project arise in the estimation of lead exposure to the receptors. Because of the lack of detailed information about the actual population exposures to lead emitted from the proposed Resource Recovery Facility in Winona, MN, (i.e. behavior patterns and lead environmental fate), many exposure assessment assumptions had to be made. The major assumptions in this exposure assessment include:

(a) Assumption that lead exposure to the populace is continuous for 70 years (neglects time spent by receptors out of the impact zone, asumes 70-year life of facility or similar replacement facility).

(b) Assumption that receptor groups reside in the area of highest ambient concentrations (urban citizens and sportsfishermen).

(c) Assumptions related to the amounts and sources of ingested agricultural foodstuffs by animals and humans.

(d) Assumption that all receptors catch and each fish from the backwater areas in the highest impact zone.

(e) Assumption that the facility will emit lead at a rate of 145 mg/m3 throughout the period modeled.

(f) Assumptions relating to the bioavailability of lead.

4.3.5.6.5 Variability due to Exposure Assessment

In order to realize the variability that exists due to the above exposure assessment assumptions, other scenarios (besides the assumptions used in the text of this document) were evaluated. As shown in Table 4.3.5G, the exposure assessment assumptions can be subdivided into two general categories - behavior patterns (e.g., how much an individual eats, from where the food originates, etc.) and air modeling [e.g., whether the receptors (or the foodstuffs, etc.) are in the 'hot spot' or are located in an area other than the 'hot spot'].

Table 4.3.5G: Variability of Health Risk from Lead Exposure in the Winona Health Risk Assessment

Receptor Population	Assumed Behavior Patternsa	Air Modeling Assumptionsb	Estimated Hazard Index
Urban Citizen	worst case	worst case	0.008
	worst case	probable case	0.004
	probable case	worst case	0.007
	probable case	probable case	0.001
Rural Citizen	worst case	worst case	0.012
	worst case	probable case	0.008
	probable case	worst case	0.007
	probable case	probable case	0.004
Sportsfisherman	worst case	worst case	0.010
	worst case	probable case	0.006
	probable case	worst case	0.009
	probable case	probable case	0.004

____ indicates scenarios described in the text

a. Worst case: 100% of all pathways in impact zone; probable case: not all of exposure from impact

b. Worst case: 'hot spot' approach; probable case: average isopleth approach

Each of the three receptor populations was modeled in four separated scenarios ranging from worst case to probable case. As can be seen with the urban citizen receptor population, there is about a 8-fold variation in risk. Much less variability is observed in the other two populations.

4.3.5.6.6 Modeling of Additional Emission Rates

In response to a request by the MPCA staff, other emission rates of lead from this proposed facility were also modeled (Table 4.3.5H).

Table 4.3.5H: Emission Rates vs Health Risk

Facility Emission Rate	LEAD RISK Urban Resident	Rural Resident	Sportsfisherman
1.45 mg/M3 (a)	0.007	0.008	0.010
207.0 mg/M3	1.00	1.20	1.40
181.0 mg/M3	0.84	1.00	1.17
145.0 mg/M3	0.72	0.86	1.00

(a) Emission rate modeled in text

___ = operative risk level for permit: other risk estimates vary in consequence

As shown in this table (4.3.5H), the 1.45 mg/M3 emission rate used for the calculations in the text leads to hazard indexes of 0.007 for urban residents, 0.008 for rural residents and 0.010 for sportsfishermen. As defined by the U.S. EPA, the hazard index (HI) for a chemical is calculated for non-carcinogenic effects (subchronic as well as chronic) by the equation below:

$$HI = \frac{\text{Estimated Exposure Dose to Receptor (mg/kg/d)}}{\text{Reference Dose (mg/kg/d)}} \quad (46)$$

It should be again stressed that these risks are associated only with the behavioral and air modeling assumptions outlined earlier.

A hazard index of 1.00 was arbitrarily set for each receptor population modeled in this assessment so that some measure of lead emissions in exceedence of the U.S. EPA reference dose could be seen. As is evident in Table 4.3.5H, much higher lead emissions would have to be generated for this facility to pose significant health risks to the public from lead exposure. However, it should be noted that the current contaminant levels of lead in the vicinity of the proposed facilities also need to be evaluated for background lead levels.

Each lifetime daily dose estimate was compared to the U.S. EPA chronic exposure reference dose for lead for quantifying non-cancer toxic effects. When these comparisons were made, it was determined that the greatest health risk due to lead emissions was to the sportsfishermen receptor group. However, the estimated dose for this group was only 1% of the U.S. EPA chronic reference dose. Therefore, it is unlikely that advese health effects from exposure to lead emissions from the proposed facilities would occur in any poupulace

4.3.5.6.7 Conclusion

An analysis of the data indicate that lead emissions would have to be generated for this facility to pose significant health risks to the public from lead exposure. However, it should be noted that the current contaminant levels of lead in the vicinity of the proposed facilities also need to be evaluated for background lead levels. The health risk assessment for lead indicate that the health risk due to lead emissions was to the sportsfishermen receptor group. However, the estimated dose for this group was only 1% of the U.S. EPA chronic reference dose. Therefore, it is unlikely that adverse health effects

from exposure to lead emissions from the proposed facilities would occur in any populace.

4.3.6 SUPPLEMENTARY HEALTH RISK ASSESSMENT FOR ARSENIC

4.3.6.1 INTRODUCTION

This supplementary Health Risk Assessment of arsenic for the Winona Resource Recovery Facility Draft EIS is based on Technical Work Paper prepared by Jeff Stevens and Associates in conjuction with MPCA and MDH staff. This assessment evaluates the potential health risk posed to the residents living in or around Winona resulting from the air emissions of arsenic from the proposed facility.

Again, three exposure populations were evaluated in this project; urban citizens of Winona; self-sufficient rural families in the Winona area, who consume mostly locally-grown foodstuff, and sportsfishermen who live in Winona and who consume approximately one meal per week consisting of fish caught in the Mississippi River near Winona. Additionally, the pathways modeled for human exposure were as follows: inhalation of contaminated air; ingestion of contaminated soil/dust; injestion of contaminated foodstuffs (garden produce, fish and agricultural products); dermal contact with contaminated soil/dust; and infant ingestion of breastmilk.

4.3.6.2 PROCEDURES FOR HEALTH RISK ASSESSMENT

As outlined in the original Health Risk Assessment (HRA) document for the proposed Resource Recovery Facility in Winona, Minnesota (Clement Associates, Inc., 1987), there are four components of any health risk assessment; hazard identification, exposure assessment, dose-response assessment and risk characterization. Again since this study is intended as a supplementary document to the original HRA, this information will not be reiterated. Please refer to ICF/Clement, TWP, sections VII for complete discussion of the Activity and Population Analysis, VI for Hazard Identification and IX for the Exposure Assessment.

4.3.6.3 HEALTH BASED STANDARDS/RECOMMENDATIONS FOR ARSENIC

Arsenic is classified as a human carcinogen by both the inhalation and the oral routes of exposure (U.S. EPA, 1984). The 10^{-5} unit cancer risk dose by the oral route of exposure is 0.67 ng/kg/d, while the 10^{-5} unit cancer risk dose for arsenic from the inhalation route of exposure is 0.20 ng/kg/d.

These two unit risk doses cannot be equated, however, since both describe tissue-specific cancer induction potentials (e.g., respiratory system cancers and gastrointestinal and systemic cancers). Therefore, for the risk characterization section of this report only the inhalation and oral routes of exposure doses are used to assess the cancer risk due to arsenic exposure. The total arsenic cancer risk, however, is determined by summing these two route-specific cancer risks.

4.3.6.4 EMISSION RATE OF ARSENIC FROM THE PROPOSED RESOURCE RECOVERY FACILITY IN WINONA

A pre-determined emission rate of 4.40 ug/sec (472 ng/M^3 of arsenic was used in this health risk assessment. This emission rate was agreed upon by J.B. Stevens & Associates and the staff of the MPCA (Air Quality Division) and represents the upper 95% confidence interval, as calculated from data of mass burn incinerators that are currently in operation and are similar in size and design to the proposed Resource Recovery Facility in Winona.

4.3.6.5 AMBIENT AIR

The ambient air concentration of all contaminants were calculated by Black and Veatch, using the Industrial Source Complex Long Term (ISCLT) air dispersion model (Nelson, 1987). Both sites yielded identical results for the maximal annual average and maximal summer average arsenic concentrations in ambient air. The annual average ambient arsenic air concentrations, 1.67 pg/M^3 (worst case) and 0.29 pg/M^3 (probable case), are used in all of the exposure pathways modeled in the exposure assessment section of this report except for garden produce/agricultural crops, where the summer months' average arsenic concentrations in air of are used. These summer months' average ambient air concentrations are 2.51 pg/M^3 (worst case) and 0.44 pg/M^3 (probable case).

4.3.6.6 RISK CHARACTERIZATION

4.3.6.6.1 Human Exposure to Arsenic

The total arsenic dose from all routes of exposure were for determining the non-cancer health risks from arsenic exposure. Stevens and Associates recommended a chronic exposure reference dose of 2.9 ng/kg/d. This recommended dose is one-tenth of the lowest doses of arsenic reported in the literature to cause non-cancer health effects via chronic exposure (Tseng, 1977).

In order to calculate any potential non-cancer health risks due to arsenic exposure, the doses calculated from all of these routes of exposure were summed to arrive at a total arsenic exposure dose. Total exposure doses were initially determined for infants, toddlers, adolescents and adults in each of the first two exposure populations. Sportsfishermen were assumed to be adults only. Daily doses in these various age groups were then used to calculate an average daily dose over a 70-year lifetime for each receptor population. These lifetime daily doses were determined using a time-weighted averaging equation, wherein infants were modeled for one year, toddlers for five years, adolescents for 12 years and adults for 52 years. Each lifetime total daily dose was compared to our recommended chronic exposure reference doses (for quantifying non-cancer health effects).

In order to calculate the cancer risk to each receptor population from exposure to arsenic, both the inhalation dose and the oral dose for each receptor population were calculated. These two lifetime daily doses were then compared to their corresponding U.S. EPA 10^{-5} unit cancer risk doses. Total cancer risk to each population was then calculated by summing the two above risk estimates. A time weighted average was used to calculate an average daily dose of arsenic over a 70-year lifetime of an individual. Four age groups are modeled in both the urban and rural family populations; adults, adolescents, toddlers and infants. Only adults are modeled in the sportsfishermen population.

The following tables (Tables, 4.3.6A, 4.3.6B, 4.3.6C) lists a summary of the human doses of arsenic to urban families, self-sufficient rural families and sportsfishermen respectively for Winona. Total population doses are presented here.

Table 4.3.6A: Summary of Human Doses of Arsenic to Urban Families in Winona

	Arsenic Dose (pg/kg/d)			
Route of Exposure	Adults	Adolescents	Toddlers	Infants
Inhalation of Air	0.18	0.19	0.22	0.25
Ingest. of Soil/Dust	0.06	0.10	4.44	0.00
Ingest. of Garden Produce	0.59	0.93	1.33	0.00
Ingestion of Milk	0.00	0.00	0.00	0.00
Ingestion of Beef	0.00	0.00	0.00	0.00
Ingestion of Other Agri. Products	0.02	0.03	0.00	0.00
Ingestion of Fish	1.27	1.98	2.12	0.00
Dermal Contact with Soil/Dust	0.00	0.00	0.00	0.00
Ingest. of Breastmilk	0.00	0.00	0.00	0.00
Total Dose	2.12	3.23	8.11	2.12

In urban families the total doses of lead across all routes of exposure for adults is 2.12 pg/kg/day, for adolescents, 3.23 pg/kg/day, for toddlers, 8.11 pg/kg/day and for infants, 2.12 pg/kg/day.

Table 4.3.6B: Summary of Human Doses of Arsenic to Rural Families in Winona

	Arsenic Dose (pg/kg/d)			
Route of Exposure	Adults	Adolescents	Toddlers	Infants
Inhalation of Air	0.03	0.03	0.04	0.04
Ingest. of Soil/Dust	0.02	0.03	0.77	0.00
Ingestion of Garden Produce	0.84	1.31	1.28	0.00
Ingestion of Milk	0.18	0.27	0.35	0.00
Ingestion of Beef	0.00	0.00	0.00	0.00
Ingestion of Other Agri. Products	2.81	4.37	0.02	0.00
Ingestion of Fish	1.27	1.98	2.12	0.00
Dermal Contact with Soil/Dust	0.00	0.00	0.00	0.00
Ingest. of Breastmilk	0.00	0.00	0.00	4.54
Total Dose	5.15	7.99	4.58	4.58

In rural families, total doses for arsenic across all pathways for adults is 5.15 pg/kg/day, for adolescents is 7.99 pg/kg/day, for toddlers is 4.58 pg/kg/day, and for infants is 4.58 pg/kg/day.

Table 4.3.6C: Summary of Human Doses of Arsenic to Sportsfishermen in Winona

Route of Exposure	Arsenic Dose (pg/kg/d)
Inhalation of Air	0.18
Ingestion of Soil/Dust	0.06
Ingestion of Garden Produce	0.59
Ingestion of Milk	0.00
Ingestion of Beef	0.00
Ingestion of Other Agricultural Products	0.02
Ingestion of Fish	127.30
Dermal Contact with Soil/Dust	0.00
Total Dose	128.15

For the sportsfisherman of Winona the total dose of arsenic across all routes of exposure is 128.15 pg/kg/day

The following lifetime average daily doses are presented in table 4.3.6D.

Table 4.3.6D: Average Lifetime Exposure Doses of Arsenic to Receptor Populations

Population	Total Exposure Dose (pg/kg/d)	Inhalation Exposure Dose (pg/kg/d)	Oral Expos. Dose (pg/kg/d)
Urban Individual	2.74	0.19	2.55
Rural Individual	5.57	0.03	5.56
Sportsfisherman	128.15	0.18	127.97

4.3.6.6.2 Estimated Cancer Risk from Arsenic Exposure

Arsenic is considered a carcinogen from both the oral route of exposure and the inhalation route of exposure. Therefore, to calculate the total cancer risk for each receptor group, each individual route-specific cancer risk must first be determined:

Below in Table 4.3.6E are the route-specific cancer risks for each receptor population, as well as their total cancer risk from exposure to arsenic.

Table 4.3.6E: Cancer Risks of Receptor Groups

	Receptor Group		
Cancer Risk	Urban Citizen	Rural Citizen	Sportsfisherman
Inhalation	9.5×10^{-9}	1.5×10^{-9}	9.0×10^{-9}
Oral	3.8×10^{-8}	8.3×10^{-8}	1.9×10^{-6}
Total	4.8×10^{-8}	8.5×10^{-8}	1.9×10^{-6}

As seen in Table 4.3.6E the receptor group that has the greatest cancer risk is sportsfishermen, and that essentially all of their cancer risk is due to ingestion of arsenic. The ingestion route of exposure is also the dominant risk route for the other two receptor populations, but not nearly to the cause extent as in sportsfishermen.

4.3.6.6.3 Estimated Non-Cancer Health Risk from Arsenic Exposure

Based on our review of the literature on arsenic and our estimation of 'safe' chronic exposure doses for arsenic exposure to the general population, chronic non-cancer health effects due to arsenic exposure should not occur to the populace in the impact zone of the proposed Resource Recovery Facility. The maximum total dose of arsenic calculated for any of these exposure populations is 128.15 pg/kg/d. This calculated dose is 4.4% of our recommended chronic reference dose.

4.3.6.6.4 Uncertainties in Above Estimates

The health risk estimates calculated above have been derived using a series of assumptions about human exposure to chemicals and about the dose-response relationships used to quantify the health hazards from exposure to this metal. Because of these assumptions and estimates, uncertainty is introduced into the final estimates of risks. These uncertainties, however, are usually conservative by design, and thus the final risk estimates are most certainly inflated.

The major uncertainties in this project arise in the estimations of chemical exposure to the receptors and the downward extrapolation model used to quantify cancer risk at low exposure doses.

Because of the lack of detailed information about the actual population exposures to arsenic emitted from the proposed Resource Recovery Facility in Winona (i.e., behavior patterns and the environmental fate of As), many exposure assessment assumptions had to be made. The major assumptions in this exposure assessment include:

(a) Assumption that arsenic exposure is continuous for 70 years (neglects time spent by individuals out of the impact zone, assumes 70-year life of facility or similar replacement facility).

(b) Assumption that receptor groups reside in the area of highest

ambient concentrations (urban citizens and sportsfishermen).

(c) Assumption that all receptors catch and eat fish from backwater areas in the highest impact zone, and that these fish are a mixture of species as listed in Appendix B.

(d) Assumptions related to the amounts and sources of ingested foodstuffs by animals and humans.

(e) Assumption that the arsenic input to the backwater areas is 100% bioavailable to fish.

(f) Assumption that the facility will emit as at a rate of 472 ng/m^3 throughout the period modeled.

4.3.6.6.5 Variability of Estimated Arsenic Cancer Rate due to Exposure Assessment Assumptions

In order to realize the variability that exists due to the above exposure assessment assumptions, other scenarios (besides the assumptions used in the text) were evaluated. As shown in Table 4.3.6F there are two general categories of assumptions in the exposure assessment -- behavior patterns (e.g., how much an individual eats, from where the food originates, etc.) and air modeling [e.g., whether the receptors (or foodstuffs, etc.) are in the 'hot spot' or are located in an area of lower air pollution levels].

Table 4.3.6F: Variability of Arsenic Cancer Risk in Winona Health Risk Assessment

Receptor Population	Assumed Behavior Pattern[a]	Air Modeling Assumptions[b]	Estimated Arsenic Cancer Risk
Urban Citizen	worst case	worst case	1.2×10^{-7}
	worst case	probable case	3.2×10^{-8}
	probable case	worst case	4.8×10^{-8}
	probable case	probable case	2.8×10^{-9}
Rural Citizen	worst case	worst case	2.7×10^{-5}
	worst case	probable case	38.5×10^{-8}
	probable case	worst case	7.3×10^{-8}
	probable case	probable case	5.3×10^{-8}
Sportsfishermen	worst case	worst case	1.9×10^{-6}
	worst case	probable case	6.7×10^{-7}
	probable case	worst case	1.9×10^{-7}
	probable case	probable case	6.7×10^{-8}

_____ indicates scenarios used in text

a. worst case: 'hot spot' approach, or all pathways 100% in impact zone;

b. probable case: average isopleth approach, or not 100% in impact zone.

Each of the three receptor populations was modeled in four separate scenarios ranging from worst case to probable case. As can be seen with the urban citizen receptor population and the sportsfisherman receptor population, the risk range varied 50-fold; from 2.8×10^{-9} to 1.2×10^{-7} and 6.7×10^{-8} to 1.9×10^{-6}, respectively. Much less variation was seen in the rural population; an approximately 5-fold difference was calculated between the probable case scenario and the worst case scenario.

4.3.6.6.6 Modeling of Additional Emission Rates

In response to a request by the MPCA staff, other emission rates of arsenic from this proposed facility were also modeled (Table 4.3.6G).

Table 4.3.6G: Emission Rates vs Cancer Risk

Facility Emission Rate (ng/M^3)	Arsenic Cancer Risk: Urban Resident	Rural Resident	Sports-Fishermen
472 [a]	4.8×10^{-8}	8.5×10^{-8}	1.9×10^{-6}
98,300	1.0×10^{-5}	1.8×10^{-5}	4.0×10^{-4}
55,000	5.6×10^{-6}	1.0×10^{-5}	2.2×10^{-4}
2,480	2.5×10^{-7}	4.5×10^{-7}	1.0×10^{-5}

a = Emission rate modeled in text

= operative risk level for permit; other risk estimates vary in consequence.

As shown in Table 4.3.6G the 472 ng/M^3 emission rate used for the calculations in the text leads to cancer risk rates of 4.8×10^{-8} for urban citizens, 8.5×10^{-8} for rural citizens and 1.9×10^{-6} for sportsfishermen. It should again be stressed that these risks are associated only with the behavioral and air modeling assumptions outlined earlier.

A 1.0×10^{-5} cancer risk level was selected for each receptor population and the corresponding emission rate was then 'back-calculated'. An emission rate of 98,300 ng/M^3 corresponds to a 1.0×10^{-5} risk to urban residents, while an emission rate of 55,500 ng/M^3 relates to a 1.0×10^{-5} risk to a rural family member. An emission rate of 2480 ng/M^3 corresponds to a 1.0×10^{-5} cancer risk for sportsfishermen.

4.3.6.6.7 Conclusions

Because of the uncertainty in dose-response modeling at low exposure levels, the U.S. EPA has developed a downward extrapolation protocol based on conservative assumptions (U.S. EPA, 1987). This protocol (the linearized, multi-stage model) is conservative because most humans are viewed as the most sensitive animal species, and because biological defense systems are neglected. (e.g. the immune system). Thus, the cancer risk estimates presented in this paper should not be regarded as accurate representations of health risks. Rather, these risks should be viewed only on a comparative basis with other risks, standards, etc. by risk managers in the decision making process.

From an interpretation of the data, conclusions can be made that the greatest cancer risk due to arsenic exposure was associated with the sportsfishermen receptor group. This cancer risk rate was 1.9×10^{-6}, if this facility emitted 472 ng/m^3 Arsenic. The cancer risk for the other two receptor populations were: 8.5×10^{-8} for the self sufficient rural individual, and 4.8×10^{-8} for a citizen of Winona. It should be pointed out, however, that these risks are specific to the assumptions made in the text of this document and, therefore, considerable variability may exist on an individual basis.

With respect to the non-cancer health risks, none of the receptor groups

should be at risk. The highest total arsenic dose was also calculated for the sportsfishermen group. This estimated daily dose is only 4.4% of our recommended chronic reference dose.

4.4 WATER QUALITY IMPACTS

It is not anticipated that the proposed project, as it has been defined by Winona County and its consultants, will have significant water quality impacts. While the facility will generate various streams of wastewater, they will either be innocuous or they will not be discharged to surface water.

4.4.1 Wastewater Generated at the Facility

4.4.1.1 Sanitary wastewater will be generated at the facility in small quantities, approximately 35 gallons per hour, maximum. This sanitary wastewater is expected to be similar in pollutant loading to normal domestic sewage, and will not pose a problem to the municipal wastewater treatment system. (MPCA, 1987; Wright, 1987a)

4.4.1.2 Industrial Wastewater will be generated at the facility. This will include boiler blowdown water, tipping floor washdown water, air pollution control equipment slurry wastewater, regenerant wastewater from boiler feedwater treatment, and housekeeping wash water. (Wright,1987a, 1987b)

The facility design calls for all industrial wastewater to be routed to the bottom ash quench pit. These industrial wastewater streams are not expected to exceed 10 gallons per minute during normal operations. Industrial wastewater will not be discharged to the Winona municipal collection system. (Wright, 1987a, 1987b, 1988) Contaminants contained in the various industrial wastewater streams will be incorporated into the bottom ash and ultimately landfilled.

4.4.1.3 Non-contact cooling water will be generated at the facility. The non-contact cooling water will not contain contaminants and will be discharged to the ash quench pit to the extent possible. Excess cooling water will be discharged to the municipal wastewater collection system. No cooling water will be discharged to the Mississippi River. (Wright, 1988)

4.4.2 Impacts of Wastewater on Water Quality

No direct discharge of any wastewater will be made by the facility to surface waters. Small amounts of domestic sanitary wastewater and unquantified amounts of non-contact cooling water will be discharged to the collection system for treatment in the Winona municipal wastewater treatment facility before subsequent discharge to the Mississppi River. The contribution of the proposed facility to effluent concentrations at the Winona treatment facility will not be measureable. No thermal discharge is expected.

REFERENCES: WINONA COUNTY RRF DEIS, WATER QUALITY IMPACTS

MPCA. 1987. Scoping Environmental Assessment Worksheet (SEAW), Winona County Resource Recovery Facility, June 8, 1987

WRIGHT, JILL. 1987a. Draft Project Description Technical Work Paper: Part I., Part II., Sections B. and G. August 14, 1987. Black and Veatch, Engineers-Architects, Detroit, MI

WRIGHT, JILL. 1987b. Letter dated Nov 11, 1987 to Eric Kilberg, Office of Planning and Review, MPCA

WRIGHT, JILL. 1988. Letter dated March 16, 1988 to Eric Kilberg, Office of Planning and Review, MPCA

4.5.1 HOUSEHOLD HAZARDOUS WASTE

Over the course of the past several years, local governments have begun to focus attention on the concern of small quantities of hazardous waste generated by the residential sector. Communities in at least forty states, Canada and Europe have begun collection programs for household hazardous waste.

This section of the EIS discusses the quantities of household hazardous waste likely to be discarded in the proposed RRF, the effect of such wastes on the air and residue ash emissions and identifies possible mitigation measures that could be employed by the Proposer to minimize the impact of household hazardous wastes on the operation of the Resource Recovery Facility (RRF).

4.5.1.1 dentification and Quantification of Household Hazardous Waste in the Mixed Municipal Solid Waste Stream

A comparison survey of household hazardous waste characterization from Marin County, California and the City of New Orleans, Louisiana conducted by the United States Environmental Proection Agency (EPA) (Rathje, 1987) determined that less than one percent of the residential solid waste stream was hazardous. It was also determined that the hazardous waste composition of the two different geographical regions was similar. Based on measurements of household wastes discarded in the New Orleans and Marin County areas, it appears that approximately 0.35 to 0.40 percent of the solid waste being discarded is hazardous. The average household in this study discarded approximately 55 to 60 grams of hazardous waste per week (not including contaminated containers and articles such as used paint brushes, oily rags, etc.). In this study, the hazardous waste was grouped into the following categories:

1. Household cleaning
2. Automotive maintenance
3. Household maintenance
4. Pesticide and yard maintenance
5. Batteries and electrical
6. Prescription drugs
7. Selected cosmetics
8. Other

The MPCA received funding in 1985 for a series of pilot collection programs from the Legislative Commission on Minnesota Resources and the results of their survey and studies are reported (Ridgley, 1987). Winona County was one of the participants in the project. The average public participation rate for these counties was approximately 2%. Most of the waste collected were oil and paints with smaller amounts of solvents and pesticides. (R.W. Beck, 1987)

4.5.1.2 Effects of Household Hazardous Waste on Incinerator Air Emissions

Incinerating those household hazardous wastes that are combined with the municipal waste stream may have an impact on the air emissions and ash from the proposed RRF. The general concern is associated with the fate of such

hazardous wastes when they are incinerated. The main concerns include the effects of the incineration of substances which are themselves hazardous, such as pesticides, solvents, cleaning agents, other substances, and materials which contain hazardous substances, such as fluorescent light ballasts which may contain polychlorinated biphenyls (PCB's) or batteries which may contain mercury, nickel, or cadmium. It should be noted that air and residue ash emissions are interrelated in that hazardous waste not destroyed in the incineration process will be emitted as an air pollutant or removed by the pollution control equipment as part of the residue ash. Such is the case with trace metal and organic emissions. (R.W. Beck)

Data collected both from research laboratories as well as emission data from new waste-to-energy facilities indicate that the destruction efficiency is quite high for organic compounds present in the waste stream. The emission rates are strongly affected by combustion conditions. In general, four factors control combustion efficiency: 1) reaction temperature within the combustion zone and post-combustion zone; 2) residence time in the combustion and post-combustion zones; 3) turbulance with mixing; and 4) air to fuel ratio. Organic compounds generally decompose with exposure to high temperatures. The thermal stability (resistance to decomposition) of an organic compound is dependent upon its composition and structure. Destruction profiles for various organic compounds including dioxins are shown in a study by the California Air Resources Board, (California Air Resources Board, 1984) which indicate high destruction efficiencies above 700°C (roughly 1300°F). Based on the profiles presented and the high temperatures that will exist in the combustion zone, approximately 1800°F., it is expected that even if certain hazardous wastes are found in the household waste stream to be incinerated they will be destroyed in the incineration process to a high degree of efficiency (Environment Canada, 1985). Considering the small percentage of the whole waste stream (less than 1%) represented by such hazardous chemicals, and considering the design characteristics of the proposed facility, the incineration of small quantities of hazardous chemicals is not expected to significantly effect the emissions from the RRF. The proposed facility will be equipped with a dry scrubber/baghouse combination which has been shown to be effective in the removal of various trace organics found in the flue gas stream (Radian, 1987). (R.W. Beck, 1987)

The incineration of solid waste containing trace metals will result either in the emission of such trace metals in the flue gas in their presence in fly ash from the air pollution control equipment. As the particulate control efficiency of the equipment increases, so does the collection efficiency of trace metals. The combined dry scrubber/particulate collection devices have had a significant effect in lowering trace metal emissions from modern plants. Also, the lower temperatures usually associated with the dry scrubber may increase the collection efficiency of certain of the trace metals such as mercury. Indicated removal efficiencies for most metals are in excess of 99.9% with the exception of mercury which shows relatively lower removal efficiencies and whose removal is dependent on the operating temperature of the dry scrubber. Overall, the data presented indicate that heavy metal stack emissions in the range of a few micrograms per cubic meter can be expected when the incinerator facilities are equipped with dry scrubbers and efficient particulate collection devices. (R.W. Beck, 1987)

4.5.1.3 Effects of Household Hazardous Waste on Residue Ash

The effect of incinerating household hazardous waste on the characteristics of the residue ash has not been quantified in studies reported in the scientific literature. However, literature is available from which the

nature of such effects may be qualified. The metals of greatest concern with respect to the toxicity of leachate from residue ash are primarily lead and cadmium. Other metals, such as mercury, may be contained in incinerator ash and are highly toxic, but may not by leachable from ash. Nevertheless, such non-leaching trace metals could pose occupational health hazards during ash handling operations. Additionally, trace metals in windblown ash may enter the environment during landfilling.

The major use of lead in the United States is in the production of batteries. In 1981, 62% of the lead consumed in batteries was recovered. Overall, about 55% of the lead produced in the United States is secondary lead recovered from spent batteries and other metal scrap. Lead use in various metal products is the next most frequent use of this metal. Lead is also used as a stablizer in flexible and rigid PVC articles, primarily in wire and cable insulation and shipping. Many uses of lead are not necessarily associated with hazardous products but are more associated with other non-hazardous household and industrial wastes. (Wojichowski, 1987).

Little cadmium is recovered in the United States because the end uses are so dissipative. More than half of the cadmium is consumed in corrosion protection of metal parts. Therefore, the small machinery and appliance tools, etc. can be a source of cadmium in municipal solid waste. Rechargeable cadmium batteries account for approximatley 20% of the cadmium and constitute the fastest growing market for cadmium. It is estimated that approximately 25% of the cadmium consumed in the United States could eventually end up in the combustible portion of the municipal solid waste. (R.W. Beck, 1987)

Approximately 50% of the yearly mercury consumption in the United States, or approximately 1000 metric tons is used in disposable batteries. A small fraction (approximately 4%) of the annual mercury consumption comes from secondary mercury, i.e. recovered from old batteries, discarded instruments and industrial waste streams. In Europe, batteries have been identified as a principal source of mercury emissions from municipal incinerators and separate collection of batteries is being emphasized (Swedish Environmental Board, cited in R.W. Beck, 1987)

In addition to the trace metals discussed above, fly ash may have certain amounts of trace organics that are removed from the flue gas in the dry scrubber/baghouse combination. From testing performed in Canada (Sawell, 1986) organic contaminant leachability is small. No organic contaminant mobility was detected in batch leaching tests for any contaminant except chlorophenols. Generally, low levels (less than 2%) of the chlorophenols were solubilized from any of the ashes. The exception was the spray-dryer ash where relatively high solubility was exhibited (20%). (R.W. Beck, 1987)

4.5.1.4 Mitigation Measures

As programs for removing hazardous materials and hazardous wastes from the municipal solid waste stream are instituted, the amounts of trace metals in the incoming waste stream would be expected to decrease. In consequence, one would expect that the concentration of trace metals in air emissions and in ash would decrease as well. Whether such recycling programs would be conclusively effective in lowering trace metal leachability to a point where the ash would be below the threshold regulatory values for such trace metals is not known. While the removal of such trace metals would have an overall benefit, it cannot be asumed that household hazardous waste programs would result in the complete elimination of trace metals and trace organics from air emissions. These materials are present in the municipal solid waste stream in other forms

besides household hazardous waste. It is also unlikely that 100% compliance could ever be achieved in a voluntary or involuntary program. (R.W. Beck, 1987)

Excluding batteries from municipal waste is an obvious recycling program. In the United States the New Hampshire/Vermont Solid Waste Project is presently engaged in a battery recovery program associated with their waste-to-energy project (Wojichowski, 1987). Exclusion and recycling of batteries would probably result in a significant reduction of mercury air emissions and ash concentrations, since batteries account for such a substantial portion of total mercury usage. Similarly, cadmium emissions and ash levels would also be expected to be reduced. (R.W. Beck, 1987)

The general types of mitigation measures that could be employed to lessen the amounts of household hazardous waste in the waste stream to be incinerated at the proposed facility RRF include: 1) establishment of collection centers; 2) public information education programs; and 3) product design. Product design is clearly a strategy that is not implementable at a local level, and this strategy will not be discussed further. (R.W. Beck, 1987)

Techniques for the collection of household hazardous wastes vary in different communities. The majority of programs sponsored to date have been collection days. Discussions with operators of many of the collection day programs reported that the scheduling of infrequent collection days is not the best solution. They believe this to be the case because of the fact that when residents are cleaning their homes, in the form of a major spring type clean up or are cleaning to vacate the property, they desire to dispose of the household waste at that point in time. Thus, they are not afforded the opportunity of waiting until the next scheduled collection day. Establishment of a permanent site provides for a location for proper disposal on a regular basis. (R.W.Beck, 1987)

Asking the public to participate in a household hazardous waste collection event is essentially asking them to change their solid waste management practices. Therefore, the public has to be aware of the collection event which can be managed through normal media channels. However, in addition to awareness, the public needs to be motivated to participate, and they need to be educated on how to properly participate. For instance, they have to be informed as to what materials are accepted. A serious investment must be made in public awareness. The inducement and sustaining of citizen participation will be a process that will take years to bring about. Many of the active programs have attracted less than one percent of the population to participate. To have a significant impact larger segments of the population will need to be encouraged to participate. (R.W. Beck, 1987)

4.5.2 NON-HOUSEHOLD HAZARDOUS WASTE

Non-Household hazardous wastes are subject to a comprehensive program of regulation at the federal and state level in Minnesota. The existing regulatory milieu is summarized below and the likelihood that unregistered quantities of hazardous waste might enter the incinerator are discussed.

4.5.2.1 Regulation at the Federal and State Level

In 1976, Congress passed the Resource Conservation and Recovery Act (RCRA) which directed the EPA to develop and implement a program to protect human health and environment from improper hazardous waste management practices. The program is intended to control the management of hazardous waste from its generation to its ultimate disposal. EPA first focused on large companies, which generate the greatest portion of hazardous waste. Business producing less than 1,000 kilograms of hazardous waste in a calendar month were exempt from most of the hazardous waste management regulations published by EPA in May 1980. (R.W. Beck, 1987)

Due to an increased public attention to small quantity generators, the Hazardous and Solid Waste Amendments to RCRA were adopted in November 1984. The emphasis of this new law was to bring small quantity generators who generate between 100 and 1,000 kilograms of hazardous waste in a calendar month into the hazardous waste regulatory system. Regulations orientated toward these generators were approved on March 24, 1986 to be effective September 22, 1986. (R.W. Beck, 1987)

On March 24, 1987, additional regulatory action was implemented whereby small quantity generators who store hazardous waste for periods longer than six months, and who perform certain kinds of waste treatment or dispose of hazardous waste on their property must apply for a RCRA permit and comply with additional rules. (R.W. Beck, 1987)

Chapter 7045 of Minnesota Rules (Minn. Rules Parts 7045.0020 through 7045.1260) were promulgated under the authority of Minn. Statutes, Section 116.07, Subds. 4 and 4b. These rules apply to all generators of any amount of hazardous waste. A generator of hazardous waste is defined in Minn. Rule Part 7045.0020, Subp. 31, as "...any person, by site, whose act or process produces hazardous waste identified or listed in parts 7045.0100 to 7045.0141, or whose first act causes a hazardous waste to become subject to regulation. Household quantities of hazardous waste are exempted. Minnesota was authorized in February of 1985 by the Environmental Protection Agency (EPA) to operate the Federal hazardous waste program in lieu of the EPA. One prerequisite of this authorization was the requirement that the State Rule be at least as stringent and the Federal Regulation. (Meyer, 1988)

Generators of hazardous waste in Winona County are required to disclose to the Minnesota Pollution Control Agency the quantity and type of hazardous waste they generate and to prepare hazardous waste management plans for those wastes. Subsequent to initial registration, annual reporting to the MPCA is required. Early in the inception of Minnesota's hazardous waste program, a list of potential generators was developed by the MPCA, using telephone directories, manufacturing directories, and other resources. These generators were contacted by the MPCA and advised of their responsibility to register, and provided with materials for registration. (Meyer, 1988)

Altogether, some 6500 generators of hazardous waste have been registered,

statewide, in Minnesota. It is estimated that an additional 10,000 to 15,000 small generators exist who are not registered, and that these unregistered generators each produce an estimated 40 gallons per year of hazardous waste. When compared to the volumes of disclosed hazardous waste for which the Agency has records of disposal, shipment, or recycling, it is estimated that the 6500 registered generators are the source of over 98% of all hazardous waste generated in Minnesota. In addition, the MPCA continues to conduct an industry by industry education and registration program to increase disclosures. Most recently, the graphic arts industry was targeted. (Meyer, 1988)

4.5.2.2 Likelihood of Unregistered Quantities of Hazardous Waste Entering the Facility

It is probable that a small quantity of non-household (registerable) hazardous waste may be delivered to the RRF or otherwise disposed of illegally. Some of these hazardous wastes will be sewered, and some will be disposed of illegally on land, but it appears likely that some material might arrive at the proposed facility. During a waste characterization field test conducted in 1986 at the Red Wing, Minnesota waste-to-energy facility, a delivery of approximately 80 kg of a potentially hazardous waste was recorded. The substance was described as 81 kg of auto paint and was hauled by the generator to the Facility. The incidence was reported as quite unusual by the plant manager (MPCA, 1980).

During the summer of 1987, a St. Croix County, Wisconsin resident reportedly contacted his waste hauler to determine the proper method of disposal for approximately 5 pounds of strychynine. The resident call was prompted by a concern to dispose of the waste properly. The resident was advised of proper disposal facilities and an upcoming clean sweep program where the resident could dispose of the substance (Anderson, L. 1987).

These reports indicate that there are quantities of hazardous material within the general waste stream which have the potential for being delivered to the incinerator facility if individuals are not aware of correct disposal options. MPCA is presently conducting workshops throughout the state to inform generators on regulatory and proper disposal procedures. Such a meeting was held October 27, 1987 in the City of Winona. Continuous information and education efforts in the form of mass mailings and personal response are utilized by the MPCA, the County, and the cities in the facility service area to increase awareness to the problem. These efforts, in combination with an already effective hazardous waste disclosure program, will assure that only a minimal quantity of registerable hazardous waste will be delivered to the facility. Inspection procedures at the facility will identify obvious offending deliveries. Nevertheless, very small amounts of registerable hazardous waste will probably be delivered undetected to the facility and be incinerated. (R.W. Beck, 1987)

4.5.3 Residual Wastes from the Facility

4.5.3.1 Review of Regulatory Environment

Laws, rules, and policies pertaining to the disposal of various residue from municipal solid waste (MSW) incinerators are rapidly evolving. The United States Congress has before it several bills. The U.S. Environmental Protection Agency (EPA) is in the process of promulgating rules. Specific legislation regarding incinerator ash was enacted in Minnesota in 1988. The following discussion is intended as a review of existing and possible future legislation and regulations which may impact the proposed incinerator. It must be recognized that the regulatory milieu affecting incinerator ash is dynamic, and that inferences pertaining to future laws and rules are necessarily speculative.

Wastes are classified as hazardous pursuant to Title 40, Part 261 of the Code of Federal Regulations (40 CFR 261). 40 CFR 261.20 lists the general characteristics (ignitability, corrosivity, reactivity, and EP [extraction procedure]) toxicity, the possession of which results in a waste being classified as hazardous. These characteristics are demonstrated by test procedures defined in 40 CFR 261. 40 CFR 261, Subpart D lists hazardous wastes from specific and non-specific sources, and also lists hazardous wastes by specific chemical name. Incinerator ash is not specifically listed as a hazardous waste in the regulation. 40 CFR 261.4 also includes a listing of exclusions from regulation under 40 CFR parts 262 through 265, 268, 270, and 271. This exclusion includes a passage which states that resource recovery facilities which do not accept hazardous waste "...shall not be deemed to be treating, storing, disposing of, or otherwise managing hazardous waste..." The passage is unclear, however, as to whether residuals produced at such facilities are also excluded. Some environmental groups, most notably the Environmental Defense Fund, have maintained that incinerator ash should be tested in accordance with the extraction procedure (EP) toxicity method described in 40 CFR 261.24, and that ash which fails the EP toxicity test should be deposited in a hazardous waste landfill. Incinerator operators, some states, and the EPA have argued that incinerator ash is either exempt from regulation pursuant to Section 3001 of the Resource Conservation and Recovery Act (RCRA) or that the EP Toxicity test is not a reliable test to use in the case of ash disposed of in a monofill. Thus the major controversy under current rules is whether the testing and disposal provisions of 40 CFR apply to incinerator ash.

The EPA is in the process of writing guidelines concerning how to sample and test incinerator ash. These guidelines are expected to state that the ash residues from incinerators which process only household waste are exempt from regulations as a hazardous waste. Beyond issuing this guidance, EPA plans to continue to test samples across the country and to research alternative technologies to decrease ash hazards. In addition to the plans to propose new guidelines concerning testing, the EPA is currently studying the ash disposal issue and is expected to present the results of their studies regarding the disposal guidelines for incinerator ash. The EPA has published data it has collected relative to the leachate concentrations of trace metals from existing, operating ash landfills. (EPA, 1987) The results of the data show that the leachate concentrations are lower than what the EP Toxicity test predicts. The EPA has recently proposed the TCLP (toxicity characteristic leaching procedure) test as a possible replacement for the EP toxicity procedure. The EPA is expected to turn its attention to the management disposal practices followed by landfills where incinerator ash is disposed. It

is expected that the EPA may still recommend that the EP Toxicity tests or the TCLP test be performed, but the results of such tests may not be absolute criteria for determining whether incinerator ash is a hazardous waste. The tests, the EPA is expected to announce, should be used only to compare the leachability of trace metals from such ashes to existing data that has been collected using the EP Toxicity test procedures. Along with the tests, consideration ought to be given to the particular disposal practices utilized. (R.W. Beck, 1987; Couppis, 1988)

Minnesota Rules Chapter 7045 is generally analogous to the Federal Hazardous Waste rules (40 CFR 261) described above, with the notable exception that Minn. Rules part 7045.0131, Subpart 6 provides additionally that oral, dermal, or inhalation toxicity is also a determinant of whether a waste is classified as hazardous. Toxicity thresholds and basic methodology for toxicity testing are described in Minn. Rules, Part 7045.0131, Subpart 6. In the past, agency staff has interpreted Minn. Rules, Part 7045.0131, Subpart 6, as requiring that oral toxicity of incinerator ash be evaluated. It now appears that recently enacted statute will supercede the requirement for oral toxicity testing, as MSW incinerator ash will henceforth be categorized as special waste.

The U.S. Congress is active in reviewing proposed legislation with regard to the disposal of incinerator ash. Several bills are being considered in the house and the senate, including S. 1894 (Mitchell) H. R. 4357 (Luken), and H.R. 2517 (Florio). The specific provisions of the bills vary, but there are certain common provisions. These common provisions include mandates for the development of new testing procedures for incinerator ash by the EPA. The bills would require that ash be deposited in monofills with various lining requirements, although co-disposal would be allowed in some versions, provided more stringent lining requirements were applied. (Couppis, 1988)

It should be pointed out that several states are already taking steps similar to what is expected EPA would announce in the near future as a result of their ongoing inhouse studies relative to the disposal of incinerator ash. States such as New York have specifically announced that residue ash from municipal incinerators processing household wastes and non-hazardous commercial and industrial wastes are specifically exempt under RCRA Subtitle C regulations according to the interpretation of the New York Department of Environmental Conservation. The State of Oregon, for instance, with respect to the disposal of the residue ash from the Marion County facility, has indicated that the EP Toxicity test performed on the ash from the facility did not clearly demonstrate that the ash is either hazardous or non-hazardous. Therefore, it chose to acknowledge the fact that great variability exists in the EP Toxicity test procedure and to place its attention on how ash is disposed of and what the impacts from such disposal actually are. In essence, the attention is placed on management practices at the landfill in combination with the EP Toxicity test procedures. The State of Washington has designated incinerator ash as a special waste and has given a five year time frame to research and study the characteristics of such ash before it can be classified as either hazardous or non-hazardous. (R.W. Beck, 1987)

In Minnesota Statutes, Section 115A.97, enacted in 1988, incinerator ash is designated a special waste during an interim period which will end on June 30, 1990, or at such time that the MPCA or EPA promulgates rules pertaining to the testing and management of incinerator ash. The statue requires the MPCA to develop an interim program for testing, monitoring, and storage of incinerator ash. Separate testing of combined and component ash streams is required unless the MPCA determines that physical constraints at the facility preclude the

aquisition of separate samples. Incinerator ash stored pursuant to interim rules will be subject to rules promulgated pursuant to the statute. (Minn. Statutes, Section 115A.97)

It is likely that the rules promulgated by the MPCA will anticipate, at least in part, those promulgated by the EPA. However, it also appears unlikely that the MPCA will approve co-disposal of incinerator ash in mixed municipal solid waste landfills. Historiclly, the MPCA has discouraged disposal of ash in the acidic environment of solid waste, since it has long been known that lower pH enhances the mobility of various metal ions (most specifically, lead) present in the ash. (Andrews, 1988a)

Overall, the present regulatory environment in terms of the methods used to dispose and manage incinerator ash can be classified as being in a state of flux. EPA, as well as many states, are currently evaluating and studying the dilemma of how to classify ash and how to dispose it. In addition, Congressional legislation is possible that would direct the EPA on how to proceed in the future with respect to this issue. In any event, it is expected that in the future, the above-mentioned legislative and regulatory activities will result in the issuance of guidelines pertaining to the testing, management, and disposal of incinerator ash. As a final note, the dynamic nature of the legislative and regulatory milieu pertaining to incinerator ash must be recognized. The information presented in this discussion may well be rendered obsolete as a result of future changes in direction by Congress and the States. (R.W. Beck, 1987; Couppis, 1988)

4.5.3.2 Test Procedures

The Resource Conservation and Recovery Act (RCRA), Section 3001, required the EPA to identify wastes which, improperly managed, pose a hazard to human health and the environment. The methods presently used to identify such wastes as "hazardous" are: (1) certain substances are listed as hazardous; and (2) through the identification of characteristics of waste which would cause harm to human health and the environment if the waste is improperly managed. Under the second approach, testing is required according to EPA protocol. (R.W. Beck, 1987)

The Extraction Procedure (EP) Toxicity test is intended to simulate the leaching conditions which are expected to occur in a sanitary landfill. The analytical procedures are described in detail in Method 1310, TEST METHODS FOR EVALUATING SOLID WASTE PHYSICAL/CHEMICAL METHOD, LABORATORY MANUAL, SW-846 (EPA, 1986a). The EP Toxicity test employs acetic acid as the leaching medium because it represents the common products of decomposition in municipal sanitary landfills. The EP Toxicity test is intended to produce information on the expected amount of groundwater contamination which could occur if the waste is improperly managed. The leachate is tested for eight metals (arsenic, barium, cadmium, chromium, lead, mercury, selenium, and silver), four insecticides (endrin, lindane, methoxyclor, and toxaphene), and two herbicides (2,4-D and 2,4,5-TP). The National Interim Primary Drinking Water Standards are used as the benchmark for determining the toxicity level of these chemicals. A generic dilution/attenuation factor of 100 units was adopted for each of these substances, to establish a regulatory threshold which is intended to account for the dilution and attenuation occurring during migration of the leachate to the groundwater. (R.W. Beck, 1987)

The area of greatest controversy associated with the EP Toxicity test with regard to its applicability to municipal incinerator ash is the question of whether it realistically represents the amount of leaching which occurs in

an ash monofill, where the ash is disposed of in such a manner as to avoid contact with acetic media. Data collected at ash monofills and presented in Section 4.5.3.5.3 suggest that the EP Toxicity test procedure overpredicts actual leachate concentrations by significant amounts. In addition, the use of drinking water standards as regulatory thresholds limited the test to establishing toxicity levels only for certain specific chemicals. Organic constituents, including volatile organics, were not addressed. More recently, advances in mathematical modeling of transport mechanisms have made species-specific dilution/attenuation factors a possibility for establishing the regulatory thresholds. (R.W. Beck, 1987)

In the course of performing the EP Toxicity test, operational difficulties are encountered which cause problems in the laboratory, particularly with the precision and reproducibility of test results, especially in cases where heterogeneous wastes such as ash are tested. (R.W. Beck, 1987)

With these shortcomings of the test procedure in mind, the EPA published Proposed Rules for amending the procedure on June 13, 1986 (EPA, 1986b). The EPA proposed a revision to the EP Toxicity test so that it would more accurately predict leaching potential; identify additional characteristics of hazardous wastes, including the ability to mobilize other toxicants (solvent-containing wastes); include additional chemicals; and introduce a new leaching test with fewer operational difficulties. (R.W. Beck, 1987)

A second generation leaching procedure, the Toxicity Characteristic Leaching Procedure (TCLP) was proposed which addresses both organic and inorganic constitutents, and which is intended to solve the above mentioned operational problems associated with the EP Toxicity test. In addition, it was proposed that the TCLP test include 38 additional compounds and utilize compound-specific dilution/attenuation factors resulting from recently developed ground water transport models. A summary comparison of the differences between the EP Toxicity test and the TCLP test is presented in Table 4.5A. The question of whether the TCLP test is applicable to wastes such as incinerator ash is still a major issue similar to the questions raised with the EP Toxicity test. (R.W. Beck, 1987)

The TCLP test as proposed is the subject of much public comment and controversy, and consequently, the promulgation of its final version has been postponed until more extensive studies on the subject are complete. The final version is now expected to be published in August, 1988. At the same time, the EPA is considering a separate test to simulate leaching conditions at an ash monofill.

4.5.3.3 Ash Handling Procedures at the Resource Recovery Facility (RRF)

The RRF is expected to produce two distinct types of ash. Bottom ash will be collected at the bottom of the incinerator and fly ash will be collected at the fabric filter. Included in the fly ash will be a certain amount of ash collected at the bottom of the dry scrubber vessel. (R.W. Beck, 1987)

Bottom ash at the RRF is expected to range from 90% to 99% of the total ash produced with the fly ash ranging from 1% to 10% percent of the total ash. The total ash produced is expected to be on the average approximately 2200 pounds per hour per combustion unit and 8190 tons per year with the RRF having an 85% capacity factor per combustion unit. The above estimates include the addition of lime and the reaction products from the air pollution control

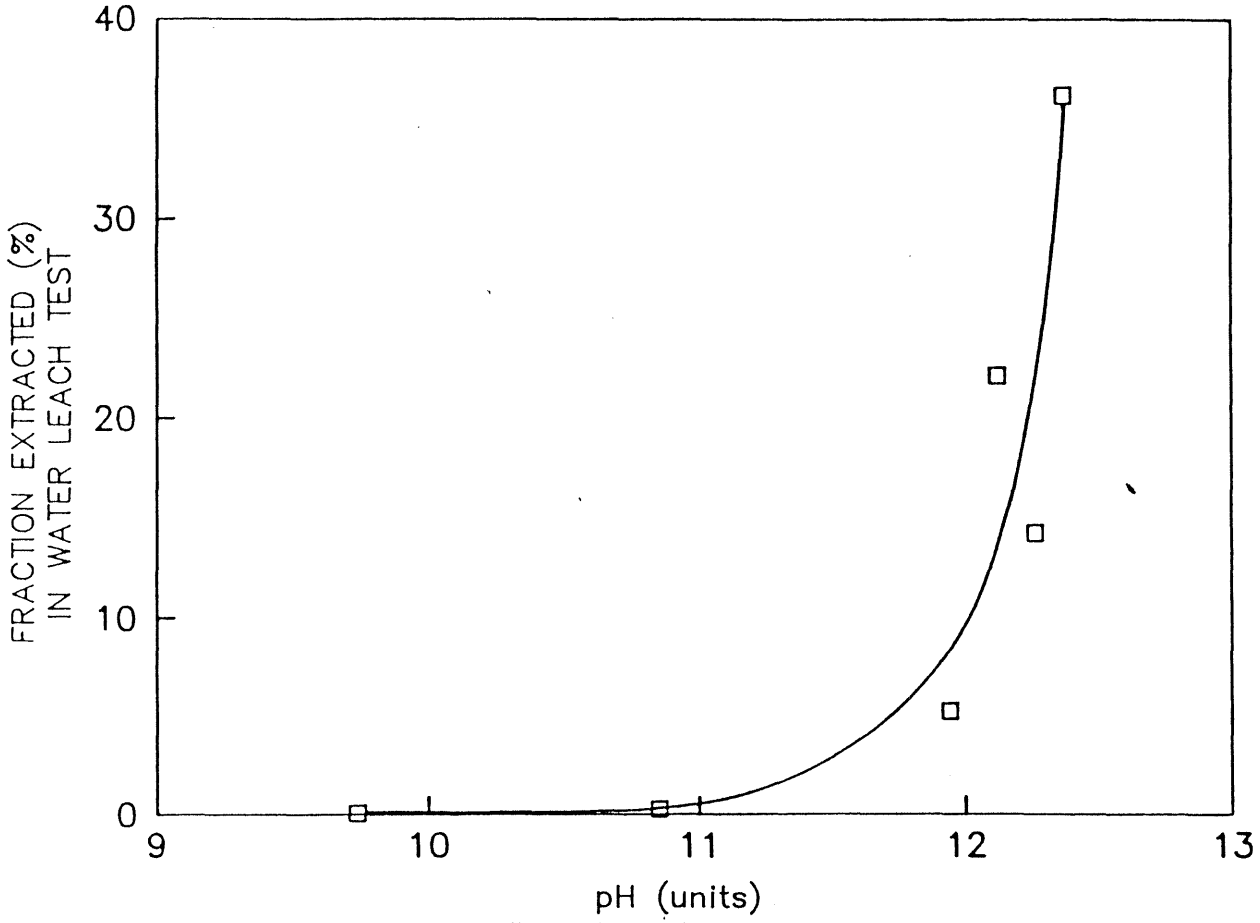

FIGURE 4-5

EFFECT OF pH ON THE SOLUBILITY
OF LEAD IN FABRIC FILTER ASHES

equipment. Therefore, total ash from the RRF is expected to average approximately 16400 tons per year at a capacity factor of 85%. A more detailed breakdown of the relative amounts of ash, reaction products and unreacted lime is given in Table 4.5B. The Proposer estimates that the total ash to be disposed including the water moisture addition will be less than 3200 pounds per hour per unit. Hence, the 3200 pounds per hour may represent a maximum amount for any given hour. The estimates of ash are based on the waste composition as shown in Table 4.5C. (R.W. Beck, 1987)

The air pollution control equipment proposed for the the facility has been described in the Project Description section this document. Briefly, the facility will be provided with an acid gas scrubber of the type referred to as a dry scrubber in the United States. A hydrated lime slurry is injected by atomization into the spray dryer where it reacts chemically with the acid gases in the flue gas. As the water in the lime spray is evaporated, the reaction products are collected in the fabric filter, where they will be periodically cleaned by a pulse jet and collected as fly ash and mixed with bottom ash from the primary combustion chamber. (R.W. Beck, 1987)

The dry scrubber proposed for the RRF will inject a slurry of hydrated lime into a reactor vessel. The spray will react with the acid gases and neutralize them while the water in the lime spray is evaporated resulting in collectable dry salts primarily of calcium chloride, calcium sulfite and calcium sulfate. The stoichiometric ratio of the hydrated lime injected into the reactor is expected to be in the range of 1-1/2 to 2. The stoichiometric ratio is defined as the relative amount of lime actually added to the reactor to the amount theoretically required to react with all of the acid gases in the flue gas on a molar basis. The flue gas is expected to enter the reactor vessel with a temperature of between 360F and 400F depending on the capacity of the system at the time. The exit temperature from the reactor is expected to be in the range of 275F to 300F. Therefore, the temperature of the flue gas is expected to decrease approximately 100F in the reactor vessel. The stoichiometric ratio actually utilized will to a large extent depend on the concentration of acid gases produced from the incoming waste and the desired degree of removal for hydrochloric acid and sulfur dioxide. The higher the removals of sulfur dioxide that are desired, the higher the stoichiometric ratio is likely to be. Sulfur dioxide is more difficult to remove from the flue gas than hydrochloric acid. It is expected that the stoichiometric ratio of 1-1/2 to 2 is a reasonable estimate as of this time. The specifications for the dry scrubber and the scrubber vendor are not known by the Proposer at the present time. (R.W. Beck, 1987; Wright, 1988)

4.5.3.4 Impact of the Dry Scrubber on Residuals

Aside from the removal of acid gases and other trace organics from the flue gas, the dry scrubber will have two primary effects with respect to the characteristics of the residue ash.

1. Since the temperature of the flue gas is decreased by approximately 100°F in the scrubber vessel, and high particulate removal efficiencies will be employed by the use of a baghouse, more trace metals are likely to be removed from the flue gas along with the fly ash in the baghouse. The lower temperatures will increase the condensation of trace metals and organics onto the fly ash for ultimate removal in the baghouse. Certain amounts of ash will also be removed from the bottom of the scrubber vessel. (Couppis, 1983; R.W. Beck, 1987)

2. The addition of hydrated lime to the reactor vessel to react with the

acid gases will result in the formation of certain reaction products, primarily the salts of hydrochloric acid and sulfur dioxide, namely calcium chloride, calcium sulfite, and calcium sulfate. Also, unreacted hydrated lime will remain in the fly ash. (R.W. Beck, 1987)

The significance of the increased amount of trace metals and trace organics removed as a result of the scrubber will increase the concentration of these contaminants in the fly ash. To the extent that these these contaminants may be in a chemical form that will be readily leached from the residue ash, the ash may be more likely to exhibit hazardous characteristics. At the present time, the data required to ascertain the significance of the increase of trace metals in the fly ash are not plentiful. (Couppis, 1988; R.W. Beck, 1987)

The significance of the addition of hydrated lime to the flue gas and the ultimate removal of such unreacted lime in the baghouse will have the tendency to increase the pH of the fly ash that is removed in the baghouse and ultimately, the combined ash if bottom ash and fly ash are mixed, as is proposed. The amount of lime added and the amount that remains unreacted could theoretically either have a positive effect or a negative effect in terms of allowing the ash to pass the EP Toxicity test. Should an excessive amount of lime be added so that the pH and buffer capacity of the ash is increased beyond approximately 12 pH units, certain metals, especially lead, are more likely to leach out from the ash at the higher pH. (Couppis, 1988; R.W. Beck, 1987) Figure 4.5 shows the leachability of lead as a function of pH (Cote, et. al., 1985).

These aspects of the dry scrubber will be examined when the leachability data is reviewed in Section 4.5.3.5.3. The amount of data that is available from plants with a dry scrubber in the United States is limited due to the fact that at the present time there are only a few plants that are operating where a dry scrubber is utilized.

4.5.3.5 Characterization of Waste

4.5.3.5.1 General

There are relatively few data available to describe residue ash from resource recovery facilities and associated leachate resulting from land application of such residue ash. Such data are generally regarded by manufacturers and operators as proprietory information. In addition, the ash quality can be expected to be dependent on such factors as the waste composition, moisture content, incinerator technology, combustion efficiency and scrubber and sorbent chemistry. Furthermore, ash collected from different points in the process can be expected to have different physical and chemical characteristics. Finally, the methods used to determine the quantity of materials that would leach out from the residue ash into water or other leaching media are not well standardized and can affect the resulting data as indicated in Section 4.5.3.2. In spite of the uncertainties described above, residue ash is known to include several components which affect its potential impact on the environment. Those components include water soluble salts, metals, organics, acidic compounds and buffers derived from combustion byproducts and scrubber reaction products and sorbent material. (R.W. Beck, 1987)

In selecting the data sources to be used to describe the physical and chemical characteristics of the residue ash as well as the leachability potential resulting from such ash, the following criteria were utilized: 1)

data from facilities with regional similarities; 2) data from facilities that are equipped with similar pollution control equipment, i.e. a dry scrubber; and 3) data from facilities that have conducted organized ash testing programs. (R.W. Beck, 1987)

As such, we have selected data from the following facilities: 1) Marion County, Oregon Facility; 2) Barron County, Wisconsin Facility; 3) Waukeshau, Wisconsin Facility; 4) Claremont, New Hampshire Facility; 5) RESCO, Massachusetts Facility; 6) Quebec City, Canada Facility; 7) New York State Facilities; and 8) City of Commerce, California Facility. (R.W. Beck, 1987).

Data from Barron County and Waukesha, Wisconsin were selected primarily because of regional similarities with the Proposer's RRF. Data from New York State and the RESCO, Massachusetts facilities were selected because of their organized data collection programs and data from Marion County, Claremont, Quebec City and City of Commerce facilities were selected because they are equipped with some type of dry scrubber. The facilities with a dry scrubber mentioned above constitute most of the United States facilities that employ dry scrubbing and have ash data available. The data collected along with conclusions reached will be presented below. (R.W. Beck, 1987; Couppis, 1988)

4.5.3.5.2 Characteristics of the Ash

Tables 4.5D to 4.5G present data collected in Europe and in Canada with respect to the characteristics of the bottom ash and fly ash, including the dry scrubber products. (Environment Canada, 1987; Flakt, 1986; Hjelmar; 1987, Brunner and Monch, 1986; Cote, 1984). The data presented indicate that there is variability in the ash composition. However, realizing that ash is a fairly heterogeneous substance, the results compare reasonably well with one another. Cadmium, mercury, zinc, molybdenum, arsenic and lead are generally present in significantly higher concentrations in fly ash than in the bottom ash. It should be noted that the concentration of copper is significantly higher in the bottom ash than it is in the fly ash. Furthermore, from an environmental point of view it is not only the total content of potentially hazardous components in the incinerator residues that is important, but rather the leachable amounts of these components that are present. (R.W. Beck, 1987)

Some of the physical properties of incinerator ashes that are important in relation to disposal are bulk density, porosity, permeability and particle size. Bulk densities of 1.1 to 1.8 tons per cubic meter for bottom ashes and 0.5 to 1.6 tons per cubic meter for fly ashes have been reported (Hjelmar, 1987). Reported porosities of bottom ash containing fly ash are approximately 30%, and effective porosities in landfills containing bottom ash and fly ash are 15% to 20%. Water permeability has been found to vary between $2x10^{-7}$ and 10^{-5} meters per second for bottom ash and $4x10^{-8}$ and $3x10^{-7}$ meters per second for fly ash. (Hjelmar, 1987). Therefore, the permeability of the ash itself will not prevent infiltrated water from flowing down through a landfill. (R.W. Beck, 1987)

From a physical standpoint, bottom ash may be characterized as large particles with relatively fewer available potential pollutants and high pH in the range of 10 pH units. On the other hand, fly ash generally contains smaller particles with more available potential pollutants including complex organic compounds. Fly ash also usually has a pH which is neutral or slightly acidic in systems without scrubbers and basic (pH of 11 to 12 units) in systems with dry scrubbers (Bridle et. al., 1987). Thus, fly ash has the greatest potential to impact the environment. (R.W. Beck, 1987)

4.5.3.5.3 Leachability Data

Data from existing operating facilities were selected to present the state of knowledge with respect to the leachability of various pollutants from residue ash from waste-to-energy plants that exhibit certain similarities to the proposed RRF. The similarities that were judged to be most significant are the regional characteristics of the facility and the type of pollution control equipment employed, namely the employment of the dry scrubber which will be installed on the proposed RRF. In addition, data that have been collected with a formalized sampling program have been given more weight in contrast to sampling programs that were sporadic in nature and were done over short periods of time. The data presented herein are the most appropriate data to indicate the possibilities of impact resulting from the proposed RRF relative to ash disposal. In this section, data is presented from the facilities selected and observations made with respect to the leaching characteristics of the residue ash as they may affect the disposal or utilization of the residue ash from the Proposer's RRF. (R.W. Beck, 1987)

Quebec City Pilot Plant Data

A pilot plant program (Sawell, 1986) was undertaken at the Quebec City, Canada Municipal Incinerator to evaluate the effect of different types of dry scrubbers on the air emissions of the facility and on the characteristics of the ash. Table 6.5D et. al., shows a summary of the data collected characterizing the composition and leachability of the fly ash. Tests utilized in Canada do not correspond with the EP Toxicity test used in the United States, therefore, direct comparisons of the Canadian data with data from american facilities cannot be made. However, certain general trends can be identified. The data indicate that most of the trace metals are readily leached from the fly ash. It does not appear that the addition of hydrated lime inhibits the leaching of metals to any degree from the fly ash. Data are not presented to identify the equilibrium pH during the leaching tests, therefore, the buffering capacity of the ash/lime mixture is known. The data shows that when the ash is leached with water, considerably fewer metals leach from the ash compared to leaching with either dilute or strong acids. The neutral leaching media is representative of what might be expected for a residue ash monofill and the dilute acid condition would be representative of what might be expected in an ordinary municipal landfill. (R.W. Beck, 1987)

Marion County, Oregon Resource Recovery Facility (RRF)

Ogden Projects, Inc. conducted research to provide data on the properties of residue ash through a sampling and analysis program to comply with the State of Oregon's "Hazardous Waste Determination" rules (Odgen, 1986a; Ogden, 1986b). Ogden Projects, Inc. reports that the sampling program and statistical analysis of the analytical results were performed in accordance with the guidelines in U.S. EPA SW-846, (EPA, 1986a). The sampling was performed on the combined residue ash from the ash discharges which includes both bottom ash and fly ash. No separate fly and bottom ash testing was performed. The residue ash samples were subjected to the following tests: 1) toxicity characteristic leachate procedure (TCLP) under review by the EPA; 2) EP Toxicity tests; 3) de-ionized water extraction; 4) analysis of total metals by acid digestion; and 5) analysis of selected cations and anions and pH of the various extraction fluids after extraction of the residue samples. (R.W. Beck, 1987)

Table 6.5H presents the results for lead of the EP Toxicity test conducted on the 18 samples of ash collected. Table 6.5I shows the results of the TCLP test, the de-ionized water leach test, the total metals by acid

digestion and the EP Toxicity threshold as set by the EPA. According to Ogden Projects, Inc. the results of the EP Toxicity tests on the 18 representative samples of residue ash randomly collected indicate that the residue ash from the Marion County Resource Recovery Facility is not a hazardous waste. Specifically, the concentrations of all the metals tested (8 metals are listed under the characteristic of EP Toxicity in 40 CFR, Part 261.24) were below the regulatory threshold, in every sample except for lead. Since the cadmium levels at other facilities have been shown to at times exceed the regulatory threshold Table 6.5J shows the results of the EP Toxicity Test for cadmium for the 18 residue ash samples collected. The concentration of the lead in the sample leachate indicate that the mean value of all the samples was 3.51 mg/liter as compared to the 5 mg/liter regulatory limit. Two samples collected were above the 5 mg/liter limit, one sample was 5.16 and the other sample was 21 mg/liter. In addition, the transformed statistical evaluation also showed the upper bound limit of the confidence interval for lead was also below the regulatory threshold. Therefore, according to Ogden Projects, Inc. the residue ash should not be considered as a hazardous waste. Taking the data collected from the 18 samples into account, the lead concentrations in the sample leachate were below the regulatory threshold the predominant number of times when the ash was subjected to the EP Toxicity test procedure. (R.W. Beck, 1987)

The results of the TCLP test also indicate that all the metals are always below the regulatory threshold except for lead. Of the 18 samples analyzed, only one sample was above the threshold of 5 mg/liter. That sample was at 6.1 mg/liter. The average concentration of lead resulting from the TCLP test was 0.455 mg/liter. The results for the EP Toxicity test indicate that higher concentrations of metals are obtained in the EP Test than from the TCLP Test. Other data (Francis and Maskarinec, 1987) comparing the two test procedures indicate that the results of the EP Toxicity test and the TCLP test using leaching medium No.2 are similar. In addition, the TCLP leaching medium No. 1 appears to better present the leaching of aklaline waste by municipal waste leachate. (R.W. Beck, 1987)

The results of the de-ionized water leach tests indicate that all the metals were below the regulatory threshold limit. Therefore, when a neutral leaching medium such as water is used, the metals are less likely to leach out from the residue ash. The buffer capacity of the residue ash collected at the Marion County Facility was not explicitly determined, however, a review of the EP Toxicity tests indicate that the endpoint pH of the leachate was 5.2 for most samples. This indicates that the ash did not have enough buffer capacity to result in a final pH near 6 or higher which would have the tendency to inhibit metals leaching during the EP Toxicity and increase the likelihood of passing the EP Toxicity test. (R.W. Beck, 1987)

The test results presented by Ogden Projects, Inc. for the Marion County Facility when taken as a whole indicate that the leachability of trace metals including lead and cadmium when subjected to leaching media that are either neutral or slightly acidic would result in leachate that would likely meet the EPA Toxicity regulatory thresholds. This observation is important in view of the fact that when residue ash is landfilled in a monofill, as is the plan for the RRF, the leaching medium would be rainwater that had infiltrated the monofill which would either be neutral or slightly acidic. The de-ionized water leach tests can be considered to represent the leaching that is likely to occur from the ash monofill. Of course, it should be noted that the de-ionized water leaching tests are not viewed by the EPA as being the tests which should be conducted to determine the hazardous characteristics of the ash, even though the de-ionized water tests results may be more representative of what will

actually occur at the residue ash monofill than the results of the EP Toxicity test. (R.W. Beck, 1987)

The results from the Marion County Facility are important in that the facility employs a dry scrubber for the removal of acid gases and, therefore, the data from that facility represents one of the first data sets from a facility with a dry scrubber. Detailed data on the amounts of hydrated lime used in the scrubber were not available. However, it is estimated that the percentage of unreacted hydrated lime and reaction products is less than 5% of the total combined residue ash from the facility. In view of the fact that the end point pH of the EP Toxicity test was in the 5.2 range, the addition of hydrated lime did not appear to buffer the mixed ash to the degree required to result in a final pH during the test of more than 5.2 which would have the likelihood of inhibiting the ash leaching characteristics. In addition increase in trace metals in combined ash which would be presumed as a result of the addition of fly ash does not appear to have resulted in elevated levels of lead and other metals as measured in the EP Toxicity test when compared to data from other facilities. In fact, the data from Marion County appear to be somewhat lower in leached lead than at other facilities. The reported data from the Marion County Facility are not conclusive to allow determination as to whether the dry scrubber (and particularly the addition of hydrated lime) has played a part in terms of decreasing the leachability of metals from the ash. Also, the pH levels of the residue ash were not elevated to those levels that would make lead more leachable. In fact, the de-ionized water test indicates that the amounts of lead that ultimately leached from the ash were below the regulatory threshold as set by the EPA. (R.W. Beck, 1987; Couppis, 1988)

Barron County Facility

The Barron County Facility is a modular facility with a capacity of approximately 90 tons per day that has been performing ash leaching tests for approximately the last six to eight months. Therefore, it is a facility that has a long history relative to other facilities in conducting such tests. Conversations with facility personnel (Anderson, G., 1987) reported that initially when the EP Toxicity tests were performed on grab samples, fly ash predominantly exceeded the regulatory thresholds for lead, and cadmium. The bottom ash also exceeded the levels for lead although not as frequently as was the case with the fly ash. Tests conducted with the combined fly ash and bottom ash also indicated that the combined ash exceeded regulatory levels for lead and cadmium on a frequent basis. As a result, the Barron County Facility decided to dispose of their fly ash as a hazardous waste and therefore, is collecting the fly ash separate from the bottom ash. Subsequent testing performed during the months of June, July, August and September on the bottom ash indicates that based on the results the EP Toxicity test, the lead levels obtained are more consistently within the same range and predominantly are below the regulatory threshold of 5 mg/liter. Of all the samples taken, approximately 1/4th of the samples exceed the limit for lead. During the last four months Barron County has changed their sampling protocol where they are taking more frequent samples and developing composites on a daily or a three day basis. This sampling protocol appears to have evened out the heterogeneous nature of the grab samples to a point where the results of the EP Toxicity test are more predictable. It is the opinion of the facility's staff that the greater the number of samples taken to be composited into a sample that will be analyzed the more accurate the results of the EP Toxicity test are likely to be in terms of reflecting the actual leaching characteristics. Based on the data presented by Barron County the average values on a monthly basis are below the regulatory threshold of EPA for lead which is 5 mg/liter. Table 6.5K shows the results from the Barron County Facility testing. (R.W. Beck, 1987)

RESCO, Massachusetts Facility

The RESCO Facility in Saugus, Massachusetts is a large waste-to-energy plant that has conducted a three year study of its residue ash to: (1) assess the long term effects of residue disposal on soil, groundwater and native biota; (2) determine the chemical characteristics of leachates from both fly ash and combined residue alone and over native soils; and (3) determine the relative contribution of fly ash and bottom ash to the overall chemistry of the residue. Tables 6.5L and 6.5M show some of the results from this study that characterize the combined ash and the bottom ash, respectively. Data collected on the fly ash alone indicate that fly ash predominantly has exceeded the EP Toxicity threshold levels for lead and cadmium. The general conclusions of this study are as follows (Mika and Feder 1985; R.W. Beck, 1987)

Fly ash did not pass the EP Toxicity test for either lead or cadmium. The buffering capacity of fly ash appears to be greater than that of residue in that generally more acid was added to fly ash samples to maintain the pH necessary during the EP Toxicity test. Leachate produced from de-ionized water poured over fly ash is initially neutral in the pH range of 6 to 7 units, extremely high in cadmium concentration (approximately 50 to 100 ppm) and relatively low in lead concentration approximately (1 to 10 ppm). The lead is mostly in the form of white precipitate. Successive leachates have higher pH values (greater than 10) and lower in cadmium. Lead continues to leach but drops off sharply in concentration following a peak of approximately pH 11 units. Bottom ash passes the EP Toxicity test at times and at other times it fails the test. Approximately half the samples fail the EP Toxicity test. The residue which is a combined fly ash and bottom ash mixture, is also buffered and produces leachates in excess of pH 7 units when leached with ambient acid rainfall of an average pH of 4 units. Residue tested in the laboratory following the standard protocol for the EP Toxicity test usually failed to pass for lead and cadmium. By contrast when regular samples which failed the EP Toxicity test in the University of Massachusetts laboratory were partitioned and given to certified commercial laboratories, they usually passed for both metals. Replicate samples tested at two other state laboratories did not pass for lead or cadmium. The discrepancies seem to be due to the interpretation of the EP Toxicity test methodology. An added acidification step prior to analysis carried out in the commercial laboratories appears to tie up the lead, thus reducing the available concentration. Cadmium is not affected. The difference in methodology leaves in doubt how much lead is actually available for leaching. Overall, the results from this study indicate that the combined residue fails the EP Toxicity test procedure the predominate number of times. This is in contrast to most of the other data presented in this TWP which shows that although the combined residue fails the EP Toxicity test procedure at times, the residue passes the test the predominate number of times. (R.W. Beck, 1987; Couppis, 1988)

Recommendations made in the University of Massachusetts report are as follows: (1) bottom ash can be disposed of as a non-hazardous solid waste without the necessity for either technology or procedures to reduce soluble salts and/or heavy metals; 2) fly ash should be handled, transported and disposed of as a hazardous waste; 3) increased effort should be made to use or adopt currently available technologies to recover lead and cadmium from fly ash. Once removed, fly ash may be mixed with bottom ash and disposed along with the bottom ash. (R.W. Beck, 1987)

Waukesha, Wisconsin Facility

The Waukesha, Wisconsin Facility has detected that when they incinerated

commercial and industrial waste at their facility, in addition to household municipal waste, an increase in concentration of lead and cadmium resulted during the EP Toxicity test. (Warzyn, 1984; Warzyn, 1985). In order to investigate methods for minimizing the lead and cadmium leaching from the ash, the Waukesha County Parks and Planning Department conducted certain tests where various types of liming agents were added to the ash at different proportions to determine whether the addition of such liming agents could inhibit the leaching of trace metals. Tables 4.5N to Table 4.5P show the results of the studies. (R.W. Beck, 1987)

The major conclusions of these studies are: 1) over the eight day testing period, lead and cadmium concentrations in incinerator ash were elevated and extremely variable when commercial and industrial wastes were accepted in addition to typical domestic solid wastes; 2) crushed dolomitic limestone does not react rapidly enough to reduce potential metals mobility and, for this reason is not acceptable as an agent for pre-landfill treatment; 3) powdered calcitic limestone at the 10% treatment level and cracking lime (mixture of lime, hydrated lime and limestone) at the 12% treatment level effectively reduced lead and cadmium concentrations to below the EPA threshold levels; and 4) the commercial and light industrial waste which appear to have contributed to elevated lead and cadmium concentrations in the ash may include rubber, plastics and colored paper. (R.W. Beck, 1987)

The significance of the testing performed at the Waukesha Facility, which was testing on the combined fly ash and bottom ash resulting from the facility, indicate that liming agents could be used to treat the ash before disposal and to reduce the leaching of lead and cadmium from such residue ash. The addition of the liming agents increases the pH of the residue ash such that the equilibrium or final pH during the EP Toxicity test is above approximately 5.5 which would inhibit the leaching of lead and cadmium. In evaluating the addition of liming agents, care has to be taken not to add an excessive amount of such liming agents so that the equilibrium pH would be elevated to a pH of 12 units or greater. If such is the case, then the leaching of lead at the pH range of above 12 increases the same way that it increases with a low pH range below approximately 5.5. (Warzyn, 1985). Powdered lime even at the 10% level, increased the equilibrium final pH to over 12. It should be noted that the addition of calcium oxide to the residue ash may result in the formation of hydrated lime upon the contact of the lime with any moisture in the ash. This would then be somewhat equivalent to the addition of hydrated lime into a dry scrubber. Table 4.5B shows the percentage of unreacted hydrated lime in the residue ash mixture of the proposed RRF which is considerably less than the 10% level that was added at the Waukesha Facility. Data are not presented from Waukesha to indicate the effect of the additional amounts lesser than 10% of lime to the residue ash mixture. Therefore, it is not known at what level lime, which in turn becomes hydrated lime, would elevate the equilibrium pH units above 12 which would initiate increased leaching of lead from the ash. Data from the Marion County Facility indicate that the amount of hydrated lime added to the dry scrubber, which is less than 10%, does not result in excessive leached levels of lead from the residue ash. (R.W. Beck, 1987)

New York State Department of Environmental Conservation Data

The New York State Department of Environmental Conservation (New York State, 1987) undertook an ash sampling program to establish as its primary objective an ash residue sampling and analysis protocol which is capable of providing data representative of the residue from waste to energy facilities. A second objective was to quantify the leaching characteristics of heavy metals from incinerator ash so that ash management and disposal options can be

evaluated. The project included ash residue sampling at six operating resource recovery incinerator facilities in New York State. Samples were analyzed utilizing the EP Toxicity test. In addition, several replicate analyses were performed utilizing the TCLP test currently under consideration by the EPA as a replacement for the EP Toxicity test. In their report, the State indicates that although the EP Toxicity test has historically been utilized to determine if a solid waste is a hazardous waste, it is important to note that regardless of the results of the EP Toxicity test, the New York State Department of Environmental Conservation has determined that the ash residue from the incineration of household and non-hazardous industrial/commercial wastes is currently exempt from regulation under Subtitle C of RCRA and the State regulatory program. Therefore, incinerator ash cannot be considered a hazardous waste. (R.W. Beck, 1987; Couppis, 1988)

A total of 37 residue samples from six facilities were collected and analyzed. Results of these analysis are shown in Table 4.5Q. Four of the twelve bottom ash samples exceeded the EP Toxicity test limits for lead. All fly ash samples exceeded the respective limits for both lead and cadmium and eight of seventeen combined fly and bottom ash samples exceeded the limits for lead or cadmium or both lead and cadmium. It should be noted that all facilities tested which combine the fly ash with the bottom ash prior to disposal. The first set of data collected indicated that the same variability inherent in the raw waste composition is also present in the ash residue. As such, it became necessary to reassess both field and laboratory protocol and to develop additional studies to characterize the entire residue stream. Therefore, based on this determination the Oswego County Energy Recovery Facility was selected for additional testing. The sample collection procedure involved core sampling of every truck load of combined ash residue generated at the facility for seven consecutive days. A large quantity of combined ash residue was then thoroughly mixed and reduced in volume using an aggregate splitter. Approximately five kilograms of sample was sent for analyses using the EP Toxicity test. Five replicates of each EP Toxicity test were performed. The results of the test are shown on Table 4.5R and Table 4.5S for cadmium and lead, respectively. (R.W. Beck, 1987)

A statistical analysis of the cadmium data show reasonable reproducibility as measured by low standards of deviation and coefficients of variations for the five replicate analyses. The results for lead show a significantly higher degree of variability. A statistical analysis of the data yields standard deviations for each of the five replicate analyses ranging from 16% to 70% of the mean. The weekly composite, which was made by combining equal quantities from each of the daily composites, had the widest variation among replicates of any of the samples. The data collected in this project clearly demonstrated the need for replicate extractions of samples for EP Toxicity tests. On five of the seven sampling dates the results could exceed or not exceed threshold limits for lead depending on which replicate was used. The 35 lead analysis (7 days, 5 replicates per day) were plotted on normal probability paper. For the seven consecutive day period, 48% of the samples analyzed for lead using the EP toxicity test were less than the regulatory threshold of 5 mg/liter. It was the opinion of the State that in view of the wide range of results observed for each sample, no meaningful conclusions could be reached on the results of the EP Toxicity tests without performing replicate analyses. The results of the test indicate that the need to assess current residue management practices is important. The Department is currently reviewing the residue data on a case-by-case basis to determine if the existing management and disposal practices afford adequate protection to human health and the environment. Therefore, it appears that the State of New York is not only looking at the leachability of the ash but also placing emphasis on how

the disposal practices are carried out, i.e. the degree of environmental impact produced as a result of such disposal practices. (R.W. Beck, 1987)

Claremont, New Hampshire Facility

The Claremont, New Hampshire Facility is a 200 ton per day facility composed of two 100 ton per day units. The facility employs hydrated lime injection into the flue gas duct for the removal of acid gases and a baghouse for the control of particulates. The hydrated lime injection differs from the acid gas control system proposed for the RRF in that the hydrated lime is injected directly into the flue gas duct as compared to a lime slurry injected into a reactor vessel which would be the case for the proposed RRF. Generally, the amount of hydrated lime needed to obtain similar removal efficiencies with the lime injection system are greater than what is required in a spray dryer type scrubber. Hence, it is expected that the amount of unreacted lime found in the residue ash of the Claremont Facility would be greater than the amount of unreacted lime found in the residue ash of the proposed RRF. (R.W. Beck, 1987)

Various tests including the EP Toxicity test and water leach test were conducted on residue ash collected from the Claremont Facility by Signal Environmental Systems (Siegler, 1987). These tests were collected during June, July and August of 1987. Results of the tests are shown on Table 4.5T and 4.5U. It should also be noted that the Claremont Facility employs a fly ash treatment process to treat the ash prior to mixing with the bottom ash for disposal. The type of treatment employed is proprietory and therefore, information is not available on the process. From what has been publicly stated the process immobilizes the lead compounds, i.e. likely changes soluble lead oxides to lead phosphates which are insoluble in water as well as possibly changing the particulate size of fly ash, thus, decreasing the surface area of the fly ash before mixing it ash with the bottom ash. Hence, the results presented in Table 4.5T include the effect of the treatment system employed at the facility. (R.W. Beck, 1987)

The fly ash treatment system was in the start mode during the initial months of plant start-up, which included March, April and May and possibly June. Therefore, the results of testing presented in Tables 4.5T and 4.5U for May and June may not totally reflect the effect of the treatment system. The results for July and August should reflect the effect of the treatment system on the leachability of the residue ash. (R.W. Beck, 1987)

Results of the EP Toxicity test during May and June indicate that the lead leachate concentrations exceeded the threshold limit values when the final pH during the EP Toxicity test procedure was near 5 pH units. It should be noted that the final pH of several of the samples were above a pH of 5 units, usually above a pH of 6 units. In such cases, the lead concentrations in the leachate were usually below the threshold values. In some cases, the final pH was as high as 12 pH units which indicates that an excessive amount of hydrated lime was added. For tests conducted during July and August, when the fly ash treatment system was operating properly, the lead concentrations of the leachate were well below the threshold limit. (R.W. Beck, 1987)

The aforementioned results indicate that the addition of hydrated lime in the scrubbing process could affect the results of the EP Toxicity test. It should be noted again that the amount of lime added with the dry injection process utilized at the Claremont Facility is greater (approximately twice) than the amount added with the dry scrubber proposed for the RRF and that utilized at the Marion County Facility. Therefore, the dry scrubber could have

a beneficial effect if an amount of lime is added that would increase the final pH during the EP Toxicity test to approximately a pH of 6 or above. It appears that the amount of lime required to raise the final pH above 6 pH units is greater than the amount required to meet acid gas removal requirements in the dry scrubber proposed for the RRF and that utilized at the Marion County facility, but may not be greater than that required for dry injection processes similar to the Claremont Facility. It should also be noted that the dry scrubber could have a detrimental effect on the leachability of lead if excessive amounts of lime are added such that the final pH is increased to above 12 pH units which was the case in some instances at the Claremont Facility. (R.W. Beck, 1987)

Water leach tests during May and June indicate that the final pH was higher than a pH of 12 units resulting in violations of the lead threshold limit. During July and August when the fly ash treatment system was operating properly, the lead concentrations were significantly below the threshold values. An interesting observation is that for some tests where the final pH during the EP Toxicity test was higher than 6, the lead concentrations for the water leach test were higher than the concentrations during the EP Toxicity test. This raises the possibility that the actual impacts at the disposal landfill may be greater than those predicted by the EP Toxicity test. Therefore, the question regarding applicability of the EP Toxicity test procedure to testing residue ash is exemplified. The water leach tests conducted at the Marion County Facility give results unlike the results at the Claremont Facility in that the concentrations are well below the threshold limits. A contributing factor could be the fact that a lesser amount of lime is added in Marion County. Another explanation could be that there are lesser amounts of lead in the Marion County solid waste. (R.W. Beck, 1987)

City of Commerce, California Facility

The City of Commerce Facility is a 300 tpd facility equipped with a dry scrubber. The scrubber vendor is the same as the vendor that provided the scrubber for the Marion County, Oregon Facility. Table 4.5V (Selma, 1987) shows results of ash using the California Waste Extraction Procedure (WET). It has been reported (Francis, 1987) that the results of the WET are comparable to those of the EP Toxicity test. The results from the City of Commerce Facility indicate that lead is consistently higher than the regulatory limit for the samples analyzed. Cadmium is below the regulatory limit of 1 mg/liter. Laboratory data forms were not obtained, therefore, it is difficult to identify whether the higher concentrations of lead in the leachate are due to the scrubber effects or due to high concentrations of lead in the municipal waste stream. (R.W. Beck, 1987)

4.5.3.6 Disposal Options

4.5.3.6.1 General

There are various options available in terms of handling and disposing of the residue ash generated at the Proposer's RRF. Those various options have varying degrees of environmental impact as well as varying levels of cost. This subsection will address the options available to the Proposer for the handling and disposing of the residue ash from the RRF. At the present time, the proposed plan for handling the residue ash at the RRF includes the collection of bottom ash and fly ash including the reaction products from the dry scrubber in the same collection container located in the collection and storage building. The fly ash will be discharged at a controlled rate of feed onto the bottom ash conveyor. (Wright, 1988; R.W. Beck, 1987)

The handling and disposal options available to the Proposer include:

1. Combined bottom and fly ash

   a. Monofill disposal of combined ash as hazardous waste or special non-hazardous waste

   b. Utilization of combined ash in useful products

   c. Treatment to render the ash non-hazardous, if tests results so indicate, with monofill disposal.

2. Separated bottom and fly ash

   a. Fly Ash
      - disposal as a hazardous waste
      - utilization in a useful product
      - treatment to render non-hazardous with monofill disposal

   b. Bottom Ash
      - disposal in a monofill as a non-hazardous waste (or hazardous waste in a Subtitle C landfill, if test results so indicate)
      - utilization in a useful product
      - treatment to render non-hazardous, if test results so indicate, with monofill disposal

4.5.3.6.2 Combined Residue

Monofill Disposal

Most existing waste-to-energy facilities in the United States today dispose of the residue ash in combined form, i.e. the fly ash is combined with the bottom ash and disposed of at approved non-hazardous landfills. As has been shown in Section 4.5.3.5.3 the leaching characteristics, mainly of lead and cadmium, of combined residues from existing waste-to-energy facilities indicate that the combined ash passes EP Toxicity test most of the time. However, due to the variability and heterogenous nature of the ash, at certain times the combined ash exceeds the limits of EP Toxicity as set forth by the EPA. (R.W. Beck, 1987)

The environmental impact associated with disposal of the combined ash in a monofill as a non-hazardous waste will result in leachate concentrations that are significantly less than what is predicted by the EP Toxicity test. As indicated earlier, the EP Toxicity test was formulated primarily to simulate the co-disposal of material with municipal solid waste. In a monofill the possibility of acidic leachate coming in contact with the residue ash is minimal. As a result, the leaching of trace metals from such a monofill is considerably less than what is predicted by the EP Toxicity test. At the present time, the MPCA believes in a policy of monofill disposal of ash as compared to co-disposal with municipal waste. As such, co-disposal options are not explicitly discussed in this TWP. (R.W. Beck, 1987)

The amount of data available to prove this point is not plentiful. However, certain data are beginning to be collected with regard to the actual leachates that results from operating monofills. Data from Denmark (Hjelmar, 1987) collected at an existing ash landfill as well as leachate column tests data performed in the laboratory indicate leachate concentrations considerably less than what has been observed during the performance of the EP Toxicity

test. Table 4.5V shows the results of this study. Similar results are reported by other studies (Cundari and Lauria, 1987).

Disposal costs in a state-of-the-art ash monofill are estimated to be in the range of $35 to $45 per ton of ash. (Winona County used an ash disposal cost of $29.81, and a transportation cost of $.72 per ton.) Assuming a combined ash monofill disposal of 16,400 tons of ash, (Section 4.5.3.3) this would equate to $574,000 to $738,000 for annual combined ash disposal expenses. If fly ash from the facility is required to be deposited in a hazardous waste landfill outside Minnesota, the disposal fee per ton averages $118 dollars, with a $.19 per mile per ton transport cost (assuming shipment in 20-yard bulk shipments. (Christensen, 1988). Assuming ten percent of the ash is fly ash, and that 1640 tons of fly ash will be generated annually, the annual disposal costs for fly ash as hazardous waste would be $318,000. Total disposal costs of 14,760 tons of bottom ash at $35 to $45 per ton in monofill disposal and fly ash disposed of as hazardous waste outside Minnesota would range between $835,000 and $982,000. If the entire ash stream must be disposed of in a hazardous waste landfill, the approximate cost, based on a $118/ton tip fee, a 400 mile transport at $.19/mile, and a tonnage of 16,400 would be $3,182,000 per annum.

Utilization

Utilization of the combined ash in useful products has not received a great deal of attention in terms of actual applications. Most of the work that has been done in this area can be classified as experimental, although several types of possible uses are identified. The only clearly demonstrated use of residue ash is in bituminous pavement construction. Other types of uses generally require further research and development work or more extensive testing. (Vence, 1985)

The European industrial community is established as a leader in residue recycling. Incineration of refuse has been practiced in Europe since the 1870's and much of the early work in residue recycling occurred in the European community. The principal uses of residue in Europe are as fill material and road construction material. Data available sources indicate that a significant quantity of residue is customarily used for these purposes. A number of facilities are designed privately and operated residue ash processing systems. They are designed primarily for the recovery of incinerated ferrous metals and some grading of residue for aggregate type uses. Widespread use of residue is reported in Germany, Switzerland, Sweden, Denmark and to a lesser extent France. In all these countries; residue is processed to remove incinerated ferrous metals and graded for use as an aggregate material which is usually used in road construction or as fill material. The vast majority of research in the United States has focused on the use of residue in various types of pavement. As documented in many reports, residue was found to perform acceptably when used in a residue/aggregate asphalt pavement or when mixed with lime as a binding agent. (R.W. Beck, 1987)

In addition to the main focus on pavement applications, research work has addressed a broad spectrum of possible uses for residue. Some of these research efforts have focused on using residues in Portland cement concrete, in the manufacture of cement and masonry bricks, as landfill cover material; and as a growth medium for different types of plants. (R.W. Beck, 1987)

While several promising applications have been identified for residue, the best demonstrated use is as an aggregate material for bituminous base course pavements. Residue has also been shown to perform excellently as

sub-base and fill material. The use of residue aggregate material in Portland cement concrete has not been demonstrated to be feasible at this time, primarily due to expansion problems resulting from residual aluminum and glass reactions with alkali materials. Portland cement concrete made with residue suffers from inadequate strength due to these expansion problems. However, residue may be utilized as a partial aggregate replacement or if methods are developed to remove aluminum or reduce alkali reactivity. For these same reasons, use of residue in cement stabilized pavements may not be considered feasible. Residue has been shown to perform well in lime stabilized pavements. Several paving projects have been completed and are performing well. Use of residue in surface course pavements, i.e., the wearing surface of the pavement, has not shown great success due to some problems of asphalt stripping. Two important by-products of any utilization scheme that can be produced from the residue are ferrous metals and incinerated aluminum. Marketing consideration must be taken into acccount with respect to their use. (R.W. Beck, 1987)

There are certain technical impediments in considering the use of ash in useful products. Perhaps the most significant factor influencing the use of residue ash is the degree of environmental uncertainty regarding its toxics characteristics. In addition, residue is not well demonstrated for any use other than bituminous pavement construction. Residue also is not well demonstrated as a wearing surface for pavement material. The use of residue in pavements is additionally limited by the lack of demonstration in pavment patching. Also, market receptivity is a factor in the utilization of ash. Market surveys and market education has to be performed in order to have the product accepted for use. The receptivity of the market could depend of the quantity of residue aggregate to the market and what share of the market that represents. (R.W. Beck, 1987)

Closely related to industry resistance is the potential for consumer reluctance to use residue aggregate. This is as a result of the fact that long term and widespread use of the residue as an aggregate material has not occurred and much concern could be expected as to the consistency and quality of the product. In addition, the environmental questions concerning leaching of trace metals and the perceived fear of the material being hazardous could diminish its use. (R.W. Beck, 1987)

Overall, it appears that until the information on the characteristics of the ash are more available in terms of its leaching and hazardous characteristics, it appears that it may be difficult to have residue ash accepted as a possible useful product. In the future, with more data and more research done on specific utilization techniques this resistance to its possible use could be overcome. Over the short term, it appears unlikely that the ash will be used in significant quantities, in any useful products until at a minimum the environmental questions are resolved. (R.W. Beck, 1987)

Treatment

Treatment processes are described below under Fly Ash, since treatment considerations are more applicable for fly ash treatments.

4.5.3.6.3 Fly Ash

Hazardous Wastes

As reported from the leachability test data presented, previously fly ash frequently exceeds the threshold limits of the EP Toxicity test. Therefore,

when disposed by itself, it is more likely to be classified as a hazardous waste and handled under Subtitle C of RCRA. The disposal cost associated with the disposal of fly ash as a hazardous waste averages $118 per ton of ash or greater depending on availability of hazardous waste landfill space and transportation considerations. (Christensen, 1988)

Utilization

Utilization of the fly ash by itself in products would be more difficult than using combined residue ash. Because of the greater likelihood of the fly ash exhibiting hazardous characteristics, it is not likely that fly ash would be used by itself in a useful product application until the issues surrounding trace metal leaching from the ash are better understood. (R.W. Beck, 1987)

Treatment

Treatment of the fly ash before disposal is a possibility for fly ash classified as a hazardous waste. Most of the waste-to-energy plants operating today in the United States do not generally treat the fly ash. It is usually combined with bottom ash and disposed of as a combined residue. There are several treatment methods for residue ash that have been discussed in the literature although their use is not readily documented. Various liming agents and other stabilizers have been used to fix the ash resulting in lower likelihood to leaching of trace metals. Such treatments have been indicated in the Waukesha, Wisconsin research work mentioned previously where the addition of specific amounts of liming agents enabled the ash to pass the EP Toxicity test. The Claremont, New Hampshire Facility is using a fly ash treatment system whereby the fly ash is treated with a proprietary system. Information on the system indicates that the fly ash is treated with an immobilizing agent, which may change the chemical form of lead from the soluble forms such as lead oxide to insoluble forms such as lead phosphate. The treatment system at the Claremont Facility may also include physical changes of the particle size distribution of the ash. Data taken on the ash at the Claremont facility indicates that the immobilization system appears to yield leachate concentrations of lead and cadmium well below the regulatory threshold limit when the combined bottom ash and treated fly ash are mixed before testing. The research work conducted at the Marine Sciences Research Center (Roethel et. al., 1987; Roethel, 1987) focused on the stabilization of incinerator ash (combined ash) into blocks which may be disposed of on land or in the ocean. The project, though still in its early stages of development, has achieved considerable success as reported by the Marine Sciences Research Center. Results of a variety of different mixes of incineration residues, lime, and cement have produced standard cement block sample with excellent structural properties. In addition, an artificial habitat using incineration ash blocks in Conscience Bay, Long Island Sound was established. Presently the Marine Science Research Center is monitoring the habitat to determine the interactions of stabilized incineration ash in the marine environment. The results of metal analysis by the use of the EP Toxicity test and TCLP test show that metal concentrations in these leachates do not exceed the limits for toxicity. In addition, biological studies using sensitive marine phytoplankton show no adverse impacts when exposed to leachate concentrations significantly higher than what could be expected under normal disposal conditions. Incineration ash blocks have also been formed which possess strength which may allow them to be used in construction in place of standard cement blocks. The American Society of Mechanical Engineers is presently investigating vitrification as a way to minimize leachate contamination (American Society of Mechanical Engineers, 1986). Vitrification is a process of smelting ash in an electric arc furnace at approximately 2600F to molten slag that is then quenched in water resulting

in a glass like frit (an amorphous, obsidian-like material). (R.W. Beck, 1987; Couppis, 1988)

The cost implications of the various treatment schemes discussed above are not known, since their development is not yet perfected. As a broad consideration, the cost for such treatments combined with monofill disposal are likely to vary between the cost of landfill disposal at a non-hazardous landfill and the cost of disposal at a hazardous waste landfill. (R.W. Beck, 1987)

4.5.3.6.4 Bottom Ash

Bottom ash from existing waste-to-energy plants has largely been found to pass the EP Toxicity test although there are certain samples that exceed the regulatory threshold limits. Disposal of this material in a monofill would generally present similar environmental impact as was discussed in Section 4.5.3.6.3 - Combined Bottom and Fly Ash. The leaching characteristics in monofills have been shown to be less than what is predicted by the EP Toxicity test. The cost for monofill disposal is expected to be approximately in the range of $35 to $45 per ton of ash. (R.W. Beck, 1987)

Utilization of the bottom ash as a useful product generally presents similar possibilities as those mentioned for the combined bottom and fly ash.

Should bottom ash be classified as a hazardous waste, if tests results so indicate, it can either be disposed of as a hazardous waste or treated to render it non-hazardous. The treatment methods would be similar to those identified in Section 4.5.3.6.3. Costs for such treatments would depend on the type of treatment. Costs associated with hazardous waste disposal of bottom ash would be substantial because of the quantity of ash to be landfilled. Approximately one-third by weight of the waste incinerated will become bottom ash. (R.W. Beck, 1987)

Table 4.5A

Comparison of the Extraction Procedure (EP) and the Toxicity Characteristic Leaching Procedure (TCLP) (1)

Item	EP	TCLP
1) Leaching Media	- 0.5 N Acetic acid added to distilled deionized water to a pH of 5 with 400 ml maximum addition. - Continual ph adjustment.	-0.1 N pH 2.9 acetic acid solution for moderate to high alkaline wastes and 0.1 N pH 4.9 acetate buffer for other wastes.
2) Liquid/solid separation	- 0.45 um Filtration to 75 psi in 10 psi increments. - Unspecified filter type.	-0.6-0.8 um Glass fiber filter filtration to 50 psi.
3) Monolithic material/particle size reduction	- Use of Structural Integrity Procedure or grinding and milling.	-Grinding or milling only. -Structural Integrity Procedure not used.
4) Extraction Vessels	- Unspecified design - Blade/stirrer vessel acceptable.	-Zero-headspace vessel required for volatiles. -Bottles used for non-volatiles -Blade stirrer vessel not used.
5) Agitation	- Acceptable agitation not well defined.	-Rotary agitation only in an end-over-end fashion at 30 +/- 2 rpm.
6) Extraction time	- 24 hours.	-18 hours.
7) Quality Control requirements	- Standard additions required. - One blank per sample batch.	-Standard additions required in some cases. -One blank per day of for every new batch of extract. -Analysis specific to analyte

1) All other attributes between the two tests are generally the same, although there are some minor differences. Note also that while the EP only addresses those species for which National Interim Primary Drinking Water Standards (NIPDWS) exist, the TCLP can be applied to other toxicants.

Table 4.5B

Composition of Fly and Combined Ash

Amount of Fly Ash (lb/hr)

Constituent	SR=1.5				SR=2.0			
	1% FA (1)		10% FA		1% FA		10% FA	
Fly Ash	13	18%	130	68%	13	14%	130	63%
Reaction Products	37	50%	37	19%	37	41%	37	18%
Unreacted Lime	24	32%	24	13%	40	45%	40	19%
Total	74	100%	191	100%	90	100%	207	100%

Amount of Combined Ash (lb/hr) (2)

Constituent	SR=1.5		SR=2.0	
Combined Ash	2167	97%	2167	96%
Reaction Products	37	2%	37	2%
Unreacted Lime	24	1%	40	2%
Total	2228	100%	2244	100%

1) Average percentage of fly ash obtained.
2) Bottom Ash includes 40% moisture.

TABLE 4.5C
COMPOSITION OF MUNICIPAL SOLID WASTE INCINERATED AT THE FACILITY (1)

COMPOSITION

Material	Typical Percent by Weight	Range Percent by Weight
Paper	35.2	25-60
Plastics	4.0	2-8
Wood	17.2	1-20
Yard Wastes	10.5	0-34
Textiles	2.8	0-9
Food Wastes	5.5	4-26
Rubber	1.5	0-3
Miscellaneous Organics	1.1	0-2
Glass	3.2	2-23
Ferrous Metals	8.1	2-11
Aluminum	1.3	0-2
Miscellaneous Inorganics	9.6	0-10

PROXIMATE ANALYSIS

Constitutent	Typical Percent by Weight	Range Percent by Weight
Combustibles	53	47-58
Moisture	26	18-40
Ash	21	20-25

ULTIMATE ANALYSIS

Constituent	Percent by Weight
Moisture	26
Ash	20.7
Sulfur	0.1
Nitrogen	0.5
Carbon	27.2
Hydrogen	3.6
Oxygen	21.5
Chlorine	0.4

1) Data supplied by Black & Veatch

Table 4.5D

Chemical Composition of Incinerator Ash

Element		Unit	Bottom Ash		Fly Ash from ESP		Natural Soil
			Inc. A	Inc. B	Inc. A	Inc. B	Sandy Clay
Silicon	Si	g/kg	271	285	172	186	298
Aluminum	Al	g/kg	49	47	78	77	37
Chlorine	Cl	g/kg	-	-	-	-	-
Iron	Fe	g/kg	27	31	18	19	16
Calcium	Ca	g/kg	68	65	116	102	83
Potassium	K	g/kg	12	13	30	44	17
Magnesium	Mg	g/kg	8.4	7.7	19	17	6.9
Sodium	Na	g/kg	37	35	23	29	6.6
Sulfur	S	g/kg	2.2	1.3	11	18	1
Phosphorus	P	g/kg	3.7	4.1	8.3	9.6	0.7
Titanium	Ti	g/kg	3.2	4.2	9.5	9.3	2.2
Manganese	Mn	g/kg	0.9	1	1	1.1	0.4
Silver	Ag	mg/kg	7.1	14	-	-	0.048
Arsenic	As	mg/kg	24	19	49	71	13
Barium	Ba	mg/kg	1900	1700	1600	1000	-
Cadmium	Cd	mg/kg	1.4	3.2	250	250	0.28
Cobalt	Co	mg/kg	30	23	61	29	-
Chromium	Cr	mg/kg	230	230	180	220	24
Copper	Cu	mg/kg	2400	4300	890	1300	8.5
Mercury	Hg	mg/kg	0.09	0.14	1.4	2.4	0.11
Molybdenum	Mo	mg/kg	2.5	5.4	26	29	<4
Nickel	Ni	mg/kg	68	97	120	110	8.3
Lead	Pb	mg/kg	3100	2300	7400	7800	11
Selenium	Se	mg/kg	6	8	6.1	11	<1
Strontium	Sr	mg/kg	250	170	250	250	300
Vanadium	V	mg/kg	36	36	52	32	34
Zinc	Zn	mg/kg	2300	2500	25000	22000	37

Table 4.5E

Major Components of Ash from Six Swedish Incinerators

Substance	Slag %	Fly Ash %	Dry flue gas product, %	Flue gas condensate, %
SiO2	49.8–60.0	31.6–63.6	4.4–14.0	14.9–17.1
Fe2O3	5.3–7.0	2.0–5.7	0.2–1.1	3.1–3.6
Al2O3	11.2–13.6	11.5–20.6	2.5–8.1	6.9–7.6
CaO	11.0–13.6	9.4–15.5	26.4–34.9	36.9–38.6
K2O	1.6–2.7	2.6–7.2	1.9–3.1	<0.1
Na2O	4.8–6.9	2.9–5.7	2.1–3.3	0.1–0.2
MgO	2.1–3.2	2.0–4.6	1.3–2.5	4.2–5.9
P2O5	1.4–2.9	1.2–2.5	0.3–0.8	0.4
TiO2	0.7–1.0	0.5–2.1	<0.4	0.1–0.2
MnO	<0.2	0.1–0.6	<0.8	0.2
S	0.2–0.8	0.4–1.7	1.0–1.3	0.3–0.4
Cl	0.3–0.9	0.3–4.7	9.5–13.9	0.6–0.9
Unburnt	3.0–4.9	4.9–9.8	<6.4	2.4–4.5

TABLE 4.5F

CHEMICAL COMPOSITION OF INCINERATOR FROM SIX SWEDISH INCINERATORS

ELEMENT	SLAG (mg/kg)	FLY ASH (mg/kg)	DRY SCRUBBER + FLY ASH (mg/kg)	FLUE GAS CONDENSATE (mg/kg)
Arsenic (As)	20-80	40-140	<10-80	30-40
Barium (Ba)	900-1600	870-2300	190-660	150-180
Lead (Pb)	1300-5400	1200-3600	2200-3300	2399-2900
Cadmium (Cd)	4-40	0.3-110	130-180	70-100
Cobalt (Co)	<10-40	<10-15	<10	<10
Copper (Cu)	900-3200	700-5000	300-630	400-550
Chromium (Cr)	300-600	260-2400	140-450	300-650
Mercury (Hg)	<0.10-3	2-40	8.8-100	1200
Molybdenum (Mo)	20-40	15-50	60-80	30-50
Nickel (Ni)	60-160	80-200	20-50	350-750
Strontium (Sr)	190-350	270-550	100-400	160-170
Tin (Sn)	<100-1300	<100-810	500-600	300-450
Thorium (Th)	70-300	80-180	190-230	320-380
Vanadium (V)	50-90	60-110	<20-30	70-90
Wolfram (W)	<20-50	<20-90	<20	<20
Zinc (Zn)	1800-5900	2500-14000	7800-11400	5700-6400

TABLE 4.5G

Fly Ash Contaminant Leachability

Scrubber Vessel Ash

Ash Composition		Available Fraction (%) for Various Leaching Media		
		Weak (Water)	Aggressive (Dilute Acid)	Severe (Strong Acid)
pH	12	-	-	-
TDS (mg/l)	3,500	-	-	-
Cadmium (ug/g)	40	0	55	55
Chromium (ug/g)	700	0	5	15
Copper (ug/g)	700	5	10	30
Nickel (ug/g)	800	0	25	70
Lead (ug/g)	2,000	5	50	75
Zinc (ug/g)	5,500	0	45	60
Boron (ug/g)	70	0	100	100
Cobalt (ug/g)	600	0	60	80
Total Sulfur (ug/g)	12,000	-	-	-

Baghouse Ash

Ash Composition		Available Fraction (%) for Various Leaching Media		
		Weak (Water)	Aggressive (Dilute Acid)	Severe (Strong Acid)
pH	11	-	-	-
TDS (mg/l)	11,000	-	-	-
Cadmium (ug/g)	235	0	80	95
Chromium (ug/g)	600	0	10	20
Copper (ug/g)	600	2	15	40
Nickel (ug/g)	150	0	15	40
Lead (ug/g)	6,500	5	60	85
Zinc (ug/g)	13,000	0	55	70
Boron (ug/g)	250	2	40	70
Cobalt (ug/g)	130	0	30	30
Total Sulfur (ug/g)	12,000	-	-	-

TABLE 4.5H

Range of Leachate Analysis of Marion County
Residue Ash - Lead - EP Toxicity Test

Sample	Leachate Concentration
	(mg/l)
1	4.10
2	2.19
3	3.13
4	0.56
5	2.34
6	1.14
7	2.12
8	5.16
9	3.39
10	4.34
11	3.42
12	21.00
13	1.05
14	1.64
15	0.74
16	3.26
17	1.07
18	2.47
Average	3.51
Variance	20.80
Regulatory Threshold	5.00

TABLE 4.5I

Range of Leachage Analysis of Marion County Residue Ash

(milligrams per liter)

Ash Composition	TCLP	DI Leach	Total Metals (2)	EPTox Limit
1. Alkalinity as CaCO3	---	147-740	---	---
2. Arsenic	<0.05	<0.05	<50.0	5.0
3. Barium	0.146-0.229	0.169-0.370	165-367	100.0
4. Cadmium	0.01-0.229	<0.05	12.3-24.9	1.0
5. Calcium	362-1400	150-304	---	---
6. Chlorine	417-625	439-611	---	---
7. Chromium	<0.01-0.073	<0.01-35.9(1)	24.6-564	5.0
8. Fluoride	---	<0.1-1.37	---	---
9. Harness	905-3650	375-984	---	---
10. Lead	0.05-6.10	<0.05-2.98	12.7-6250	5.0
11. Magnesium	0.14-25.1	<0.001	---	---
12. Mercury	<0.1	<0.1	<5.0	.02
13. Nitrate	---	0.1-0.2	---	---
14. Selenium	<0.05	<0.05	<50.0	1.0
15. Silver	<0.05	<0.05	<2.5	5.0
16. Sulfate	260-1450	245-571	---	---

(1) The 35.9 level appears to be either an error in reporting or in analysis of the data since the total metals by acid digestion also indicates a level of 35.9 milligrams per liter. This would mean that all the chromium in the ash leached out, an unlikely event.

(2) mg/kg

TABLE 4.5J

LEACHATE ANALYSIS OF MARION COUNTY

RESIDUE ASH - CADMIUM - EP TOXICITY TEST

Sample	Leachate Concentration (mg/l)
1	0.35
2	0.18
3	0.21
4	0.09
5	0.23
6	0.10
7	0.24
8	0.35
9	0.36
10	0.22
11	0.13
12	0.21
13	0.20
14	0.31
15	0.19
16	0.36
17	0.21
18	0.44
Average	0.24
Variance	0.009
Regulatory Threshold	1.0

TABLE 4.5K

BARRON COUNTY FACILITY EP TOXICITY TESTS - LEAD (1)

JUNE 1987		JULY 1987		AUGUST 1987		SEPTEMBER 1987	
DATE	CONC.(mg/l)	DATE	CONC.(mg/l)	DATE	CONC.(mg/l)	DATE	CONC.(mg/l)
6/11	0.8	7/1	3.7	8/2	4.0	9/2	2.3
6/12	2.9	7/5	1.5	8/5	0.5	9/6	9.5
6/13	1.4	7/8	1.8	8/9	2.2	9/9	1.4
6/14	1.9	7/12	6.4	8/12	2.1	9/13	8.2
6/15	8.0	7/15	5.6	8/16	1.2	9/16	0.8
6/16	3.6	7/19	1.2	8/19	1.7	9/20	1.0
6/17	17.0	7/22	4.9	8/23	4.5	9/23	2.3
6/18	3.1	7/26	11.0	8/26	0.4		
6/21	3.4	7/29	6.6	8/30	0.7		
6/24	3.6						
6/28	8.3						
Aver.	4.9		4.7		1.9		3.6

1) Bottom Ash only. Fly Ash is disposed of as a hazardous waste.

2) Sampling was done hourly and composited into daily or three-day composite samples for laboratory testing.

TABLE 4.5L

EP TOXICITY TEST RESULTS ON LEAD AND CADMIUM
FROM COMPOSITE RESIDUE SAMPLES AT RESCO, MASSACHUSETTS INCINERATOR

SAMPLE DATE	SAMPLE NO. *	LAB **	LEAD REP 1	LEAD REP 2	CADMIUM REP 1	CADMIUM REP 2
5/7	1	SES	7.77	8.36	0.45	0.48
		Micro	9.10	9.10	0.45	0.45
	2	SES	6.01	6.51	0.38	0.40
		Micro	8.10	8.20	0.40	0.40
5/8	1	SES	10.73	11.25	0.84	0.88
		Micro	13.00	13.20	0.90	0.90
	2	SES	8.87	8.80	0.78	0.78
		Micro	10.00	9.70	0.75	0.75
5/9	1	SES	15.60	15.57	0.85	0.84
		Micro	17.20	17.80	0.85	0.84
	2	SES	16.00	16.22	0.87	0.87
		Micro	18.50	18.50	0.85	0.85
5/10	1	SES	8.13	8.36	0.62	0.63
		Micro	10.00	10.20	0.65	0.65
	2	SES	7.77	7.74	0.65	0.65
		Micro	9.40	9.40	0.65	0.65
5/11	1	SES	7.49	7.87	0.57	0.60
		Micro	9.00	9.10	0.57	0.60
	2	SES	7.87	7.74	0.52	0.51
		Micro	9.30	9.20	0.50	0.50
5/12	1	SES	5.85	6.02	0.62	0.63
		Micro	6.40	6.50	0.60	0.60
	2	SES	4.96	4.86	0.57	0.56
		Micro	6.60	6.70	0.55	0.55
5/13	1	SES	0.20	0.25	0.17	0.17
		Micro	<0.10	<0.10	0.15	0.15
	2	SES	3.65	4.46	0.28	0.34
		Micro	6.40	6.10	0.30	0.30
5/13	1	SES	0.59	0.73	0.18	0.18
quench		Micro	2.40	2.70	0.20	0.25
5/24	1	SES	47.37	46.42	0.70	0.69
		DPW	58.30	58.60	0.84	0.85
	2	SES	6.69	6.59	0.89	0.90
		DPW	4.49	3.88	0.92	0.92

TABLE 4.5L

EP TOXICITY TEST RESULTS ON LEAD AND CADMIUM
FROM COMPOSITE RESIDUE SAMPLES AT RESCO, MASSACHUSETTS INCINERATOR

SAMPLE DATE	SAMPLE NO. *	LAB **	LEAD REP 1	LEAD REP 2	CADMIUM REP 1	CADMIUM REP 2
6/1	1	SES	11.73	11.91	0.89	0.90
		DPW	14.19	13.79	1.11	1.09
	2	SES	5.31	5.68	0.79	0.77
		DPW	5.56	5.79	0.97	0.97
6/7	1	SES	17.48	17.60	0.88	0.90
		DPW	18.69	18.19	0.96	0.96
	2	SES	9.50	9.57	0.66	0.67
		DPW	11.25	10.96	0.81	0.83
6/10	1	SES	5.70	LOST	0.54	LOST
		DPW	6.31	6.70	0.74	0.74
	2	SES	10.35	10.41	0.68	0.68
		DPW	11.69	11.39	0.87	0.90
6/17	1	SES	5.56	5.36	0.63	0.62
		DPW	6.58	6.27	0.81	0.79
	2	SES	6.31	6.52	0.59	0.56
		DPW	7.16	7.23	0.77	0.77

* "SAMPLE NO.": Each residue sample was divided into 2 sub-samples for EP testing.

** "Lab": SES = Suburban Experiment Station Soils laboratory, UMass, Waltham (ICAP = Inductively Coupled Argon Plasma Emission spectrophotometer); Micro = Microanalysis Laboratory at UMass, Amherst

(AA = Atomic absorption spectrophotometer); DPW = Department of Public Works Labroatory, Wellesley, MA (AA)

TABLE 4.5M

EP TOXICITY TEST DATA ON LEAD AND CADMIUM
IN BOTTOM ASH AT RESCO MASSACHUSETTS INCINERATOR
(Expressed in ppm.)

COLLECTION PERIOD (1982)

DAY	TIME	LEAD	CADMIUM
6/3	8AM	12.40	0.28
6/3	9AM	0.41	0.01
6/3	10AM	0.38	0.03
6/3	11AM	2.62	0.08
6/3	12N	7.11	0.06
6/3	1PM	3.65	0.03
6/3	2PM	0.18	0.02
6/3	3PM	0.15	0.02
6/3	4PM	1.70	0.04
6/3	5PM	1.81	0.03
6/3	6PM	1.52	0.03
6/3	7PM	3.04	0.04
6/3	8PM	3.01	0.05
6/3	9PM	0.57	0.04
6/3	10PM	0.98	0.02
6/3	11PM	1.19	0.03
6/3	12M	3.23	1.59
6/4	1AM	7.93	0.08
6/4	2AM	0.53	0.12
6/4	3AM	1.56	0.11
6/4	4AM	4.22	0.21
6/4	5AM	3.69	0.07
6/4	6AM	2.11	0.04
6/4	7AM	9.44	0.10
6/4	8AM	4.27	0.07
6/4	9AM	0.51	0.04
6/4	10AM	0.40	BDL
6/4	11AM	1.79	0.04
6/4	12N	1.26	0.02
6/4	1PM	5.28	0.12
6/4	2PM	0.34	0.02
6/4	3PM	1.10	0.02

TABLE 4.5M

EP TOXICITY TEST DATA ON LEAD AND CADMIUM
IN BOTTOM ASH AT RESCO MASSACHUSETTS INCINERATOR
(Expressed in ppm.)

COLLECTION PERIOD (1982)

DAY	TIME	LEAD	CADMIUM
6/4	4PM	0.65	0.02
6/4	5PM	0.98	0.03
6/4	6PM	2.04	0.02
6/4	7PM	1.60	0.03
6/4	8PM	4.43	0.06
6/4	9PM	0.08	BDL
6/4	10PM	0.59	0.01
6/4	11PM	9.21	0.02
6/4	12M	2.87	0.03
6/5	1AM	2.54	0.06
6/5	2AM	1.81	0.02
6/5	3AM	0.21	BDL
6/5	4AM	3.66	0.03
6/5	5AM	1.15	0.04
6/5	6AM	1.58	0.09
6/5	7AM	15.08	0.08
8/10	8AM	4.88	0.07
8/10	9AM	2.33	0.06
8/10	10AM	15.70	0.43
8/10	11AM	7.67	0.06
8/10	12N	16.52	0.05
8/10	1PM	5.98	0.06
8/10	2PM	1.97	0.02
8/10	3PM	10.93	0.02
8/10	4PM	4.59	0.01
8/10	5PM	0.48	0.01
8/10	6PM	1.27	0.04
8/10	7PM	2.00	0.06
8/10	8PM	14.39	0.03
8/10	9PM	3.87	0.02
8/10	10PM	3.06	0.01
8/10	11PM	11.54	0.03
8/10	12M	6.06	0.06
8/11	1AM	9.80	0.02
8/11	2AM	8.64	0.03
8/11	3AM	9.44	0.04
8/11	4AM	5.24	0.04
8/11	5AM	1.05	0.02

TABLE 4.5M

EP TOXICITY TEST DATA ON LEAD AND CADMIUM
IN BOTTOM ASH AT RESCO MASSACHUSETTS INCINERATOR
(Expressed in ppm.)

COLLECTION PERIOD (1982)

DAY	TIME	LEAD	CADMIUM
8/11	6AM	4.41	0.09
8/11	7AM	5.71	0.04
8/11	8AM	5.92	0.03
8/11	9AM	2.85	0.07
8/11	10AM	1.19	0.06
8/11	11AM	21.19	0.12
8/11	12N	1.19	0.04
8/11	1PM	0.04	0.02
8/11	2PM	14.67	0.40
8/11	3PM	11.30	0.33
8/11	4PM	33.42	0.18
8/11	5PM	27.34	0.53
8/11	6PM	20.70	0.07
8/11	7PM	7.28	0.03
8/11	8PM	1.72	0.02
8/11	9PM	1.98	0.04
8/11	10PM	8.30	0.05
8/11	11PM	4.64	0.14
8/11	12M	2.05	0.03
8/12	1AM	2.76	0.03
8/12	2AM	1.33	0.02
8/12	3AM	1.71	0.02
8/12	4AM	1.66	0.02
8/12	5AM	2.92	0.01
8/12	6AM	1.36	0.02
8/12	7AM	5.59	0.18

BDL = Below Detectable Limits

TABLE 4.5N

COMPARISON OF EQUILIBRIUM pH UNDER
EP TOXICITY TEST EXTRACTION CONDITION OF
DOLOMITIC LIMESTONE VERSUS POWDERED CALCITIC LIMESTONE

Limestone Added (%)	Dolomitic Limestone $Ca/MgCO_3$	Calcitic Limestone $CaCO_3$
5	4.4	4.3
10	4.8	5.9
15	5.0	5.9
20	5.1	5.9
25	5.3	6.2
30	5.3	6.3

* In equilibrium with 200 ml distilled water and 50 ml 4N acetic acid

TABLE 4.5O

COMPARISON OF EP TOXICITY EQUILIBRIUM pH OVER TIME
OF ASH TREATED IN DUPLICATE AT THE 10% LEVEL WITH DOLOMITIC LIMESTONE,
POWDERED CALCITIC LIMESTONE AND LIME

	Dolomitic Limestone $Ca/MgCO_3$		Powdered Calcitic Limestone $CaCO_3$		Powdered Lime CaO	
TEST TIME	1	2	1	2	1	2
Initial	10.6	10.6	10.6	10.6	10.6	10.6
+ Liming Agent	10.6	10.5	10.6	10.6	12.2	12.3
+ Acetic Acid	4.3	4.2	5.1	5.1	12.1	12.1
After 1 Hour	4.8	4.7	5.8	5.8	12.2	12.1
After 2 Hour	4.8	4.8	5.9	5.9	12.3	12.2
After 3 Hour	4.8	4.8	5.9	5.9	12.0	12.0
After 24 Hour	4.9	4.9	6.0	6.0	12.0	12.1
After 120 Hour	4.9	4.9	6.1	6.1	12.0	12.1

TABLE 4.5P

RESULTS OF EP TOXICITY TESTING ASH TREATED WITH CRACKLING LIME

	Concentration (mg/l) Sample Designation				
	1201	1201	1201	1201	Blank
Lime (gms)	15	20	30	50	100
Final pH	9.1	11.3	12.2	12.3	12.3
Arsenic	--	--	--	--	<.01
Barium	--	--	--	--	0.16
Cadmium	<.01	<.01	<.01	<.01	<.01
Chromium	--	--	--	--	<.01
Lead	<.01	<.01	4.58	4.08	<.01
Selenium	--	--	--	--	0.02
Silver	--	--	--	--	<.01
Mercury	--	--	--	--	<.0005

TABLE 4.5Q

NEW YORK EXTRACTION PROCEDURE TOXICITY TEST RESULTS

(FIRST ROUND TESTING)

Lead (ppm)

Residue Sample	# Samples	Mean	Std. Dev.	Range
Bottom Ash	12	5.6	7.8	<0.2-27
Fly Ash	8	17.8	8.8	8.6-34
Combined Ash	17	6.8	6.9	0.2-21

Cadmium (ppm)

Residue Sample	# Samples	Mean	Std.Dev.	Range
Bottom Ash	12	0.094	0.16	<0.02-0.58
Fly Ash	8	30.5	38.5	2.1-90
Combined Ash	17	0.7	0.8	0.11-2.8

Statistical Functions calculated in accordance with
"Test Methods for Evaluating Solid Waste - Physical/Chemical Methods",
USEPA, SW-846 2nd Ed.

TABLE 4.5R

EXTRACTION PROCEDURE TOXICITY TEST RESULTS FOR CADMIUM (mg/l)
Statistical Analysis of Replicate Chemical Extractions
OSWEGO FACILITY

SAMPLE	MEAN	MEDIAN	RANGE	STD. DEV.	VARIANCE	COEF. OF VAR.	80% CONF. INTERV. LL	80% CONF. INTERV. UL
Day 1	1.15	1.10	0.93-1.30	0.16	0.026	13.19	1.04	1.26
Day 2	0.92	0.90	0.86-1.00	0.057	0.003	6.20	0.88	0.96
Day 3	0.58	0.58	0.46-0.74	0.11	0.012	18.96	0.51	0.66
Day 4	0.53	0.51	0.46-0.65	0.07	0.005	13.21	0.49	0.57
Day 5	0.41	0.40	0.39-0.45	0.03	0.001	7.32	0.39	0.43
Day 6	0.51	0.52	0.49-0.52	0.01	0.0001	1.96	0.50	0.52
Day 7	0.42	0.42	0.41-0.43	0.01	0.0001	2.38	0.41	0.43
Weekly Composite	0.53	0.49	0.47-0.71	0.10	0.01	18.97	0.46	0.60
Mathemat. Composite	0.65	0.51	0.39-1.30	0.27	0.074	41.53	0.59	0.71
TCLP	0.32	0.33	0.25-0.36	0.042	0.00175	13.13		

TABLE 4.5S

EXTRACTION PROCEDURE TOXICITY TEST RESULTS LEAD (mg/l) - OSWEGO FACILITY

Statistical Analysis of Replicate Chemical Extractions

SAMPLE	MEAN	MEDIAN	RANGE	STND. DEV.	VARIANCE	COEF. of VARIANCE	80% CONF. INTERVAL LL	UL
Day 1	7.52	6.7	6.4-9.5	1.37	1.877	18.21	6.58	8.45
Day 2	9.6	9.7	7.7-12	1.57	2.465	16.35	8.52	10.67
Day 3	3.1	2.7	1.9-5.4	1.35	1.823	43.55	2.17	4.02
Day 4	3.4	2.9	1.6-5.1	1.44	2.074	42.35	2.41	4.38
Day 5	6.0	5.7	2.7-13	4.21	17.724	70.18	3.11	8.88
Day 6	6.1	4.9	2.7-13	3.99	15.920	65.41	3.36	8.83
Day 7	7.6	8.1	3.2-13	3.64	13.250	47.89	5.10	10.09
WEEKLY COMPOSITE	8.1	3.9	1.1-25	9.81	96.236	121.11	1.37	14.82
MATHEMAT. COMPOSITE	6.18	5.6	1.6-13	3.37	11.36	54.53	5.48	6.92
TCLP	0.29	0.3	0.24-0.35	0.048	0.0023	16.55		

LL - Lower Confindence Unit
UL - Upper Confidence Unit

TABLE 4.5T

CLAREMONT,NH SOLID WASTE PROJECT

EXTRACTION PROCEDURE TOXICITY OF MUNICIPAL INCINERATOR ASH

TEST DATE	SAMPLE NO.	INITIAL pH	FINAL pH	LEAD (mg/l)	CADMIUM (mg/l)	TEST HOURS	#ofLEAD VIOL.	#ofCAD. VIOL.
26-May-87	1	12.7	6.44	1.5	0.58	24	0	0
26-May-87	2	12.73	7.99	0.15	0.31	24	0	0
26-May-87	3	12.73	5.39	7.3	0.4	24	1	0
26-May-87	5	12.69	5.49	7.3	0.5	24	1	0
26-May-87	6	12.68	5.7	6.3	0.57	24	1	0
26-May-87	7	12.64	5.26	13	1.1	24	1	1
26-May-87	8	12.67	5.31	16	0.69	24	1	0
26-May-87	1DUP.	12.69	6.48	1.5	0.59	24	0	0
26-May-87	2DUP.	12.73	7.58	0.16	0.54	24	0	0
26-May-87	3DUP.	12.7	5.38	5.6	0.4	24	1	0
26-May-87	5DUP.	12.74	5.46	6.8	0.49	24	1	0
26-May-87	6DUP.	12.73	5.45	12	0.6	24	1	0
26-May-87	7DUP.	12.66	5.17	17	0.7	24	1	0
27-May-87	8DUP.	12.71	5.29	15	0.53	24	1	0
18-Jun-87	COMP.1	12.06	5.03	9.9	0.72	24	1	0
18-Jun-87	COMP.2	12.09	5.13	12	0.84	24	1	0
18-Jun-87	CLAR.1	12.09	5.04	12	0.62	24	1	0
18-Jun-87	CLAR.2	12.08	5.18	10	0.77	24	1	0
18-Jun-87	CLAR.3	12.22	6.31	2.3	1.2	24	0	1
18-Jun-87	CLAR.4	12.15	5.28	8.2	0.9	24	1	0
18-Jun-87	CLAR.5	12.03	4.82	17	0.7	24	1	0
18-Jun-87	CLAR.6	12.00	4.82	12	0.65	24	1	0
18-Jun-87	CLAR.7	12.04	4.82	8.8	0.66	24	1	0
18-Jun-87	CLAR.8	11.97	4.69	12	0.57	24	1	0
08-Jun-87	1000	12.07	6.55	0.27	0.56	1	0	0
08-Jun-87	545	12.01	5.98	0.34	0.69	1	0	0
08-Jun-87	1100	12.03	12.19	1	0.02	1	0	0
08-Jun-87	1110	12.05	12.04	0.54	0.02	1	0	0
08-Jun-87	1120	12.04	11.97	0.64	0.03	1	0	0
08-Jun-87	1130	12.09	12.04	0.66	0.03	1	0	0
08-Jun-87	1140	12.07	11.93	0.68	0.02	1	0	0
09-Jun-87	1100	12.47	11.05	0.19	<0.004	24	0	0
22-Jun-87	COMP.1	12.34	8.5	0.11	0.10	24	0	0
22-Jun-87	COMP.1	12.37	9.42	0.11	0.022	24	0	0
22-Jun-87	COMP.1	12.38	9.35	0.11	0.026	24	0	0

TABLE 4.5T

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CLAREMONT,NH SOLID WASTE PROJECT

EXTRACTION PROCEDURE TOXICITY OF MUNICIPAL INCINERATOR ASH

TEST DATE	SAMPLE NO.	INITIAL pH	FINAL pH	LEAD (mg/l)	CADMIUM (mg/l)	TEST HOURS	#ofLEAD VIOL.	#ofCAD. VIOL.
22-Jun-87	COMP.2	12.38	9.28	0.11	0.024	24	0	0
22-Jun-87	2	12.27	5.47	0.22	1.20	24	0	1
22-Jun-87	3	12.35	5.28	0.13	0.66	24	0	0
22-Jun-87	4	12.36	9.74	0.11	0.008	24	0	0
22-Jun-87	5	12.27	12.12	4.9	<0.005	24	0	0
22-Jun-87	6	12.26	12.1	3.5	<0.005	24	0	0
22-Jun-87	7	12.27	12.21	6.7	<0.005	24	1	0
22-Jun-87	8	12.32	12.29	5.1	<0.005	24	1	0
20-Jul-87	1	12.10	6.32	<0.1	0.44	24	0	0
20-Jul-87	2	12.03	6.16	<0.1	0.49	24	0	0
20-Jul-87	3	12.08	6.31	<0.1	0.43	24	0	0
20-Jul-87	4	12.07	6.03	<0.1	0.42	24	0	0
20-Jul-87	5	11.7	4.97	<0.1	0.76	24	0	0
20-Jul-87	6	11.68	4.91	<0.1	0.81	24	0	0
20-Jul-87	7	12.28	6.61	<0.1	0.14	24	0	0
20-Jul-87	8	12.26	6.2	<0.1	0.17	24	0	0
20-Jul-87	9	10.92	5.11	0.18	0.16	24	0	0
20-Jul-87	10	10.4	4.99	0.35	0.51	24	0	0
04-Aug-87	1	11.63	6.41	<0.05	0.45	24	0	0
04-Aug-87	6	11.38	5.2	0.075	0.79	24	0	0
04-Aug-87	7	12.07	6.33	<0.05	0.2	24	0	0
04-Aug-87	9	10.55	5.07	0.48	0.35	24	0	0
26-Aug-87	1	9.96	5.15	<0.1	0.69	24	0	0
26-Aug-87	2	10.02	5.09	<0.1	0.69	24	0	0
26-Aug-87	3	8.99	5.2	<0.1	0.37	24	0	0
26-Aug-87	4	8.91	5.2	<0.1	0.35	24	0	0
26-Aug-87	5	9.92	5.19	<0.1	0.41	24	0	0
26-Aug-87	6	9.87	5.18	<0.1	0.43	24	0	0
26-Aug-87	7	10.0	5.0	<0.1	0.61	24	0	0
TOTALS							21	3
AVERAGE				3.75	0.45			
MAXIMUM				17.0	1.2			

TABLE 4.5U

CLAREMONT,NH SOLID WASTE PROJECT

WATER LEACHING TOXICITY OF MUNICIPAL INCINERATOR ASH

TEST DATE	SAMPLE NO.	INITIAL pH	FINAL pH	LEAD (mg/l)	CADMIUM (mg/l)	TEST HOURS	#ofLEAD VIOL.	#ofCAD. VIOL.
26-May-87	1	12.68	12.69	15	<0.005	24	1	0
26-May-87	3	12.67	12.72	8.3	<0.005	24	1	0
26-May-87	6	12.65	12.73	8.2	<0.005	24	1	0
18-Jun-87	COMP.1	12.12	12.03	6.8	<0.005	24	1	0
18-Jun-87	CLAR.1	12.09	12.03	9	<0.005	24	1	0
18-Jun-87	CLAR.2	12.1	12.03	5.3	<0.005	24	1	0
18-Jun-87	CLAR.3	12.27	12.16	12	<0.005	24	1	0
18-Jun-87	CLAR.4	12.2	12.11	8	<0.005	24	1	0
18-Jun-87	CLAR.5	12.07	11.99	7.9	<0.005	24	1	0
18-Jun-87	CLAR.6	12.03	11.97	6.3	<0.005	24	1	0
18-Jun-87	CLAR.7	12.06	11.97	5.4	<0.005	24	1	0
18-Jun-87	CLAR.8	11.99	11.92	5.2	<0.005	24	1	0
22-Jun-87	COMP.1	12.41	12.42	7.00	<0.005	24	1	0
22-Jun-87	2	12.31	12.43	6.8	<0.005	24	1	0
22-Jun-87	3	12.36	12.44	4.4	<0.005	24	0	0
22-Jun-87	4	12.41	12.45	9.8	<0.005	24	1	0
22-Jun-87	5	12.34	12.41	9.4	<0.005	24	1	0
22-Jun-87	6	12.38	12.45	7.3	<0.005	24	1	0
22-Jun-87	7	12.34	12.42	9.4	<0.005	24	1	0
22-Jun-87	8	12.4	12.42	7.2	<0.005	24	1	0
20-Jul-87	1	12.02	12.2	1.3	<0.005	24	0	0
20-Jul-87	2	12.08	12.25	1.4	<0.005	24	0	0
20-Jul-87	3	12.08	12.23	1.6	<0.005	24	0	0
20-Jul-87	4	12.05	12.26	1.5	<0.005	24	0	0
20-Jul-87	5	11.83	11.96	1.1	<0.005	24	0	0
20-Jul-87	6	11.76	11.91	0.96	<0.005	24	0	0
20-Jul-87	7	12.31	12.39	4.6	<0.005	24	0	0
20-Jul-87	8	12.32	12.35	4.5	<0.005	24	0	0
20-Jul-87	9	11.07	11.53	<0.1	<0.005	24	0	0

TABLE 4.5U

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CLAREMONT,NH SOLID WASTE PROJECT

WATER LEACHING TOXICITY OF MUNICIPAL INCINERATOR ASH

TEST DATE	SAMPLE NO.	INITIAL pH	FINAL pH	LEAD (mg/l)	CADMIUM (mg/l)	TEST HOURS	#ofLEAD VIOL.	#ofCAD. VIOL.
20-Jul-87	10	11.02	10.64	<0.1	<0.005	24	0	0
25-Aug-87	1	10.2	10.01	<0.1	<0.005	24	0	0
25-Aug-87	2	10.19	10.15	<0.1	<0.005	24	0	0
25-Aug-87	3	9.18	9.32	<0.1	<0.005	24	0	0
25-Aug-87	4	9.28	9.38	<0.1	<0.005	24	0	0
25-Aug-87	5	10.25	9.8	<0.1	<0.005	24	0	0
25-Aug-87	6	10.11	9.71	<0.1	<0.005	24	0	0
25-Aug-87	7	10.03	9.67	<0.1	<0.005	24	0	0
TOTALS							19	0
AVERAGE				4.75	<0.005			
MAXIMUM				15.0	<0.005			

TABLE 4.5V

COMMERCE REFUSE TO ENERGY FACILITY ASH ANALYSIS

California Waste Extraction Test in mg/l
Sample Dates

METALS	LIMIT	3-20-87	3-27-87	4-17-87 (orig.)	4-17-87 (dup.1)	4-17-87 (dup.2)	4-17-87 (avg.)	4-23-87 (orig.)	4-23-87 (dup.1)	4-23-87 (dup.2)	4-23-87 (avg.)	5-1-87	5-28-87
ANTIMONY	15.0	0.2	1.5	1.25	1.25	1.41	1.3	1.0	1.24	1.75	1.33	0.96	0.53
ARSENIC	5.0	0.2	0.9	0.1	0.1	0.087	0.1	0.096	0.1	0.086	0.095	0.066	0.085
BARIUM	100.0	1.9	0.8	1.33	2.73	2.25	2.11	1.34	1.42	1.8	1.52	1.58	0.53
BERYLLIUM	0.75	<0.01	<0.01	<0.004	0.008	0.008	0.0065	<0.003	0.006	0.006	0.006	0.0047	0.004
CADMIUM	1.0	0.5	0.1	0.5	0.54	0.44	0.49	1.17	0.86	0.75	0.93	0.34	0.83
CHROMIUM (IV)	5.0	<0.1	<0.1	NA	NA	NA	NA	NA	NA	NA	NA	<0.024	<0.004
CHROMIUM (III)	560.0	0.6	0.5	0.54	0.8	0.84	0.73	0.38	0.66	0.78	0.61	0.48	0.36
COBALT	80.0	0.2	0.1	<0.042	0.2	0.21	0.15	<0.034	0.065	0.14	0.079	0.07	0.04
COPPER	25.0	<0.1	0.1	<0.042	<0.04	0.11	0.06	<0.034	<0.034	0.1	0.057	0.11	0.27
LEAD	5.0	2.4	<1.5	7.07	14.14	3.39	8.2	12.31	9.25	8.94	10.17	9.87	5.79
MERCURY	0.2	0.0003	0.0002	NA	NA	NA	NA	NA	NA	NA	NA	0.0006	0.0002
MOLYBDENUM	350.0	0.5	0.5	<0.042	0.48	0.52	0.35	<0.034	0.35	0.43	0.27	0.28	0.33
NICKEL	20.0	0.6	0.4	2.7	2.67	2.34	2.57	1.2	1.33	1.44	1.33	1.13	0.84
SELENIUM	1.0	0.001	NA	NA	NA	NA	NA	NA	NA	NA	NA	<0.06	<0.05
SILVER	5.0	<2.0	<2.0	0.32	0.28	0.23	0.28	0.23	0.19	0.22	0.22	0.14	0.12
THALLIUM	7.0	<0.025	<0.01	<0.05	<0.05	<0.05	<0.05	<0.04	<0.04	<0.04	<0.041	<0.026	<0.026
VANADIUM	24.0	0.2	0.2	<0.042	0.44	0.47	0.32	<0.03	0.25	0.31	0.2	0.20	0.15
ZINC	250.0	52.8	42.8	79.87	56.99	64.06	66.98	84.62	79.8	66.34	83.59	40.89	78.59

4.5.4 WASTES FOUND UNACCEPTABLE AT THE FACILITY

4.5.4.1 Unacceptable Waste Defined

Winona County intends to enter into contractual agreements with the other participating counties in order to ensure delivery of a consistent quantity of accepable solid waste to its RRF. In the proposed waste assurance agreement (Winona County, July, 1987), the Proposer has designated a wide variety of waste materials as "unacceptable waste". Table 5.1 lists such unacceptable wastes. In general, items fall into this classification due to factors of size, physical or chemical nature or environmental and public health considerations. Unacceptable materials have a potential for being delivered to the RRF but are not to be accepted for processing. The proposed waste assurance agreement is oriented toward directing participating county's acceptable waste to the RRF and to continue providing proper disposal alternatives for the unacceptable fraction of the waste stream.

Within the proposed agreement, the County defines unacceptable waste as:

"(i) animal waste used as fertilizer, "white goods", demolition debris, earthen fill, construction operations, [sic] sewage, silt, (ii) any material defined as "hazardous wastes" under the Minnesota Statutes, Section 116.06, or the Occupational Safety and Health Act of 1970 (Public Law 91-596), Section 10.6 or any other similar law; and (ii) any material described in Appendix A hereto."

Winona and participating counties are individually responsible for not only non-delivery of unacceptable waste materials but also for providing for a means to cause only acceptable waste to be brought to the facility. If unacceptable materials are delivered to the RRF, the delivering county is to be held responsible. The Proposer reserves the right to dispose of any unacceptable item by whatever means the county may reasonably determine, which will include delivery to a landfill. All cost for disposal will be incurred by the delivering county. It is customary for incineration facility operators to separate waste which are considered unacceptable while the waste is handled on the tipping floor prior to charging into the incinerator. It is expected that the Proposer will follow similar procedures.

4.5.4.2 Quantity Estimates of Unacceptable Waste

County-specific estimates of the quantity of each component of the unacceptable waste stream are difficult to determine given that participating counties have not undertaken comprehensive waste composition studies. Had the counties participated in waste composition studies it is unlikely that the evaluation would have been specific in quantifying the generation of unaccepable items since such investigations are most often centered around combustible properties of the waste stream. Several waste component reports have been prepared on both state and national levels that present average generation rates. Additional information from landfill operators, industry representatives and Proposer county personnel have been used to determine the applicability of the generation rates to this project. (R.W. Beck, 1987)

For planning purposes, the Proposer has utilized a factor of 14% of the total waste stream in order to quantify its aggregate unprocessed waste (Sundberg, 1987). The County anticipates that in 1990, 6643 tons per year (18 TPD) of unprocessible items will be generated from a total waste generation of 47,450 tons per year from Winona, Houston, and Wabasha Counties. No determination is given by the Proposer of the composition of the unacceptable

waste stream or if it includes all or part of the items which are defined in the waste assurance agreement.

The following sections provide discussion of individual unacceptable waste items, estimates of generation and available disposal methods. Many of these items specified are components of the commercial or industrial waste stream. It is reasonable to expect that acceptable waste delivered to the RRF will be made up of only the residential waste stream unless a specific commercial or industrial waste stream is directed to the RRF.

4.5.4.2.1 Tires

Based on a total population of 83,973 and a generation rate of 0.8 tires per person per year, Winona, Wabasha, and Houston County could be expected to generate approximately 67,200 waste tires in passenger tire equivalents, which would equate to roughly 850 tons per year. (MPCA, 1985).

Disposal of tires in a landfill is prohibited in the State of Minnesota due to legislation passed in 1984. Presently, the Proposer has utilized the services of waste tire processors to clean up nuisance accumulation of tires. Municipal solid waste incinerators are able to incinerate certain quantities of waste tires which may prove an alternative disposal option.

4.5.4.2.2 Steel Fencing

The rural nature of Winona County suggests that steel fencing (barbwire, cattle fence, etc.) is likely to be a minor component of the waste stream. Typically, these items are disposed of on the "back forty" or at designated locations at the landfill for metal items. Generation amounts are not available.

4.5.4.2.3 Sludges

The City of Winona Sewage Treatment plant is the only generator of sludge within the county. Approximately 60 tons per week of sludge is generated. The sludge is dewatered and land applied to agricultural crop land according with MPCA guidelines (Larimar, 1987). According to the sewage treatment plant superintendent, there are no intentions of changing disposal practices. All other communities within the county utilize sewage treatment lagoon systems which require no sludge disposal.

4.5.4.2.4 Agricultural Wastes

Agricultual residues from crops or livestock are not generally deposited in landfills. Items such as empty fertilizer containers, if triple rinsed, may be disposed of in sanitary landfills.

4.5.4.2.5 Radioactive Waste

No major generators of radioactive waste are present within the participating counties. Discussions with the Minnesota Health Department indicated that low level radioactive waste if generated by clinics, industry or research institutions within the county must be transported and disposed of in accordance with NRCA Federal Code 10 CFR 20, 30, 40, 61, 70, 71. Presently, Minnesota generators are aligned under the Midwest Compact which utilizes disposal sites in North Carolina and Washington (Tate, 1987).

4.5.4.2.6 Animal Remains

Generators (feed lots, meat processors) of these waste typically utilize rendering companies for disposal. Presently, the rendering market is healthy, offering pick up services and payment for carcasses. Determination of generation is not available (Berlin, 1987).

4.5.4.2.7 Propane Tanks

Although a small component of the waste stream, this item constitutes a potential fire and explosion hazard. Propane tanks can be categorized into two general areas:

1. Disposal/Non-Refillable - These canisters which are typically used by homeowners and in small commercial applications, will be present in the general municipal waste stream. Presently, there are no disposal alternatives or economic incentives for removal of this item from the waste stream.

2. Reusable/Rechargable - These canisters (20 pounds or greater) typically represent some economic incentive to the user and may also have a deposit charge associated with usage. The canisters are considered a durable good having relatively high purchase price and a long utilization period. This in turn discourages indiscriminate disposal and encourages reconditioning of cylinders or disposal through sale for ferrous scrap.

4.5.4.2.8 Mining Waste

There are no mining operations within the County except for general quarrying operations which will generate minimal, if any, waste which would enter the general solid waste stream.

4.5.4.2.9 Street Sweepings

Within Winona County, both municipalities and the County provide street sweeping services. Major components of the waste generated by these services are sand, leaves and seal coating materials. The City of Winona utilizes: (1) the sand materials for fill on city owned industrial properties; (2) the leaves for compost and horticultural needs; and (3) reuses recovered seal coat materials (Giles, 1987). County sweeping activities generally produce sand materials which are used as fill material (Arnibeck, 1987).

4.5.4.2.10 Hospital Waste

Pathological and infectious waste are considered a hazardous waste by both state and federal regulatory bodies. Hospitals are advised to follow guidelines which have been drafted by both MPCA and EPA for the handling of these waste materials and regulated to dispose of these items in accordance with Minnesota Rules Ch. 7045. Infectious hospital waste are not allowed to be landfilled without being autoclaved or incinerated in accordance with regulations (Metz, 1987).

Community Memorial Hospital is the only hospital located within Winona County. It presently disposes of waste items which meet this characterization at an on-site MPCA permitted incinerator. The facility has a permitted capacity of 900 pounds per day which is presently not being totally utilized.

The quantity of waste generated in physicians offices and clinics is not available. If infectious or biological waste are generated at these practices,

they are generally diposed of by:

1) Autoclaving and then landfilling; 2) utilizing a disposal service for infectious waste; and 3) indiscriminate landfilling without regard for guidelines (Olson, 1987).

4.5.4.2.11 Auto Hulks

Though it is estimated that the quantity of vehicles removed from service each year is substantial, a small fraction enters the landfill or municipal waste stream. There are no figures available to determine the number of vehicles per year which are "junked" within the County. Generally, one could assume that for every new car licensed, one vehicle would be retired. (Franklin, 1986) Estimates on new car registrations are not available on a County basis. Most retired vehicles are typically disposed in "salvage yards" which dismantle autos for parts or metal recovery.

4.5.4.2.12 Plastics in Large Quantities

An informal telephone survey of firms located in Winona County which utilize or produce plastic materials in their process, indicated that most recycle plastic waste unless the material is contaminated. Generally, some plastics do go to the landfill but usually only in small quantities. One of the three firms interviewed indicated disposal of between 40-80 tons of waste per year in the landfill with the greater percentage being a plastic fiberglass composit material. (Clark, 1987; Klinger, 1987; Barger, 1987)

4.5.4.2.13 Liquid Waste

MPCA Rule 7035.1700, requires that liquid wastes not to be disposed of in a landfill. If the liquids' composition is non-hazardous, generators are encouraged to first recycle and as a last measure to dispose in a sanitary sewer. If the material is considered hazardous, the liquid must be disposed of in accordance with MPCA Hazardous Waste Rules 7001, 7045 and 7046. No applicable data is available to determine generation rates for the County. It is reasonable to expect that small quantities will be part of the municipal waste stream.

4.5.4.2.14 Used Engine Oil

A recent study (Minnesota Waste Management Board, 1987) estimates that approximately the following quantities of waste oil are generated within the participating counties:

Winona - 286,320 gallons
Houston - 95,440 gallons
Wabasha - 95,440 gallons

Collection and recycling services for large quantity generators (service stations, industry) do exist. It is estimated that 82% of the used oil generated in the state is sold for fuel purposes to asphalt production plants or recycled or used on the site of the generator. Oil generated by "do-it-yourselfer" oil changers, (estimated at appproximately 10% of the generation) continues to be disposed of in the waste stream in two forms (1) containerized liquid; and (2) more recently in absorbed form due to the introduction of the disposal oil drain pan.

4.5.4.2.15 Construction and Demolition Rubble

Up until recently two privately owned and operated demolition landfills existed in the City of Winona area. With the closure of these landfills all materials were diverted to the County-owned landfill. The County has recently received approval to locate a designated landfill cell for demolition materials in conjunction with a landfill expansion project. No data specific to the County is available to determine generated amounts. Rates of generation vary greatly and are specific to each generation area. Utilizing a national average of 0.72 pounds per capita per day the following estimated generations could be anticipated. (Franklin, 1986).

	Population	TPD
Winona	46,256	16
Wabasha	19,335	6.96
Houston	18,382	6.61
Fillmore	21,936	7.89

4.5.4.2.16 Large Metal Bulky Items

Bulky metal items (appliances, engine blocks, steel post, bicycles, etc.) do enter the waste stream and are typically separated at the landfill for future transportation to markets or processors. Large items are delivered to a facility by either self-haul methods or by haulers making separate trips to keep the items segregated from the general waste flow. No estimates to the generation of these wastes are available.

4.5.5 NON-HAZARDOUS INDUSTRIAL WASTE

Presently, industrial non-hazardous waste is not regulated by MPCA in a comprehensive form. MPCA has drafted for adoption in late 1988 rules specifically addressing the management of industrial waste which are to be delivered to waste management facilities. (Draft rule 7035.2535 subpart 5). The draft rules would require the waste management facility operator to submit an industrial waste management plan. The plan would require the facility owner to discuss how all industrial waste received at his facility will be managed. The plan is to include how facility operators will interact with the generator, waste stream characteristics, procedures for management and inspection and address specific categories of waste and their disposal. This plan is to be reviewed and approved by the MPCA.

Presently, ash from the Community Memorial Hospital waste incinerator is being deposited into the County landfill. Ash produced by power generation facilities is disposed of in designated landfills outside the County. No quantity amounts are presently available for these facilities.

Volume and storage records indicate that approximately 5,842 tons of industrial waste were transported by two haulers who service the majority of the industrial waste stream in Winona County. Based on 365 days of generation, approximately 16 TPD of industrial waste are generated. Presently, this industrial waste stream is being landfilled and will continue to be landfilled unless a specific item within the waste stream is designated to be delivered to the proposed RRF. The Proposer has no readily available information on the composition of this waste stream. A study of waste entering the Red Wing Facility (Table 4.1) may provide insight into the industrial waste stream composition since the service area contain both demographic and regional similarities (MPCA, 1980).

The effect of non-hazardous industrial wastes delivered to the RRF on the air and residue ash emissions of the RRF is similar to that discussed under Section 2 for household hazardous wastes. It is expected that the primary impact of incinerating non-hazardous industrial waste at the RRF is the presence of trace metals such as lead, cadmium and mercury in the residue ash waste stream. These contaminants could be present in air emissions from the facility although levels are expected to be low due to the high efficiency of the particulate collection equipment. It is possible, however, that specific types of wastes may contain high amounts of trace contaminants which may result in increased levels of contaminants in ash or in higher air emissions. It is also possible that industrial wastes of certain kinds may contain precursors to toxicants, such as chlorine. It therefore appears to be appropriate to carefully review the types of industrial wastes accepted at the facility.

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4.5.6. SOLID WASTE PLANNING IN WINONA COUNTY

The requirements for solid waste management planning in Minnesota have evolved from the early 1970's when the original solid waste regulations were established during a time when landfilling was the primary form of disposal of solid waste. Since that time a number of changes have occurred in the planning process which now requires the examination of a number of alternatives to landfilling of solid waste including waste reduction, recycling, yard waste composting, waste to energy, and municipal solid waste (MSW) composting. (RCM, 1987)

Due to increasing concerns regarding the safety of sanitary landfills, Winona County entered into the solid waste planning process in 1983 when it began work with three other counties on a multi-county solid waste management plan. Although the plan was never formally completed and adopted, the results of the study have been implemented in Winona County. (RCM, 1987)

The County plan considered a variety of alternatives to landfilling of the County's solid waste. The final recommendation was for the construction of a solid waste incinerator facility to burn solid waste from Winona and, possibly, neighboring counties. Other recommendations were for the development of recycling and yard waste composting facilities in addition to the incinerator. (RCM, 1987)

The County began implementation of the recommendations of the plan by establishing a county Task Force to review the recommendations and oversee implementation. The Task Force was divided into several subcommittees related to the various areas of interest: recycling, public education, waste to energy. In each area, the subcommittees solicited additional studies and made recommendations for the Task Force and County Board to follow. (RCM, 1987)

The Source Separation/Recycling Subcommittee worked on the establishment of a combination of waste abatement programs including curbside collection in the city of Winona and other cities in the County, yard waste composting drop sites, and multi-dwelling recycling programs. Although the participation rate has been less than anticipated, the County has continued the expansion of these programs over the years and has begun working with neighboring counties to establish recycling programs in their communities. (RCM, 1987)

The Public Education Subcommittee began work on the establishment of a information network to inform residents of the County about solid waste issues. This work resulted in the establishment of a speakers bureau, a series of articles in local newspapers, other media contacts and public meetings. The Public Education Subcommittee was combined with the Source Separation/Recycling Subcommittee in 1986 to work on implementation of the waste abatement program. (RCM, 1987)

The Waste to Energy Subcommittee recommended Black and Veatch be retained to study, in detail, potential waste to energy alternatives. Their study, completed in 1985, evaluated several landfill alternatives, incineration with steam sales to a local industry, a refuse derived fuel (RDF) facility in the County with haul of the RDF to NSP plants in either Red Wing, Minnesota or LaCrosse, Wisconsin, or transportation of solid waste to the RDF plant proposed for LaCrosse. The final recommendation was for construction of an incinerator facility in Winona with steam sales to a local industry (the market was later changed to electric sales when negotiations with the proposed market for steam sales poved unproductive). Following acceptance of the Black and Veatch report, the County proceeded into the design phase. (RCM, 1987)

Upon completion of the work of the subcommittees, the County began the implementation of the Task Force recommendations. These programs are still in effect today and have been expanded and revised periodically to increase the amount of waste handled through the programs. (RCM, 1987)

4.6 FIRE AND EMERGENCY SERVICES

In a telephone interview with Minnesota Pollution Control Agency (MPCA) Staff, the Assistant Fire Chief of the City of Winona indicated that the Department possessed equipment and staffing adequate to respond to fire and emergency needs at the proposed incinerator. (Yackel, 1988)

Winona is served by two fire stations. The central fire station is located at 421 East Third Street and is roughly equidistant from the two sites. The other station is located at 301 Cummings. One ladder company (one truck) and one engine company (one engine) are stationed at the central fire station, and a single engine company of one engine is stationed at 301 Cummings Street. The Department has 37 staff members. The Department's firefighting capability includes the use of foam and aqueous film forming foam (light water).

Fire and emergency response are coordinated by the City/County law enforcement center, which has access to statewide emergency services. Fire companies from both stations will routinely respond to fire calls. Maximum response time from the central fire station to either the Riverview Drive site or the Riverbend II site is estimated to be 3 minutes. Response time from the Cummings Street station to the Riverview Drive site would be less than three minutes, and to the Riverbend II site would be somewhat greater.

All personnel are, at a minimum, first responder trained, and some are trained emergency medical technicians. Fire response vehicles are equiped with radios which will facilitate contact with ambulance, hospitol and statewide emergency services.

REFERENCE FOR SECTION 4.6 FIRE AND EMERGENCY SERVICES

Yackel, Henry, City of Winona Assistant Fire Chief, May 25, 1988.

4.7 FUTURE REGULATORY CLIMATE

A discussion pertaining to the future regulations and procedural requirements which might apply to incinerator ash may be found in Section 4.5.3.1. Briefly, that discussion included the observation that the requlatory milieu applicable to incinerator ash was quite dynamic. It appears, however, that disposal of ash in land disposal facilities will likely continue to become more expensive, simply because more stringent requirements are likely to apply to ash disposal, whether it is considered to be hazardous waste or special waste.

Future developments in the area of air emission regulations are similarly difficult to predict. However, it appears likely that regulatory requirements will continue a trend established over the last several years towards more stringent controls. Over the life of an operating facility, this may translate into increased costs as requirements for new equipment or more stringent operating requirements apply.

V. SOCIAL AND ECONOMIC EFFECTS OF THE PROPOSED PROJECT

5.1 EFFECTS ON PROPERTY VALUES

The proposed facility is not expected to have a significant impact on nearby property values. At both sites, the area is zoned M-2, for heavy industrial use, and the proposed incinerator is a permitted land use. The building itself will not be particularly conspiuous in its appearance, and no visible or otherwise notable emissions will be associated with it. A slight traffic increase may result.

5.2 EFFECT ON EMPLOYMENT

The proposed facility will result in a small increase in employment opportunities during its construction and operation. Information as to the number of such employees was not readily available. However, given the relatively large population in Winona, it is unlikely that the facility will result in any notable economic impacts.

VI. ALTERNATIVES

This chapter of the EIS is a review of alternatives to the proposed solid waste incinerator facility. The alternatives are reviewed on the basis of cost and the potential environmental impacts. The environmental impacts of the incinerator facility proposed by Winona County have been discussed in Section 4.0.

6.1 REVIEW OF SOLID WASTE MANAGEMENT ALTERNATIVES

Solid Waste Management Alternatives

The following alternatives are described below:

- Continued use of the existing disposal system
- Landfilling with programs to maximize the amount of recycling and yard waste composting
- Refuse derived fuel system
- Municipal solid waste composting with programs to maximize the amount of recycling and yard waste composting

6.1.1 Continued use of existing solid waste management system

Currently, Winona County generates about 90 tpd of solid waste. According to monthly landfill records, this amount can vary from approximately 67 tpd to 114 tpd due to seasonal variations in the generation rate. The 1985 Black and Veatch study estimated a seasonal range of 70% to 130% of the average generation rate, which would result in an annual variation from 67 to 124 tpd of solid waste (7 day week basis). (RCM, 1987)

Wabasha and Houston Counties are estimated to generate approximately 4000 to 10,000 tons per year of solid waste (11 to 27 tpd). This volume compares to an estimated 56 tpd (of which 41 tpd is available for the incinerator) presented in the Multi-County Solid Waste Management Plan and 18 tpd used in the Black and Veatch study. If an assumed average of 8000 tons per year is used, approximately 22 tpd is available in the two counties, of which Houston County generates approximately 80% (18 tpd) and Wabasha County generates 20% (4 tpd). (RCM, 1987)

In addition to the solid waste generated in Winona, Wabasha, and Houston Counties, Winona County has agreed to allow Fillmore County to utilize their landfill for the disposal of excess waste and residuals from Fillmore County's MSW composting facility. This quantity of waste is estimated to be between 1100 to 4000 tons per year (3 to 11 tpd), depending on the volume of residuals screened out at the plant. (RCM, 1987)

The four counties are in the process of siting a new regional landfill. A new landfill facility will be necessary whether the proposed incinerator facility is constructed or not. The existing Winona County Landfill has approximately 5 years of remaining life at current generation levels. For the analysis, it was assumed that the existing landfill would continue in operation for its remaining life and that a new facility, when necessary, would be located adjacent to the existing site. (RCM, 1987)

The County currently operates a curbside recycling program in the City of Winona and other cities in the County through a contract with the Occupational Rehabilitation Center (ORC). The ORC provides the collection and processing of the material through a sheltered workshop. ORC retains all revenues from their

marketing of the recycables in addition to a payment of $37,000 per year from the County. The contract has some escalator clauses if participation increases above existing levels. In addition, the County provides publicity and some administrative services for the program. In its second year of operation, the program recycled approximately 1 percent of the solid waste stream (420 tons of glass, newsprint, tin and aluminum). In addition to the curbside program, several redemption centers and private programs are in operation in the County which collect an unknown quantity of waste. (RCM, 1987)

The County has also established several yard waste composting sites in the County. These sites are operated by their respective municipalities at no cost to the County. The County provides some publicity and administrative services. The City of Winona operates the only staffed composting site in the County. The City's records indicate about 4400 users visited the site in one year, depositing approximately 440 tons of yard waste, or about 1 percent of the waste stream. (RCM, 1987)

The existing landfill facility is owned by Winona County. The site was purchased by the county from a private owner in 1983 and consists of two phases. As part of the rehabilitation of the site after county purchase of the facility, a perimeter collection system was installed in the Phase I area to collect leachate at the site. Leachate collected is piped to the City of Winona's wastewater treatment system. Phase I of the landfill has approximately 188,000 cubic yards, or 2.5 years, of remaining capacity. Phase II, currently under design by the county, will last approximately 3.5 years and handle 227,000 cubic yards of solid waste with construction in an area adjacent to Phase I. (RCM, 1987)

Following completion of filling operations in Phase II, a new site will need to be established by the county. Winona County, along with Wabasha, Houston, and Fillmore Counties, is in the process of siting this new facility. One potential site under consideration is located adjacent to the existing site and subject to the same site design constraints as Phase II of the existing facility. Since the facility is located in an area of karst formations, the new site (and Phase II) will be required to have an artificial membrane liner and a backup liner system of 2 feet of clay. A complete leachate collection system will be required in the facility design with the collected leachate pumped or hauled to the city of Winona's wastewater treatment system. For this analysis, it was assumed that the site adjacent to the existing facility would be utilized. In addition, in approximately five years, if landfilling remains the primary disposal method utilized in the county, solid waste from Wabasha and Houston Counties would be landfilled with Winona's and Fillmore's solid waste in a new joint landfill facility. (RCM, 1987)

6.1.2 Landfill with Recycling

The second alternative under consideration is the development of a landfill in the manner discussed above but with attempts to increase the amount of material that is recycled and composted. Landfill development would occur in a manner similar to that described under Alternative 1. The primary differences in the programs would be in the areas of recycling and yard waste composting. (RCM, 1987)

The following list represents some items which may increase the degree of reduction of the waste stream. Each item is discussed as to its potential cost and anticipated impact on the waste stream. (RCM, 1987)

- mandatory source separation law

- ban on yard waste at disposal facilities
- economic incentives for residential participation
- economic incentives for businesses
- collection bins for recyclables
- twice a month pickup of recyclables
- increased number of materials handled
- increased local advertising
- curbside collection of yard waste

Mandatory recycling laws are in effect in a number of communities in other states. The city of St. Cloud and Fillmore County in Minnesota have mandatory recycling ordinances. Based on data from other sources, mandatory separation laws have historically increased participation rates to 50 to 60 percent from average levels of 20 to 30 percent. Strict enforcement is a key component of the effectiveness of mandatory separation laws. In Winona County, collection services are provided primarily by commercial haulers who would be required to carry most of the burden of enforcement of the ordinance in the county by denying service or reporting noncompliance to the county. Since people would still be free to use other options to recycle their materials, the county may not see a dramatic increase in the use of their program, although some increase is likely. Unless some system is arranged to estimate the amount of recyclables handled by redemption centers and other programs, the overall effectiveness of this type of change or its impact on the waste stream can not be estimated. The estimated cost involved with developing and passing such an ordinance is small, however, it may be necessary to expand the curbside collection program to handle the increased participation and to provide for enforcement of the ordinance, each resulting in increased costs. The City of Winona is currently considering a mandatory recycling ordinance. (RCM, 1987)

A ban on yard waste at the landfill is another method to reduce the volume of waste sent to a landfill. It is currently the policy of the County Board to discourage yard waste from going to the landfill but there is not an outright ban. This option is similar to a mandatory separation ordinance in that it puts a great deal of the enforcement onto the haulers, a burden they have not agreed to accept. At this time few records are maintained on the volume of yard waste received at the yard waste sites. The City of Winona, which operates the only staffed site in the county, estimates they had 4400 visits to the site involving an approximately 440 tons of yard waste in 1986. A ban on yard waste at the landfill would certainly increase this volume but the degree of increase is unclear as other alternatives will continue to exist for the disposal of the material such as on-site composting, nongeneration of the material by residents, and promiscuous dumping on vacant lots or in wooded areas. Overall, it will be difficult to accurately assess the degree of reduction in the waste stream due to the implementation of a ban. Again, the initial cost is small but long term costs may include the need to develop additional yard waste composting sites and provide enforcement action against those not in compliance with the ordinance. (RCM, 1987)

Economic incentives to participation in recycling and yard waste programs have been tried in other communities and have generally resulted in short term increases in the participation rates in the programs. Whether these increases will represent a long term change in behavior is unknown. (RCM, 1987)

Recycling bins have been used in some communities to promote awareness of recycling programs with, potentially, an increase in participation. (RCM, 1987)

Twice a month, or more frequent, collection, to increase the convenience and availability of service in the area could potentially increase the overall

participation rate. (RCM, 1987)

Another potential method of increasing the amount of the waste stream affected by a recycling program is an increase in the number of materials handled. Currently the program recycles aluminum and tin cans, plastic jugs, glass, newspaper and brown paper bags. Other materials that have been successfully recycled in the state and elsewhere include corrugated cardboard, mixed paper, phone books, used oil and car batteries (now that they can no longer go to the landfill). Removing these materials and others from the waste stream may have a significant impact on the overall waste volume sent to the landfill. The other side of this option is the increased costs and need to find new markets for the materials handled. (RCM, 1987)

From discussions with the operator of the current recycling program, an increase in the amount of direct household advertising of the recycling program is suggested. (RCM, 1987)

Curbside collection of yard waste is another means of increasing participation in a yard waste compost program although depending on the method used for collection, the program can be expensive. (RCM, 1987)

The impact of implementing any of the above options in terms of reducing the volume of material landfilled, the cost of landfilling or the size of a landfill, is small on an annual basis. Most cost and landfill capacity savings will be recognized over the life of the facility. For example, the County's current waste abatement programs annually removes approximately 900 tons of recyclables and yard waste from the waste stream. This quantity results in an annual landfill savings of approximately 0.03 acres. Over twenty years the savings would be 0.6 acres. The cost savings in operational cost, due to the reduced volume of waste, are not measurable on an annual basis but would accrue over the life of the facility. Likewise, construction cost savings will be minor on an annual basis but more significant when considered over the life of the site. (RCM, 1987)

6.1.3 Refuse Derived Fuel Option

A method of recovering energy from waste is to produce a fuel from solid waste that can be burned in a conventional solid fuel fired boiler. The starting material, raw municipal waste is composed of a combustibles fraction, a glass fraction, a metals fraction and a miscellaneous materials fraction. (RCM, 1987)

A refused derived fuel (RDF) processing plant separates the combustibles fraction from the other fractions through the use of an air classifier, a trummel, and hand sorting. (RCM, 1987)

Shredding or "size reduction" is an essential step in the preparation of RDF. While a variety of unit processes are available to accomplish size reduction, hammermills are the type most widely used. (RCM, 1987)

For purpose of evaluation of a RDF technology, the Buhler-Miag technology has been selected. This technology has been selected because it best allows for the use of an integrated system approach to the overall problem of solid waste management. This RDF technology can be integrated with a composting plant by sending the fines fraction from the trommel process to composting. Further, the process equipment can be arranged such that hand sorting of glass, corrugated, plastic and aluminum can be accomplished. (RCM, 1987)

6.1.4 MSW Composting

Composting of municipal solid waste can cause a significant reduction in volume of the organic fraction of a waste and can allow reuse of the material. As discussed in a previous section, MSW composting facilities were considered briefly and rejected as an alternative in the county's solid waste management plan prepared in 1984. The primary reason that the alternative was not given further consideration was due to a lack of a verifiable market for the compost in the study area. In addition, the system costs were high and few systems were in existence in the United States at the time, especially in the size range contemplated for Winona County. A cost estimate was prepared for the system but the recommendation was not to proceed at that time with a composting facility. (RCM, 1987)

In the time since the preparation of the original management plan, the concept of MSW composting has gained greater acceptance in the United States and in Minnesota. A number of facilities are in operation or contemplated for construction over the next few years. The flexibility of the processing in a composting plant has great appeal in that it allows for the development of an integrated management system incorporating a number of components. For this reason, MSW composting is being evaluated along with maximized recycling, in this Environmental Impact Statement. (RCM, 1987)

Composting is a biological process used to partially decompose the organic materials in solid waste and convert it to a-humus like material. The decomposition may take place aerobically or anaerobically, but is normally carried out aerobically to avoid odors. The bacterial activity produces a dark, humus-like material characterized by a low nutrient value and high moisture retention. The nutrient value of solid waste compost can be supplemented by co-composting the waste with sludge from a wastewater treatment facility or with animal manure. However, many systems are designed to be self-supporting and rely only on the addition of water to the composting MSW. (RCM, 1987)

Most composting operations consist of three steps: waste separation, decomposition and product processing. Each step may be very simple or complex depending on the size of the waste stream, the objectives of the operation and the equipment utilized. (RCM, 1987)

In a minimum processing operation, large bulky items such as tires and appliances are separated from the organic wastes and the remainder of the waste is shredded, mixed with water or other additives, depending on the system, and composted, usually by windrowing or in static piles. (RCM, 1987)

Following the composting (decomposition) period, the compost is allowed to cure and dry for a period of time. The finished product may require grinding, blending and mechanical screening to remove metals, glass or other impurities from the finished product. (RCM, 1987)

With larger waste streams, more complicated equipment may be incorporated into the system to increase the removal of impurities, allow for removal of recyclables, or to increase the value of the end product. These systems involve more intense capital expenditures and have higher operation and maintenance costs so an assured market is desirable to offset some of the system costs. At this time, potential compost markets generally express some interest in the purchase of the compost, pending the outcome of tests on the compost quality. It is likely that as more plants are developed in the state, markets can be developed, but currently most system economics are based on a no

market alternative assuming that the producer can dispose of the compost at no additional cost but no revenue is generated. (RCM, 1987)

Large scale systems generally have a tipping floor where non-compostable materials are removed. From there the system varies with the type of equipment selected. Some systems are similar to RDF systems in the screening, shredding and mixing steps of the waste stream to produce a material suitable for the decomposition phase. In the decomposition phase, the raw compost is placed in windrows or static piles for approximately six weeks or more. A second type of system uses a rotating drum operation to break down the waste and provide screening and mixing of the waste without the necessity for shredding and its associated risks. Following the drum operation, which may last from hours to days, the raw compost is placed in windrows or static piles for the decomposition process. Subsequent to the decomposition phase for either process train, the material is cured, dried, screened, ground or otherwise processed to prepare it for the proposed market. Other digestion processes have been tried involving large reactor systems. These systems are usually very capital intensive and expensive to operate. For this reason, we will focus attention on the previously discussed systems. (RCM, 1987)

For preparation of this EIS, it was proposed to evaluate an MSW composting system along with maximized recycling and source separation. The Buhler-Miag facility can be designed to accommodate handpicking of recyclables from the waste for recycling. This system, along with continued operation of the county's source separation program, could potentially maximize the removal of material from the waste stream through recycling of resources. The DANO line operates with a drum system for preprocessing of the waste followed by static pile composting to complete the decomposition process. (RCM, 1987)

6.2 ENVIRONMENTAL CONSIDERATIONS

This section discusses the known and unknown environmental impacts of the disposal alternatives. The discussion of environmental considerations includes sanitary landfilling, energy recovery, composting, and recycling. (RCM, 1987)

The different alternatives under review in this EIS were evaluated as to monetary costs and environmental considerations. Monetary costs included capital costs and operation and maintenance costs. These costs were converted to net present worth and equivalent annual cost for comparative purposes. (RCM, 1987)

Each technology, including RDF, composting, landfilling and recycling, was also evaluated as to its potential environmental impact. Potential impacts related to worker safety, ground and surface water impacts, air quality, aesthetics and other factors were considered. (RCM, 1987)

It should be borne in mind when considering any solid waste system that there are a variety of risks associated with all systems, due to the nature of the material that is being handled. Even those systems involving recycling and yard waste composting may represent a risk to workers and the environment if improperly handled or if hazardous materials are mixed into the waste stream. What is placed into the waste stream will end up in some form in the byproducts of any disposal system. Many systems have the ability to breakdown some components of the solid waste but no system can handle all potential types of waste. Sorting out the hazardous or undesirable materials before they reach the facility, whether it is an incinerator, composting plant or recycling facility, is the best way to help control the potential impacts of these

materials and even then a separate disposal system, with its associated risks, must be developed to dispose of these materials. (RCM, 1987)

Factors discussed under environmental considerations include impacts on surface water quality, groundwater quality, air quality (point and non-point sources), noise, aesthetics, public health, and land use. The potential development of other nuisance conditions also should be considered. (RCM, 1987)

6.2.1 Environmental Effects Associated With Sanitary Landfills

Sanitary landfilling has for many years been the most common means of ultimate disposal for solid waste in Minnesota and most of the United States. In Minnesota, landfills became prevalent in the early 1970's as an alternative to the numerous small open dumps present throughout the state. At that time it was thought that a sanitary landfill system would eliminate many of the problems found with open dumps. (RCM, 1987) In the years since the use of sanitary landfills became popular, a lot more information has become known about their impact on the environment, and new control methods have been developed to control some of the risks associated with these types of facilities. This section reviews the known and currently unknown risks associated with landfill facilities. (RCM, 1987)

The risks associated with landfills have been well documented over the years and in numerous sources. The risks which are summarized here can be divided into the following areas: worker safety, air and water pollution, and nuisance conditions. The risks presented are general in nature since risks are site specific. When Winona County sites a new landfill facility, a separate Environmental Impact Statement/Environmental Assessment Worksheet will be prepared. (RCM, 1987)

6.2.1.1 Worker Safety

Worker safety is a major concern in any facility dealing with solid waste. Because of the many unknowns in the constituents of the waste stream, workers are potentially exposed to a wide variety of hazardous chemicals on a regular basis. Household hazardous wastes are routinely disposed of through the solid waste system and end up at the landfill. In addition, the decomposition of solid waste generates more byproducts, some of a hazardous nature. Landfill workers are exposed to these byproducts through several trains: direct exposure when working on covering or sorting of solid waste and through indirect exposure such as off gases from the landfill and leachate collection system. (RCM, 1987)

The direct exposure route involves daily operations of the site during which time the workers may be called on to handle materials received at the site through the collection system or to move items manually due to the size of the item. Many times the materials or items are not identified and the risks the worker faces may be unknown. Also, repeated exposure to the materials at the site may work to reduce the caution a worker takes in approaching an unknown substance. (RCM, 1987)

Indirect exposure of the worker to risks at the site relates to some of the chemical reactions taking place during the decomposition process. During the decomposition of solid waste, leachate is produced along with methane and other gases. While it is known that these gases contain some volatile organics which are known carcinogens, the impact on the individual worker has not been quantified as to level of exposure or health risk involved. Because the worker

is in a well ventilated environment, the risk may be somewhat alleviated, however the long term effect of repeated exposure is unknown. In addition, the leachate produced contains a number of heavy metals and volatile organics. Gases are routinely emitted from leachate collection system manholes and tanks which are potentially harmful to workers in the area. Methane is one gas emitted in this manner which results in an explosion potential near these facilities. Care must be taken when approaching work on any parts of the leachate collection system to ensure proper ventilation is maintained. (RCM, 1987)

6.2.1.2 Water and Air Pollution

Two of the largest concerns related to the landfilling of solid waste is the potential for air, ground water and surface water contamination. As more landfill sites are studied through Minnesota, the more is known about the potential of these sites, if improperly designed and/or operated, to contaminate an area's water and air resources. (RCM, 1987)

6.2.1.2.1 Surface Waters

Surface waters near a landfill are usually impacted in one of several ways: the collection of blowing papers in the stream or lake creating a nuisance condition for those who use the water; erosion and runoff of silty water into a body of water which clogs or pollutes the waterway; direct runoff from the landfill by leachate or other polluted water which carries contaminants in to the water; and groundwater supplies feeding into the surface water carrying contamination. Of greatest concern to surface water supplies is the addition of organics and nutrients to the water system which can increase the eutrophic rate and the clogging of waterways. VOC's will dissipate from the water in the presence of oxygen and metals will settle into bottom sludge deposits. (RCM, 1987)

6.2.1.2.2 Groundwater

Groundwater supplies near a landfill, especially in Minnesota where the majority of residents rely on groundwater as a primary source of drinking water, are particularly vulnerable to contamination. Leachate, containing heavy metals and volatile organics, routinely leaks from landfill sites into area groundwater supplies. New design features on landfills are designed to eliminate, as much as possible, the amount of leachate that reaches the groundwater but it is generally accepted that no system will eliminate all risks. Groundwater supplies are of particular concern in that the ability to control the spread of contaminated groundwater or to cleanup in situ pollution is limited. (RCM, 1987)

6.2.1.2.3 Air Pollution

Air pollution risks from a landfill vary in the degree of risk from the emission of volatile organics and methane through the leachate collection system to the spread of dust in the environment due to daily site operations (RCM, 1987).

6.2.1.2.3 Carcenogenic Risk from Water and Air Pollution

Carcinogenic risk, as summarized on table 6.2A, was evaluated for inhalation of toxic air pollutants from two hypothetical municipal landfill alternatives and two active municipal landfills. The two active landfills employ gas control devices. In the assessment of carcinogenic risk from the

hypothetical landfills assumed that no gas controls would be used. Carcinogenic risk for the two hypothetical landfills ranged from 0.1 to 43 additional cancer cases in a population of 100,000 people. The carcinogenic risk for the two working landfills ranged from less than 0.1 to 1.7 additional cancer cases in a population of 100,000 people. The assessments summarized in table 1 tend to confirm expectation that landfill gas mitigation technology can significantly reduce gas emissions and the toxic pollutants contained in the gas. (Minott, 1988)

Carcinogenic risk was assessed for eleven planned solid waste energy recovery facilities in the United States. Refer to table 6.2B for a summary of the results. The exposure pathways evaluated and the pollutants which were considered vary. All of the facilities employ flue gas scrubbers and either electrostatic precipitators or fabric filters. Carcinogenic risk ranged from 0.03 to 1 additional cancer cases in a population of 100,000 people. None of the facilities evaluated show a carcinogenic risk above the Minnesota Department of Health guidline for tolerable risk of 1 additional cancer case predicted in a population of 100,000 people. (Minott, 1988)

Table 6.2C contains a summary of carcinogenic risk associated with three MSW landfills in Minnesota. The carcinogenic risk from the Pine Bend Sanitary Landfill was quantified using contaminant concentrations in well water. Since residents with affected wells have been provided with an alternative source of drinking water, no one is now exposed to the carcinogenic risk reported. (PBL, 1987) The first value reported for the Anoka Regional Sanitary Landfill (ARSL) represents the total carcinogenic risk. The second value reported for the ARSL is the carcinogenic risk for ingestion of ground water. (FVD, 1988) Since none of the residential wells in the vicinity has been effected by the ground water contamination and perimeter monitoring wells continue to be sampled for contaminant migration, no one is exposed to the risk from the ingestion of ground water contaminated by the landfill. (Sarappo, 1988) Therefore, the carcinogenic risk that the public is exposed to would be the difference between these numbers, which is 1 in a population of 100,000. The carcinogenic risks reported for the Waste Disposal Engineering Sanitary Landfill assume that no mitigative actions will be taken. (CRA, 1986) The measures which are planned to mitgate the carcinogenic risks posed by the WDE Sanitary Landfill are anticipated to be completed in 1990. (Haberman, 1988)

6.2.1.3 Nuisance Conditions

A variety of nuisance conditions can develop from the improper operation of a landfill facility. Blowing paper is one area of major concern especially to neighbors of the site as it collects in their yards and farm fields. Of more particular concern is the presence of vectors (rats, birds, flies) due to incomplete covering operations of the solid waste. These vectors can migrate from the site onto neighboring property causing problems to other areas. Birds can present problems at landfills, especially if located near an airport. Large flocks of birds are also disruptive and messy when nesting in trees on neighboring property. Dust and odor are other complaints that can be lodged against landfills although given the usual isolation of the sites, these problems are generally not realized off site if proper site operation is maintained. Again, proper operation and regular covering of the solid waste can eliminate many of these problems, or minimize their impact to the landfill site. Noise from site operations can be a problem but controlling site operations to times of day when other farm equipment is in use makes this problem less noticeable. (RCM, 1987)

6.2.2 Recycling

Source separation involves the separation of recyclable materials from the main solid waste stream at their source by the waste generator. The separated materials are then transported by collection vehicle or the generator to a recycling center for additional processing and resale or reuse. Source separation is one method of recycling materials from the waste stream. Other recycling programs use human or mechanical means to separate the recyclables from the waste stream at the processing center. Recycling programs of this kind, with separation following collection, is more commonly associated with RDF and MSW composting facilities and can potentially impact a larger portion of the waste stream, depending on the processes used in the separation of the recyclables. One potential problem with processing after collection is the potential contamination of the recyclables with other materials. Source separation, which is the more common method of recycling currently in operation in Minnesota, has less potential for contamination of the recyclables, however may have a significantly smaller recovery rate due to the greater effort that is required by the homeowner. (RCM, 1987)

Generally, recycling programs present minimal risk to the environment and have the beneficial effect of reducing the volume of waste in the system which must be handled through other more risky methods which may pose a greater risk. The impact of each system is related to the type of program that is proposed and the materials being collected. Obviously, the collection and recycling of household hazardous waste presents greater risks than a paper/glass/metal processing facility (without weighing the relative merits of either type of facility). Risks associated with recycling can generally be controlled by the proper training and supervision of workers. (RCM, 1987)

For this discussion, types of risk have been divided into the following areas: worker safety, nuisance conditions, and water and land pollution.

6.2.2.1 Worker Safety

Worker safety and associated health risk implications, are major areas of concern associated with recycling programs. The degree of risk generally varies with the material in the waste stream. With the presence of household hazardous waste in the system, the potential increases for either collection or processing workers to be exposed and/or injured by these hazardous elements in the waste. This topic was discussed in greater detail in the section on composting and readers are referred to that section for more information. In addition, improper handling of materials by workers can result in injuries. Glass breakers, can flattening equipment and balers can be dangerous if handled improperly. Dust exposure to workers is a potential problem depending on the materials processed and the equipment used. Proper training and supervision of all workers can help minimize these risks. (RCM, 1987)

6.2.2.2 Nuisance Conditions

Nuisance conditions such as odor, litter and vectors can develop with recycling programs if they are not properly managed. If collections are not made in a timely manner, litter can become a problem along collection routes. Materials at the processing center should be stored inside to prevent blowing papers or litter in the area of the center. Odors should not be a problem if the materials are properly cleaned prior to collection. Non recyclables received at the plant, especially those of a putrescible nature, should be disposed of promptly to prevent odor or vector problems from developing. Proper maintenance of the facility and regular marketing of the materials will limit the potential for the formation of vectors at the site. Site traffic can be an annoyance to facility neighbors depending on site location and hours of

operation. (RCM, 1987)

6.2.2.3 Water and Land Pollution

Water and land pollution should also present little risk with a properly managed site. Litter should be collected and disposed of properly to prevent its collection in local streams and storm sewer systems. Stockpiled materials should be marketed regularly to prevent the formation of leachate from those stockpiles maintained outside. Hazardous materials received at the site should be disposed of properly and not dumped on the ground or to the sewer system. Likewise, spills of liquid materials (waste oil, etc.) received at the site should be cleaned up properly to prevent groundwater contamination. (RCM, 1987)

6.2.3 Environmental Affects Associated with RDF Facilities

The environmental effects are discussed below for both RDF processing facilities and for the burning of RDF.

6.2.3.1 Environmental Effects Associated With RDF Processing Facilities

The environmental effects associated with RDF processing facilities can be divided into four general areas: air discharges, water discharges, solid waste disposal and worker safety. While the RDF is not combusted at a RDF facility, certain air pollution control devices are part of the processing facilities. In particular, where air classification is utilized, it is necessary to install fabric filter baghouses to remove entrained particles from the air stream prior to discharge to the environment. (RCM, 1987)

Modern RDF plants also utilize proper ventilation within the enclosed areas to minimize dust concentrations in the plant. This air is usually filtered prior to discharge by fabric filters. (RCM, 1987)

Fabric filters are very high efficiency devices for the particle size range encountered in this application and there should be a minimal impact on air quality near the processing facility. (RCM, 1987)

There is no water utilized in the processing of RDF except for some indirect cooling in some cases. The only wastewater discharge would be from sanitary facilities and would typically be directed into a municipal wastewater treatment system. (RCM, 1987)

The facility will reject approximately 50 tons per day of the 150 tons per day processed. This material will consist primarily of white goods, organics, glass and yard wastes, which must be disposed of in a sanitary landfill. The organics could be directed to a composting plant if one were available. Provided that the sanitary landfill is permitted and properly designed, the types of materials rejected from the processing facility are compatible with this type of disposal and should have a minimal impact on the environment. (RCM, 1987)

Worker safety is a concern at all industrial plants. A RDF facility must provide an environment that protects against such dangers as electrical shocks, falls, tripping and injuries associated with coming in contact with moving parts. Of particular concern to RDF facilities are protection from explosions, high dust exposures and high noise exposure. (RCM, 1987)

The presence of flammable and explosive materials in the solid waste stream is a reality that must be accepted by operators of RDF facilities. The potential for explosions to occur in a RDF facility is real. The unit processes most commonly considered candidates for an explosion are the size reduction equipment such as shredders and ballmills; dust hoods; air conveying lines and storage silos. (RCM, 1987)

To date, most of the interest in explosion protection has been focused on the shredders. The ASTM Committee E-38 on Resource Recovery has published "Guidelines for Shredder Explosions Protection" which lists a number of approaches to explosion protection. However, no single approach is 100 percent safe and it is necessary to imploy a combination of approaches to minimize the potential for explosion. (RCM, 1987)

Dusts are generated at a RDF facility and these dusts may contain both biological and chemical substances of health significance. Sampling programs at resource recovery plants equipped with modern dust control systems have shown that most of the dust is fiberous organic materials. Chemical analyses of the dusts have shown nothing alarming. Microbiological analyses of the dusts have indicated the presence of airborne bacteria and fungi. However, there have been no reports of any outbreaks of disease, or any particular health problems among resource recovery workers. (RCM, 1987)

Various surveys of noise within resource recovery plants indicate that there are locations within the plant that exceed the OSHA 8 hour exposure of 90 dBA. These areas tend to be close to the shredding processes where noise levels can exceed 110 dBA. However, workers in the plant do not typically remain in the shredder processing areas for any length of time due to the risk of explosions. While it is difficult to retrofit an existing plant with noise reduction equipment, new plants can be designed to meet all OSHA standards related to noise. (RCM, 1987)

The issues associated with the processing of solid waste are similar to those discussed for composting, presented elsewhere in this section.

6.2.3.2 Environmental Affects Associated with the Combustion of RDF

The environmental affects associated with the combustion of RDF can be divided into four areas: air emissions, wastewater discharges, solid waste disposal and worker safety. (RCM, 1987)

The air emissions from the combustion of RDF are not well known. While it would be expected that the emissions from RDF combustion would be similar to a waste to energy plant, little definitive information is available. The Minnesota Pollution Control Agency is currently preparing to conduct measurements of emissions from the combustion of RDF at the NSP Red Wing and Mankato stations, however these measurements have not yet been made. The results of tests conducted by the MPCA on the combustion of d-RDF in the boiler plant at Northwest Medical Center indicated lower than expected emission rates of chlorinated dioxins and furans but higher than acceptable particulate emissions. (RCM, 1987)

Currently, the MPCA has taken the position that further combustion of d-RDF must be accompanied by the use of high efficiency particulate removal devices such as fabric filter baghouses or electrostatic precipitators. (RCM, 1987)

Both the NSP Red Wing and NSP Mankato plants are equipped with particulate

control devices so that it is not proper to draw conclusions from the d-RDF tests and apply them to the combustion of coarse RDF at the NSP plants. Further conclusions concerning the air emissions from the combustion of RDF will be available after the MPCA conducts the proposed emission testing. (RCM, 1987)

The wastewater discharges from a power plant combusting RDF consist of boiler blowdown, sanitary facilities and water used to convey bottom ash if a wet system is utilized. Boiler blowdown and sanitary wastewater discharges are typically directed to a wastewater treatment system. Wet ash conveying systems typically are closed loop and should not result in a wastewater discharge. (RCM, 1987)

Ash from the combustion of RDF is expected to be similar in chemical composition to the ash from a waste to energy plant. The issue of ash disposal from waste to energy facilities is receiving more attention because of the collection of heavy metals in both the fly and bottom ash. Toxic metals and metallic compounds enter the incinerator and are not destroyed in the combustion process. They leave the incinerator in the bottom ash, fly ash or flue gases. Metals and metallic compounds in the flue gases enter the scrubber/fabric filter system as solids, liquids and vapors. (RCM, 1987) Ash disposal is discussed at length in Section 4.5.3. of this document.

Safety issues associated with the combustion of RDF in a power plant are similar to the issues previously discussed. In addition to the RDF storage and handling issues, the proper operation of the boiler system is a consideration. It is expected that the combustion of RDF will most likely occur in a boiler specifically designed and operated for that purpose and staffed with operators familiar with boiler operation. For this reason, no additional safety issues are addressed. (RCM, 1987)

6.2.4 Composting Systems

Many questions are still unanswered regarding environmental impacts of MSW composting, but research has been conducted toward finding answers. This section is a review of published information related to the known and suspected risks associated with composting municipal solid waste. The risks are addressed in the three categories of worker safety, site consideration, and the compost end product. (RCM, 1987)

6.2.4.1 Worker Safety

During the shredding process there is a continual potential for hazard with respect to explosive materials entering the shredder. There is little control over what can enter the municipal solid waste (MSW) stream, and prior separation efforts may not be 100 percent successful, therefore a real risk to workers is present in the form of explosions and flying debris or equipment. (RCM, 1987)

Workers are exposed to further hazard with respect to pathogens present in the raw MSW. These pathogens may enter the body through either the oral or respiratory tract. The degree of the risk is not definite as few studies have addressed this topic at the present time. (RCM, 1987)

Dust particles, household hazardous waste, and know carcinogenic volatile organic compounds present in the air may be inhaled to increase the risk of cancer or other diseases. (RCM, 1987)

Extended exposure to the high decibel noise associated with shredding

equipment is a further health risk that must be dealt with through proper ear protection and strict operating rules. Risks associated with noise pollution can be greatly reduced when these items are properly dealt with. (RCM, 1987)

In addition to these risks are the risks common to the operation of all heavy machinery. To reduce these risks to a level commensurate with other industries requires skilled and knowledgeable workers who have been trained to fit into a program that stresses safety. (RCM, 1987)

The separation of compostable from noncompostable waste may be done at the beginning or the end of the composting process. If a shredding or recycling step is to be incorporated in the system, separation is usually at the beginning of the process. (RCM, 1987)

Separation of the waste involves the closest physical contact of workers with the waste. Because of this the potential exists for cuts, abrasions and contamination from pathogens, dust, or other hazardous materials. Gloves, face masks, and strict safety rules will decrease the risks of manually separating waste, but the potential for injury is still present. Only testing and time will prove the degree of risk posed by each of these potential hazards. (RCM, 1987)

Decomposition and curing are the steps in which the majority of the microbial action takes place. As such, a large number of pathogens are present in the waste. In addition to the pathogens, dust, household hazardous wastes, volatile organic carbons and glass fragments are still present in the waste. (RCM, 1987)

Although physical contact with the waste is not as great as during the separation step, the potential still exists for workers to receive these hazardous materials through tactile, oral or respiratory routes. (RCM, 1987)

6.2.4.2 Site Considerations

A compost facility may cause detrimental effects to the area immediately surrounding it. Many of the concerns will be very similar to any solid waste facility, such as traffic, noise, and wind-blown debris, but other concerns will be more specific to the compost process. Careful design and planning will limit the extent of any adverse impacts both on the site and around it. (RCM, 1987)

There is a large uncertainty in predicting the quality of surface runoff from a compost facility. If the windrows or static piles are covered from precipitation, the runoff concerns will be minimized. If the piles are not covered, it can be expected that the composting material will absorb a significant amount of water before releasing any to runoff. (RCM, 1987)

The constituents of the runoff from the composting material may contain many items of concern in compost. At a minimum, nutrients, bacteria and intermediate products of decomposition, such as organic acids, can be expected. Other constituents may also contaminate runoff depending on their occurrence in the waste and their availability to be extracted from the composting material. These may include metals, toxic organics, pathogens, and floating debris. (RCM, 1987)

The runoff from a compost facility should be captured for monitoring and possible treatment. If treatment is necessary, it may consist of transferring the water to a treatment facility or stabilization by natural biological

processes in an onsite holding pond. Recirculation of the runoff through the plant is also potentially feasible. Monitoring and further research are needed to determine the need for treatment and the most appropriate method of treatment, if necessary. (RCM, 1987)

Facility design will have a major impact in controlling surface water pollution. The most important feature of the design will be to divert water away from the composting pile or windrow so that the material is neither standing in water nor in the flow path of runoff. The capacity of the holding pond and outlet structure must be chosen to allow desired treatment of the expected flow. No recommendations have been made to date relating to the treatment of surface runoff from composting facilities. The usual procedure is to collect any runoff and reroute it back through the plant. (RCM, 1987)

Ground water pollution may be caused by improper management of surface water runoff. The two possible routes for water to enter the ground are the pile or windrow area and the surface water collection and holding system. Proper facility design should include impervious surfaces for the management of all runoff water. Maintenance should also be stressed to repair points where surface water may leach into the ground water. (RCM, 1987)

Airborne particulates, volatile organic compounds and pathogens are all concerns in the area of a compositing facility. The risks associated with each of these in relation to composting are unknown at the present time, however the degree and length of exposure will no doubt play an important role in determining the degree of risk. (RCM, 1987)

6.2.4.3 Compost End Product

The significance of certain contaminants in the compost end product will depend on the ultimate use of the material. Stringent requirements should be placed on any compost that will come in close contact with humans, either by use in the production of food, or in direct handling of the compost as a soil conditioner. Less stringent requirements may be appropriate where the product will have minimal contact with humans, as in landfill cover or land reclamation. The following list shows some possible uses of compost products. (RCM, 1987)

SOIL CONDITIONER

Private

- flower beds
- vegetable gardens
- potting soil
- landscaping

Recreational

- parks
- golf courses
- landscaping

Public - Limited Contact

- highway right-of-ways

Commercial

- agricultural
  - food (direct consumption by humans)
  - food (indirect consumption by humans, e.g. animal feed)
  - non-food (sod, etc.)
- nursery and greenhouse
  - starter flowers
  - starter food crops
  - shrubs and trees
  - house plants
- cut flower growth media

LAND RECLAMATION

Landfill cover
Strip mine fill
Sand and gravel pit fill

In general, there are four areas of concern associated with compost products, including aesthetics, stability, metals, and toxic organics.

6.2.4.3.1 Aesthetics

With the exception of glass fragments, the items that cause aesthetic concern in a compost product will not cause physical harm to the users of the material. Aesthetics, however, will have a strong impact on the marketability of the product. The main aesthetic concerns are particle size and the presence of foreign material (e.g. glass, plastics, metal pieces, wood, etc.). (RCM, 1987)

Aesthetics of the compost are controlled by the design of the facility, specifically in the choice of the separation, shredding, and screening methods used in the pretreatment, the post-treatment or both. More stringent control of aesthetics will increase the capital and operation costs. (RCM, 1987)

6.2.4.3.2 Stability

There have been complaints in the past that compost products have hurt crop production rather than helped. Most researchers believe that these complaints result from the use of immature compost products. Mature compost will have an insignificant amount of the phytotoxic components. A number of studies have been conducted to determine the components of immature compost that are phytotoxic (toxic to plants). Studies have also been conducted to develop testing methods for determining compost maturity. (RCM, 1987)

A quantitative, reliable definition of mature compost has not been established to date, but many testing methods have been evaluated. The simplest method of predicting compost maturity is time. MPCA proposed rules (July, 1987) set 6 months as a minimum decomposition time.

In summary, unstable compost can have a very detrimental effect on plant growth, but this risk is minimized in properly stabilized material. A standard test for stability has not yet been established, but a few proposed methods seem to provide a reliable indication of maturity. (RCM, 1987)

6.2.4.3.3 Metals

Certain metals that may occur in the compost product warrant concern in soil conditioning because they are potentially toxic to plants or to animals and humans that eat the plants. The composting process has little affect on metals. (RCM, 1987)

Toxicity to plants will be exhibited by poor germination, stunted growth, or reduced yield (Loehr, et. al. 1979). In general, phytotoxic effect from metals will be much less significant than that caused by immature compost (Wong, 1985).

Metals have a variety of toxic effects in humans ranging from irritability and nausea to death in extreme cases (Sax, 1979). Acute toxicity will be readily apparent due to high concentrations of some metals. Chronic toxicity may also be caused by some metals if ingested at low concentrations over a long period of time. (RCM, 1987)

Not all metals that are placed in the soil by compost will be moved from the soil into edible portions of a food crop. A large amount of research has been conducted to examine plant uptake of metals from sewage sludge or sewage sludge compost. Some research has also been related to municipal solid waste compost. There is some variation in the conclusions made from the studies, but a few important points can be made. First, different species of plants and different varieties within each species may extract different amounts of metals from a soil (Loehr, et. al. 1979). Second, each part of the plant (i.e. roots, stem, leaves, fruit, etc.) will accumulate different amounts of metals (Loehr et. al. 1979; Chu and Wong, 1984; Dyer and Razvi, 1987). In general, the edible fruit or grain accumulated less than the leaves, stalks and roots. Third, the increased pH and cation exchange capacity caused by the compost product may serve to reduce the availability of metals to plants by reducing metal solubility and by holding the metal ions (Dyer and Razvi 1987). Fourth, the metal extracted from the soil by plants in the first growing season is a small fraction of the total metal content of the soil media (Petruzzelli, et. al., 1985; Dyer and Razvi 1987; Chu and Wong, 1984).

Metal toxicity should cause less concern in compost uses that do not directly affect humans. In cases like highway right-of-ways and land reclamation the greater concern should be leaching of the metals into groundwater. (RCM, 1987)

Leaching of metals into groundwater will be possible any time a MSW compost is incorporated into a soil or used as fill material. Each situation will have a different level of susceptibility to metals contamination. Determining the risk level in each application will involve a number of questions. First, what metals are expected in the compost? Some metals of concern for phytotoxicity are not harmful to humans (eg. copper, iron, manganese, zinc). MPCA rules will regulate important metals, and that regulation should minimize groundwater pollution to a great extent. Second, what is the distance to groundwater and what type of soil is below the compost amended soil? The cation exchange capacity of the amended soil and the underlying soil will serve to adsorb the metals and limit the amount passing to the groundwater. One contributing factor in limiting problems with leachate is that metals (except for cadmium) in the compost product are held in forms that are not readily extracted by water (Petruzzelli, 1985).

The quantity of metals in a MSW compost may vary considerably. Metals in the raw material will not be affected by the biological process of composting except for possibly complexing with organic matter. Pre- and post-processing may remove a significant quantity of toxic metals. For example, magnetic separation will not only remove iron, but also the cadmium, zinc, chromium, and other metals that may be plating on the iron or steel. Ballistic separation may also reduce toxic metals. Source separation may have the greatest affect on metals reduction, but some toxic metals may be in forms not readily noticed by the general public. (RCM, 1987)

All of the metals of concern in composting naturally occur in all soils at some level. The concern in composting, as in all solid waste handling systems, is to control metals additions to acceptable levels. (RCM, 1987)

6.2.4.3.4 Toxic Organics

The large volume and wide variety of organic compounds used in the United States causes concern in composting of municipal solid waste. Toxic organics appear in cleaners, cosmetics, disinfectants, pesticides, medical supplies, paints, preservatives and many more products used daily by the general public.

It is inevitable that some of these compounds will enter a composting system. (RCM, 1987)

It is difficult to predict the effect of the composting process on all organics due to the wide variety of compounds , but much research has been conducted to determine the degradability of specific compounds. Volatile compounds will be removed from the compost pile or windrow by the aeration process to some extent, but each compound varies in its volatility. (RCM, 1987)

Some organics decompose readily while others are more persistent. If time was not a concern, almost all organics, hazardous or not, could be biologically degraded. Unfortunately, some organics would take an unfeasibly long time to degrade (Savage, et. al. 1985a).

6.2.4.3.5 Nutrients

Nutrients are a concern in compost if their concentration is too high or too low. If the concentration is too low, supplemental nutrients must be added to provide what is necessary for good plant growth. Over-application of compost can cause leachate problems just as over-application of any fertilizer can cause leaching of excess nutrients (Dyer and Razvi, 1987; Golueke, 1976). Careful agricultural practices will minimize the problems related to both lacking and excessive nutrient concentrations. (RCM, 1987)

6.2.4.3.6 Pathogens

The end product of municipal solid waste (MSW) composting is a "stabilized" material which can no longer be considered a present or future nuisance. (RCM, 1987)

Although pathogens may be present in the final compost product, the compost material may still be considered "stabilized" when considering the final point of application or disposal. (RCM, 1987)

Recent papers have demonstrated the growing concern that pathogenic microorganisms may be present in the final compost product by trying to isolate and enumerate specific pathogens. (RCM, 1987)

The complex nature of compost and co-compost with respect to the action and interaction of a multitude of various organisms makes it very difficult to conduct studies on compost. It is unknown if all the variables associated with various composting studies have been recognized let alone identified and understood. (RCM, 1987)

Earlier studies and papers have indicated a more complete destruction of pathogens during high temperature composting processes, however recent work suggests this may not be so. (RCM, 1987)

Early studies that dealt with pathogen destruction often implied total pathogen destruction based solely on temperature reached within the composting mass. This may not always be true beacause all particles in the mass may not be at the same temperature. In addition, temperatures previously established as lethal to certain microorganisms may not always be accurate. (RCM, 1987)

6.2.4.4 Conclusions on Composting Risks

As in any solid waste disposal method, composting of MSW is not without

Table 6.2A

ESTIMATED HEALTH RISKS DUE TO INHALATION OF TOXIC AIR POLLUTANTS FROM MUNICIPAL LANDFILLS

Landfill Location	Status	Gas Control	Pollutants Considered	Carcinogenic Risk (Chances per Million)	Reference
San Diego, CA (2,250 TPD)	Hypothetical Landfill(1)	None	Benzene, 8 other organics; vinyl chloride not studied	430	32
Montgomery Co., MD (1,800 TPD)	Hypothetical Landfill(1)	None	Vinyl chloride, benzene, other organics	1-50(2)	33,34
Sylmar, CA (unk. TPD)	Active	Flare	Vinyl chloride, other organics	<1	35
Simi Valley, CA (est. 600 TPD)	Active	Engine	Vinyl chloride, other organics	17	36

(1) Risks for a hypothetical landfill were assessed as the "alternative" to a proposed energy-recovery facility.

(2) The range is indicative of analytical uncertainty.

Table 6.2B

RECENT HEALTH RISK ASSESSMENTS PREPARED FOR PLANNED ENERGY RECOVERY FACILITIES

Facility	Setting	Flue-Gas Controls	Exposure Pathways	Pollutants Considered	Carcinogenic Risk* (Chances per Million)	Data Reference
Lancaster Co., PA (1200 TPD)	Rural	Scrubber, Baghouse	Inhalation, Soil Ingestion, Food Chain, Dermal Contact	Dioxins, Furans Other Organics, Trace Metals	4	21
N. Hempstead, NY (990 TPD)	Urban	Scrubber, Baghouse	Inhalation, Soil Ingestion, Food Chain, Dermal Contact	Dioxins, Furans Other Organics, Trace Metals	1	22
Erie, PA (RDF-710 TPD)	Urban	Baghouse, Furnace Scrubber	Inhalation	Dioxins, Furans	0.3	23
York Co., PA (1344 TPD)	Rural Industrial	Scrubber, Baghouse	Inhalation, Soil Ingestion, Food Chain, Dermal Contact	Dioxins, Furans, Other Organics, Trace Metals	5	24
Brooklyn, NY (3000 TPD)	Urban	Scrubber, Baghouse	Inhalation, Soil Ingestion, Food Chain, Dermal Contact	Dioxins, Furans, Other Organics, Trace Metals	2	25
Washington Co., NY (400 TPD)	Rural	Scrubber, ESP	Inhalation, Soil Ingestion, Food Chain, Dermal Contact	Dioxins, Furans	10	26

* Upper-bound estimate of the number of cancer cases per million people exposed, based on continuous exposure to maximum impacts 24 hours per day, everyday, for a 70-year lifetime.

Table 6.2B continued

RECENT HEALTH RISK ASSESSMENTS PREPARED FOR PLANNED ENERGY RECOVERY FACILITIES

Facility	Setting	Flue-Gas Controls	Exposure Pathways	Pollutants Considered	Carcinogenic Risk* (Chances per Million)	Data Reference
Boston, MA (1516 TPD)	Urban	Scrubber, ESP	Inhalation, Soil Ingestion, Dermal Contact	Dioxins, Furans Other Organics, Trace Metals	2	27
Philadelphia, PA (2250 TPD)	Urban	Scrubber, Baghouse	Inhalation, Soil Ingestion, Food Chain, Dermal Contact	Dioxins, Furans Other Organics, Trace Metals	1	28
Stanislaus Co., CA (500 TPD)	Urban & Rural	Scrubber, Baghouse	Inhalation Soil Ingestion, Food Chain, Dermal Contact	Dioxins, Furans Other Organics Trace Metals	2	29
San Marcos, CA (1600 TPD)	Urban	Scrubber, Baghouse	Inhalation	Dioxins, Furans Other Organics,. Trace Metals	3	30
Irwindale, CA (3000 TPD)	Urban	Scrubber, Baghouse	Inhalation, Soil Ingestion, Food Chain, Dermal Contact	Dioxins, Furans Other Organics Trace Metals	5	31

Landfill Location	Pollutants Considered	Pathways of Exposure Considered	Potential Carcinogenic Risk (chances per 100,000)	Reference
Pine Bend Sanitary Landfill	Aromatics, halogenated and non-halogenated hydrocarbons	Ingestion: soil, surface water, and ground water	1) 0 to 495 2) 0 to 0.27	(PBL 1987)
Anoka Regional Sanitary Landfill	Halogenated hydrocarbons, ethers, and aromatics	Ingestion: vegitables, soil, ground water Inhalation: vapors from soil, flaring vents, and showering Dermal contact: soil	3) 2,100 4) 2,099	(FVD 1988)
Waste Disposal Engineering Sanitary Landfill	Volitile organic compounds, aromatics, and heavy metals	Ingestion: soil, surface water, ground water, aquatic organisms, exposed wastes and leachate, and lime sludge Inhalation: vapors, soil Dermal contact: vapors, soil, surface water, stream sediment, lime sludge, exposed wastes, and exposed leachate	5) 0.86 6) 0.028 7) 0.06 8) 0.78 9) 0.013 10) 0.082	(CRA 1986)

1) Range of carcinogenic risk from ingestion of ground water from onsite monitoring wells.

2) Carcinogenic risk for ingestion of ground water from affected residential wells. Actually residents with water supplies affected by ground water contamination have been provided an alternative drinking water supply.

3) Carcinogenic risk from all pathways considered.

4) Carcinogenic risk from ingestion of ground water assumed to contain the mean concentraition of contaminants found on site.

5) Carcinogenic risk to a hypothetical residential population from ingestion of ground water from wells outside of the landfill boundry. The carcinogenic risk is derived from the concentraition of the contaminant methylene chloride, which is present in a monitoring well at the perimeter immediatly up gradiant of the hypothetical residential wells.

6) Carcinogenic risk from dermal exposure from wading in Coon Creek, which is a stream contaminated by ground water discharge from the landfill.

7) Concentrations of contaminants in Coon Creek and the Mississippi River were compared to the Water Quality Criteria for the Protection of Human Health in the Federal Register Vol. 45, No. 231, November 28, 1980. Coon Creek contaminant concentrations were compared to the criteria water concentrations for cancer risk for fish consumption.

8) Since Coon Creek is a tributary to the Mississippi River, present contaminant levels from the landfill for the Mississippi were calculated and compared to the criteria water concentration levels for cancer risk for consumption of water and fish. The criteria water concentration levels were previously mentioned in footnote 7.

9) Future concentrations were modeled for Coon Creek and compared to the criteria water concentrations for cancer risk for fish consumption. The criteria water concentration levels previously mentioned in footnote 7.

10) Future concentrations from the landfill's contamination of the Mississippi River via Coon Creek were compared to the criteria water concentration levels mentioned in footnote 7 for cancer risk for ingestion of water and fish.

References: Final Remedial Investigation of Pine Bend Sanitary Landfill, Inver Grove Heights, Minnesota, Harding Lawson Associates, Sunde Engineering, Inc., and Pace Laboratories, Inc., November 1987.

Feasibility Study, Detailed analysis Report for the Anoka Regional Sanitary Landfill, Volume I, Foth & Van Dyke January 1988.

Detailed Analysis Report, Waste Disposal Engineering Sanitary Landfill, Conestoga-Rovers & Associates October 1986.

Table 6.2C

risk. There is a range of risks that must be considered before, during, and after implementation of a system. The process of composting MSW is relatively new in Minnesota and in the United States, but it has been established in other parts of the world. This precedent serves to indicate that composting is a reasonable approach to waste management as long as the potential risks are properly addressed. (RCM, 1987)

6.3 Cost Basis Evaluation of Alternatives

The following discussion summarizes the monetary costs associated with each alternative evaluated for this study. These costs do not include the cost of existing waste abatement programs as these costs are assumed to be equivalent for all systems. Costs are averaged over a twenty year facility life assuming an average interest rate of 8 percent and an inflation rate of 5 percent. These costs were developed by the MPCA's consultant, Rieke, Carroll, Muller Assoiciates (RCM) as part of the technical work paper TWP on solid waste management planning and alternatives. (RCM, 1987) The TWP is available at the offices of the MPCA.

However, monetary costs represent only one element of the "total cost" picture. The environmental risks associated with the alternatives evaluated in the previous section reflect hidden costs that may not be realized until a later date.

TABLE 6.3A

COMPARATIVE COSTS OF SOLID WASTE MANAGEMENT ALTERNATIVES

	Existing Landfilling System	Refuse Derived Fuel	MSW Composting (Buhler-Miag)	MSW Composting (Dano)
Average Equivalent Annual Cost	$1,430,429	$3,519,758	$3,284,578	$2,781,973
Net Present Worth	$13,157,528	$23,196,557	$29,249,716	$25,229,123
Estimated Average Cost per Household Per Year	$88	$132	$124	$105
Estimated Average Cost per Household Per Month	$7.30	$11	$10.30	$8.70
Estimated Average Cost per Capita Per Year	$30	$47	$43	$37
Estimated Average Cost per Capita Per Month	$2.50	$3.90	$3.60	$3.10
Estimated Average Tipping Fee	$39	$87	$82	$69

Source: RCM (1987)

The above summary presents only the cost for landfilling under the existing system. In the text a discussion was provided describing several potential methods for increasing the volume of materials that are recycled or composted. These suggestions are based on methods that have been sucessful elsewhere. It is difficult to estimate the actual impact each option or combination would have on the waste stream and the resulting cost, or cost savings, in regards to the landfill. To accurately assess the impact of each option it would be necessary to get more information on the volume of waste that is recycled through other private systems in the County and how these systems would be impacted by the different options.

It is not unreasonable to achieve a 15 percent reduction in the waste stream through an intensive waste abatement program. In the County, this size of reduction would result in an annual savings in landfill capacity of approximately 0.2 acres, or about 3.2 acres over 20 years. This reduction compares to 0.03 acres per year, or 0.6 acres over 20 years, with the current level of abatement. On an annual basis it would be difficult to calculate the actual savings in operation and maintenance costs or in construction cost. These savings are likely to become apparent when considering life cycle costs of the system. Therefore, operational costs for the landfill system with programs to maximize recycling and yard waste composting, within the accuracy of this estimate, would be similar to those for the existing landfill system.

During preparation of this Draft Environmental Impact Statement (DEIS), it was determined that a comparison of the above costs to those associated with the proposed alternative would be useful. MPCA staff prepared cost estimates which were considered roughly equivalent to the calculations performed by RCM, (McCarron, 1988) utilizing net annual cost data provided by Springsted, Inc., the County's financial consultant (Sundberg, 1988). The input data from Springsted appears in the Net Required (column 8) of Table 6.3B. MPCA staff assumed a 5% inflation per year in disposal costs, and assumed constant population and household size.

Costs associated with ash disposal were revised as follows. The volume of MSW processed was derived from Column 3 of Table 6.3C. It was assumed that incoming processed MSW would be reduced to roughly 35% of its original weight, including entrained moisture. Ten %of the ash was assumed to be fly ash and the remaining 90% was assumed to be bottom ash. Ash disposal costs were calculated for three scenarios; Fly ash disposed of as hazardous waste at $160 per ton and bottom ash disposed of at $40 per ton, All ash disposed of at $160 per ton, All ash disposed of at $40 per ton. The tonnage costs to represent a reasonable estimate of the range of disposal costs which might result from various ash possible disposal requirements. The cost of disposal of unprocessable waste is not estimated in the calculations. Table 6.3D presents the results of the calculations.

Table 6.3B

CASH FLOW PROJECTION: Source: Sundberg, (1988)

YEAR (1)	DISPOSAL COSTS (a) (2)	O & M EXPENSES (3)	NET DEBT PAYABLE(b) (4)	REPLACEMENT NET DEBT PAYABLE (5)	TOTAL EXPENSES (6)	LESS ELECTRIC REVENUE(c) (7)	NET REQUIRED (8)	TOTAL TIPPING FEE/TON (d) (9)
1990	443,384	800,000	1,777,200	0	3,020,584	380,308	2,640,276	55.64
1991	470,206	840,000	1,775,400	0	3,085,606	396,359	2,689,247	56.11
1992	498,596	882,000	1,775,800	0	3,156,396	408,766	2,747,630	56.77
1993	528,655	926,100	1,778,000	0	3,232,755	436,332	2,796,423	57.22
1994	560,505	972,405	1,776,600	0	3,309,510	440,388	2,869,122	58.14
1995	594,185	1,021,025	1,776,600	0	3,391,810	470,394	2,921,416	58.63
1996	629,865	1,072,076	1,777,600	0	3,479,541	472,788	3,006,753	59.78
1997	667,594	1,125,680	1,774,200	0	3,567,474	428,415	3,139,059	61.82
1998	707,512	1,181,964	1,776,400	0	3,665,876	439,603	3,226,273	62.95
1999	749,762	1,241,062	1,773,400	0	3,764,224	451,246	3,312,978	64.05
2000	794,509	1,303,115	1,775,200	0	3,872,824	463,361	3,409,463	65.32
2001	841,812	1,368,271	1,776,000	0	3,986,083	475,965	3,510,118	66.64
2002	891,906	1,436,685	1,775,400	0	4,103,991	489,080	3,614,911	68.01
2003	944,856	1,508,519	1,268,000	507,000	4,228,375	502,725	3,725,650	69.48
2004	1,000,862	1,583,945	1,268,800	506,200	4,359,807	516,920	3,842,887	71.03
2005	1,075,615	1,663,142	1,269,800	505,200	4,513,757	528,958	3,984,799	73.02
2006	1,155,805	1,746,299	1,270,600	504,400	4,677,104	541,405	4,135,699	75.13
2007	1,241,324	1,833,614	1,270,800	504,200	4,849,938	554,276	4,295,662	77.36
2008	1,332,506	1,925,295	1,270,000	505,000	5,032,801	567,584	4,465,217	79.74
2009	1,429,703	2,021,560	1,252,800	522,200	5,226,263	581,345	4,644,918	82.25
2010	1,533,289	2,122,638	0	1,775,000	5,430,927	595,573	4,835,354	84.91
2011	1,643,659	2,228,770	0	1,775,000	5,647,429	610,285	5,037,144	87.72
2012	1,761,232	2,340,209	0	1,775,000	5,876,441	625,498	5,250,943	90.69
2013	1,886,454	2,457,219	0	1,775,000	6,118,673	641,228	5,477,445	93.83
2014	2,019,794	2,580,080	0	1,775,000	6,374,874	657,492	5,717,382	97.15
2015	2,161,753	2,709,084	0	1,775,000	6,645,837	674,310	5,971,527	100.66
			31,958,600	14,204,200				

NOTES:
(a) Transportation and landfilling costs for ash and unprocessed waste.
(b) See bond computations.
(c) Based on B&V projected electric production and NSP's January 16, 1987 proposed rate schedule.
(d) Tipping fee calculations based on tons available to facility (Column 2 on Waste Flow projections).

Table 6.3C

WASTE FLOW PROJECTION (TONS/YEAR): Source: Sundberg (1988)

YEAR (1)	WASTE AVAILABLE (2)	WASTE PROCESSED (3)	WASTE UNPROCESSED (4)	WET ASH (5)	ASH LANDFILLED (6)
1990	47,450	40,807	6,643	17,547	8,774
1991	47,925	41,216	6,709	17,723	8,862
1992	48,400	41,624	6,776	17,898	8,949
1993	48,875	42,033	6,842	18,074	9,037
1994	49,350	42,441	6,909	18,250	9,125
1995	49,825	42,850	6,975	18,426	9,213
1996	50,300	43,258	7,042	18,601	9,301
1997	50,775	43,667	7,108	18,777	9,389
1998	51,250	44,075	7,175	18,952	9,476
1999	51,725	44,484	7,241	19,128	9,564
2000	52,200	44,892	7,308	19,304	9,652
2001	52,675	45,301	7,374	19,479	9,740
2002	53,150	45,709	7,441	19,655	9,828
2003	53,625	46,118	7,507	19,831	9,916
2004	54,100	46,526	7,574	20,006	10,003
2005	54,575	46,538	8,037	20,011	10,006
2006	55,050	46,538	8,512	20,011	10,006
2007	55,525	46,538	8,987	20,011	10,006
2008	56,000	46,538	9,462	20,011	10,006
2009	56,475	46,538	9,937	20,011	10,006
2010	56,950	46,538	10,412	20,011	10,006
2011	57,425	46,538	10,887	20,011	10,006
2012	57,900	46,538	11,362	20,011	10,006
2013	58,375	46,538	11,837	20,011	10,006
2014	58,850	46,538	12,312	20,011	10,006
2015	59,325	46,538	12,787	20,011	10,006
TOTALS	1,388,075	1,166,919	221,156	501,772	250,895

TABLE 6.3D

COMPARATIVE ANNUAL HOUSEHOLD COSTS OF THE PROPOSED INCINERATOR, GIVEN VARIOUS ASH DISPOSAL COSTS

	Ash Disposal Costs		
	$40/ton	Fly Ash $160/ton Bottom ash $40/ton	$160/ton
Annual			
Cost/Household	$104	$106	$169
Year 1	$183	$143	$370
Year 20	$138	$193	$256

Source: McCarron, 1988

Estimated Tipping fee			
Year 1	$68	$70	$112
Year 20	$105	$127	$244
20-year Average	$82	$94	$169

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