APPENDICES

1.0 – Early Site History

2.0 – Detailed Labor Job Descriptions

3.0 – Leach Pad Chemistry Trends

4.0 – Identification of Applicable or Relevant and Appropriate Requirements (ARARs)

5.0 – Toxicity Profile Summaries

1.0 EARLY SITE HISTORY

1.1 Pre Open Pit Era - 1883-1977

Fort Belknap was established in 1869 near the present town of Chinook, Montana. The new fort served as a trading post and became the government agency for the Gros Ventre and Assiniboine Indians living in the area. Later that year, the agency moved from Chinook to its present location 5 miles east of Harlem. Prospectors, Frank Aldrich, Dutch Lewis, Pike Landusky, and Charlie Brown made the first discovery of placer gold along Alder Gulch on July 3, 1884. The news of the discovery quickly spread when Landusky traveled to Lewistown to establish his claim. Within a month, 2,000 miners stampeded into the area, establishing a mining district and a recorder to register their claims (Burlingame and Toole, 1957). Because this area was within the area that Congress had allocated to the Northern Tribes in 1874 (Acts of the 43rd Congress-First Session, 1874), federal troops were sent to investigate and to keep order at the peak of the rush. By the time the Agreement of January 21, 1887 settled a new boundary and a May 1, 1888 act of Congress established the Fort Belknap Indian Reservation, the placers in Ruby Gulch and Alder Gulch had played out and only a few diehard prospectors remained.

Pike Landusky later staked the first lode claims in the Little Rocky Mountains, locating the Julia in 1887 and the Gold Bug in 1890. Then he and Robert Orman found a high-grade ore body in the August Mine, which was within the then boundaries of the Fort Belknap Indian Reservation. Because of the increased mining activity in the Little Rocky Mountains, Montana politicians influenced the establishment of a commission to identify the mineral lands and to negotiate concessions of these lands from the Native Americans. The result was the Grinnell Agreement of 1896, in which the Native Americans at Fort Belknap ceded a portion of the Little Rocky Mountains from the southern part of the reservation to the U.S. government for $360,000.[1]

Pike Landusky built a three stamp mill, which employed amalgamation rather ineffectively to process the ore from his mine. Although Landusky was killed in a celebrated gunfight with Kid Curry in 1894, his heirs along with C. L. Manning proceeded to develop the Gold Bug. By the mid-1890's, the Gold Bug had a 40 feet deep shaft leading to three tunnels. In the lowest tunnel, a winze dropped another 50 feet. Due to mill recovery problems, the Gold Bug was sold to a North Dakota group composed of Jacobson, Torgenson and Nobin. They constructed and expanded the mill with amalgamation plates in 1902. After a short period, this mill also proved ineffective and closed down.

In 1899, Oliver "Pete" Zortman, A. H. Reser and the heirs of Thomas O'Hanlon patented the Alabama claim. They hit high-grade ore between 1899 and 1900. In 1902, Zortman, George Putnam, and E. W. King formed the Alder Gulch Mining Company. Zortman was established as a mining camp in 1903 with the construction of the mining company's 100-ton cyanide mill in Alder Gulch about a mile west of town. This mill processed ore from the Alabama and Pole Gulch mines. This mill worked through 1908.

Charles Whitcomb, Bob Coburn, Bill Coburn, and B. D. Phillips formed the Ruby Gulch Mining Company, which bought the Ruby, Mint and Divide claims. They re-opened the Ruby Mine, which had been founded by Warren Troop and George Putnam. The Ruby Gulch Mill was constructed in 1905. After opening surface operations on these claims, an adit, located on the Independent claim, was driven at mill level into the body of a brecciated ore-body. The plant had six 300-ton tanks and six 110-ton tanks for cyanide leaching. The mill was powered by wood burning steam engines. It was destroyed by fire in 1913 and was rebuilt in 1914. The new mill had a 600 ton per day capacity and was powered by electricity.

In 1907, Whitcomb, Phillips, the Coburns and Louis Goslin organized the August Gold Mining Company and bought up that group of claims. They bought the old Landusky mill from the Mission Peak Mining Company, tore out the stamps, and installed roll crushers and cyanide tanks. In 1909, the Ruby Gulch

[1] The Assiniboine and Gros Ventre Tribes of Fort Belknap maintain they were coerced to sell this land in 1896. Claims against the government were addressed in proceedings of the Indian Claims Commission in 1981.

Mining Company bought a two-thirds interest in the August Gold Mining Company consolidating most of the better claims and milling facilities in the area. Throughout the early years, high-grade ore was shipped directly to the smelter in East Helena.

The Ruby Gulch Mining Company reached its peak activity in 1917. After 1918, ore grades dropped and miners' wages rose causing the company to suspend operations. Operations resumed for a short period after December, 1922. However, after the Ruby Mill burned down in October, 1923, the company decided not to rebuild. After B. D. Phillips died in 1926, his family sold their holdings in the mining company. All interests in the mining company, with the exception of the Charles Whitcomb interest, were sold to the Zimmerman family of St. Paul, Minnesota.

Whitcomb, on the other hand, persisted in his efforts to continue mining in the Little Rockies. In 1929, he interested George McGee, Norman Holter, Charles Powers, John Corette and James Finlen in backing a new venture. Based on additional exploration of the August Mine, they formed the Little Ben Mining Company and reopened the August to exploit high-grade ore. They also built a new, larger mill near the head of King Creek. Profitable operations were maintained until 1939. Thereafter, the company shut down and liquidated its assets. Charles Whitcomb died in 1936. His son, George, became actively involved in management of the company, but was killed in Helena under mysterious circumstances in 1937. When the Little Ben Mining Company liquidated, Katie P. Whitcomb bought up the claims.

The towns of Zortman, Landusky and Whitcomb were all founded as a direct result of mining. Whitcomb was abandoned in the 1940's. Much of Zortman burned in 1929. In 1936, a forest fire that burned over 23,000 acres of timber also seriously affected these towns.

During 1939 and 1940, Bert and Tom McGuire bought a 1-2 yard dragline from the Harlem Ditch Company and walked it into the confluence of Swift Gulch and South Big Horn Creek. A force of six men operated the dragline, hopper and trammel, and placer mined about a 1000-foot section of the creek bottom, removing 186 ounces of gold.

During the final years of his life, Whitcomb also set up a venture with Carl J. Trauerman. Under this venture, the Ruby and Alabama mines were reopened in 1935. It is not fully understood whether Ruby Gulch Mining Company stock was acquired from the Zimmerman family or whether the Zimmerman family retained some interest. In any case, a 300-ton cyanide plant was constructed by 1936. By all accounts, these were very profitable operations employing over one hundred men until 1940. These operations were shut down by order of the War Production Board in 1942. The Whitcombs and Carl Trauerman resumed operations in July, 1946. Despite a few high-grade discoveries and the innovative use of a "coyote drift" to blast the west wall of the Alabama glory hole, serious mining ceased by 1951. Block caving was attempted in1953 but the effort failed because the chutes plugged and could not be freed. All of the property was sold at sheriff's sale for $60,000 in 1954.

Between 1951 and 1959, only six ounces of gold was mined from the entire district. Individuals, such as the Wright's and Kolczak's, continued to mine and develop underground workings in the Zortman and Landusky properties. For the most part, the days of the large-scale operations were gone until modern, surface mining operations opened in 1979.

It has been estimated that over 380,000 ounces of gold were mined from the Little Rocky Mountains prior to 1979, contributing significantly to the region's economy. The literature indicates that most of the gold produced was from lode deposits. Early day ore carried about an ounce of silver to the ton and from 0.14 ounce to 1 ounce of gold per ton. The mill run averaged about a 1/3 of an ounce per ton. However, limited high-grade ore was produced from the Little Ben and Ruby Gulch mines. The ore in the Little Rocky Mountains was not of consistently high quality to sustain most of the mines utilizing the mining techniques of the day. Between 1934 and 1968, mining of these low-grade gold deposits was hampered by the price of gold, which was fixed at $35 per ounce by Presidential Proclamation during this period.

The initial stampede of prospectors to the area followed the discovery of gold in alluvial deposits in 1884. The first lodes were not discovered until 1893, when gold was discovered in the August mine. The alluvial deposits along the mountain flanks were 5 to 50 feet thick. Placered areas were located in People Creek, Lodge Pole Creek, Ruby Creek, Alder Creek, Camp Creek, Grouse Creek, Mill Gulch and Rock Creek. The placer gold was weathered from the large low-grade deposits. Lode production came from the August, Gold Bug, and Little Ben Mine north of Landusky; the Mint, Independent and Alabama on Ruby Creek; the Hawkeye on Alder Creek; the Pole Gulch Mine on Pole Gulch; and the Beaver Creek Mines. Minerals produced from the area include gold, silver, copper, lead, and zinc. Prior to 1979,

significant physical disturbance had occurred in Montana Gulch, Beaver Creek and Pony Gulch. Mill tailings were deposited in King Creek, Alder Gulch, and Ruby Gulch.

In 1968, American Exploration Co. (Amex) of Vancouver, B. C., a subsidiary of Placer Development Ltd., Little Rocky Mining Co. Ltd., and Kaiser Aluminum (through its association with Little Rocky) began reevaluating prospects around Zortman and Landusky. At this time, Gold Reserve Mining Company controlled the Ruby Gulch Mines, as well as many claims in the Shell Butte-Scraggy Peak area. During the mid-1970's, Pegasus Gold Corporation was able to identify a viable low-grade reserve. An environmental impact analysis was completed in 1978. This was followed by issuance of a mining permit and the beginning of large-scale open pit mining and heap leaching in 1979.

1.1.1 Historical Geology of the Early Mines

The Little Rocky Mountains form a dome-shaped uplift of sedimentary and metamorphic rocks, which has been modified by the intrusion of a thick sheet of porphyry and by erosion of the younger beds from the top of the dome.

- Metamorphic Rocks – The oldest rocks are crystalline schists of the pre-Cambrian age. They are overlain by Cambrian quartzite.
- Sedimentary rocks – The Cambrian beds rest unconformably upon the metamorphic rocks or are separated from them by intruding porphyry. At the base of the Cambrian is a quartzite bed that is about 75 feet thick and is overlain by shales and limestone. Resting upon these limestones are large massive beds of white or light gray limestone.
- Porphyry – A large, thick sheet of igneous rock forms the central axis of the mountains and is composed of syenite porphyry and varieties of alkali rich rocks. The mass is elliptical and is about 10 miles in diameter.

The ore deposits of the Little Rockies are either (1) zones of fractured porphyry replaced and cemented by quartz and pyrite; or (2) replacement deposits in limestone near intruding porphyry. The ore carries gold and silver. The most important deposits were found in the porphyry. These lodes ranged in width from a few inches to 70 feet. Nearly all the lodes were cut by fissures. Dyson (1938) states that the ores "were deposited in the epithermal zone of ore deposition by ascending thermal solutions emanating from a cooling igneous mass into its consolidated border".

Rodgers and Enders (1982) concluded that the epithermal gold deposits in the Little Rocky Mountains were deposited in structurally prepared areas within the intrusive rocks. "The deposits are localized in strongly fractured areas which have undergone repeated intrusions and structural adjustments. Each deposit's major structural control is subparallel to one of the three main regional trends. Most of the deposits lie along the major axis of the mountain range. Mineralization appears most directly related to the mechanical behavior of the host rock. Those lithologies that shattered most and maintained open fractures became the major host for these structurally controlled, epithermal deposits." Gold and silver mineralization was deposited in veins, shear zones, and fracture stockworks developed in syenite porphyry and quartz monzonite porphyry along major structural zones. "In Zortman, mineralization is developed predominantly in a fracture stockwork system in syenite porphyry adjacent to a strong fault zone between syenite porphyry and a large block of metamorphic gneisses and schists. In Landusky, mineralization is developed predominantly along shear zones in syenite porphyry and quartz monzonite porphyry within a major shear structure. The grade of the Landusky shears and veins is similar but lower than the Zortman stockwork deposits. Landusky deposits have a higher content of silver, iron, and manganese.

The Mint, Independent, and Pole Gulch mines were all worked by the open-stope method. The ore was soft and required little breaking for passage through chutes. Little timbering was necessary as the walls stood fairly well.

Mint Mine - was located near the head of Ruby Gulch. The country rock was porphyry. The lode was a zone of shattered, cemented and replaced porphyry.

Independent Mine – was located 1200 feet south of the Mint on the O.K. fissure. The lode was a fault zone in porphyry with mixed oxide and sulfide ore. It was mined to a depth of about 500 feet underground.

Ruby Mine – was located 1200 feet south of the Mint. It mined a vein to a depth of about 700 feet

underground along the Ruby shear zone.

Alabama Mine – was located 1500 feet west of the Independent. The lode was a shear zone in phorphyry. The ore was brecciated porphyry, replaced and cemented by quartz, pyrite and other minerals.

Pole Gulch Mine – was located 3500 feet south of the Zortman Mill. The ore was soft and partially decomposed limestone, locally silicified and stained with iron oxide.

Gold Bug Mine – was located 1½ miles northwest of Landusky between Mill Gulch and Montana Gulch. The gold bearing lodes were sheared and brecciated zones in phorphyry.

August Mine – was located about three-quarters of a mile north of the Gold Bug. The ore consisted of brecciated porphyry cemented by quartz and stained by limonite.

1.1.2 Historical Areas Within the Site Area

Three mills and the Whitcomb townsite were located near the head of Ruby Gulch. This area is within the Zortman Mine site. The ruins of the Whitcomb Mill, which was constructed in 1936, are still present as is a refinery building and shop building. The older mills both burned. Nearly the entire Whitcomb townsite area is buried beneath leach pads.

1.2 Zortman-Landusky History After 1977

1.2.1 Landusky Open Pit Mining

Landusky Mine operations were initially permitted for hard rock mining by the former Montana Department of State Lands in 1979 under permit number 00095. The surface mine development followed the same structural trends as the earlier underground mines and excavated a substantial portion of the underground workings. Surface excavation stayed above the elevations of the Gold Bug and August drain tunnels. So, underground development below the elevation of 4650 feet would not have been affected. When mining ceased in 1996, nearly the entire defined ore body, which had been controlled by ZMI had been removed. The alternative discussed in the March,1996 EIS covering “Reclamation Plan Modifications and Mine Life Extension” suggest that only an additional 7.6 million tons of ore would have been moved if the expansion alternative had been implemented.

1.2.2 Zortman Open Pit Mining

The Zortman Mine operations were initially permitted for hard rock mining by the former Montana Department of State Lands in 1979 under permit number 00096. Surface mining followed the same structural trends as the earlier Ruby, Independent, and Alabama mines. The open pits mined through the glory holes areas and excavated a substantial portion of the underground workings. When mining ceased in 1989, Zortman Mining Inc. submitted plans to expand the pit complex. It was to be deepened by up to 500 feet in some ore zones, thereby removing all of the known underground workings. The expansion alternatives discussed in the March, 1996 EIS covering “Reclamation Plan Modifications and Mine Life Extension” suggest that an additional 60 million tons of ore and 50 million tons of waste would have been moved if the expansion alternative had been implemented. About 1 million ounces of gold would have been mined if the expansion plans had been implemented. In March 1998, ZMI cancelled its mine expansion plans and liquidated the company in bankruptcy proceedings.

2.0 DETAILED LABOR JOB DESCRIPTIONS

Job Descriptions:

Water Treatment Plant Operator

1. Certification and Training:
    - Must have a Montana industrial waste water treatment plant operator license with a Class 1D Industrial rating
    - Must maintain continuing education training for certification
    - Must have 40-hour OSHA training with 8-hour yearly refresher
    - At least one person on-site must have a State of Montana First-Class Crane Hoisting Operator license
    - MSHA task trained for operation of Cat 16G motor grader, Cat D9N bulldozer, Backhoe, Bobcat, Boom truck, Forklift, 4 Wheel drive pickup, ATV 4-wheeler, and snowmobile
2. Duties
    - Understand computerized controls at plants
    - Daily water sampling with field lab analyses for pH, specific conductivity, and nitrates
    - Weekly measure water levels in leach pads and ponds
    - Daily recording of flow and discharge from all water sources
    - Fencing and fence repair, welding and use of arc welder and cutting torch, plumbing including operation of the HDPE fusion machine, and containment liner repairs
    - Pump and pumping networks – maintenance for continuous operation of pump, change out submersible and centrifuge pumps, maintain packing in pumps, maintain electrical lines and heat trace lines, maintain valves and hoses for water system
    - Maintenance of roads, minor reclamation, stormwater controls, pipe networks, snow removal, and freight delivery using motor grader, bulldozer, and other maintenance equipment

Electrician / Construction Maintenance

1. Certification and Training:
    - Must have electrical training
    - Must have 40-hour OSHA training with 8-hour yearly refresher
    - At least one person on-site must have a State of Montana First-Class Crane Hoisting Operator license
2. Duties
    - Wiring of pumps, auto valves, flow switches, flow meters, heaters, lighting, electrical panels and fans
    - Daily pumpback checks for continuous operation of pumps
    - Oil changes, part changing, repair and maintenance of hydraulics, cutting edges, and lining buckets
    - Fencing and fence repair, welding and use of arc welder and cutting torch, plumbing including operation of the HDPE fusion machine, and containment liner repairs
    - Pump and pumping networks – maintenance for continuous operation of pump, change out submersible and centrifuge pumps, maintain packing in pumps, maintain electrical lines and heat trace lines, maintain valves and hoses for water system
    - Maintenance of roads, minor reclamation, stormwater controls, pipe networks, snow

removal, and freight delivery using motor grader, bulldozer, and other maintenance equipment
- Pulling and placing large pumps at pump stations on leach pads
- Carpenter
- Assist water treatment plant operators in all aspects of plant maintenance

Clerk / Engineering Technician / Water Sampler

1. Certification and Training:
   - Must have 40-hour OSHA training with 8-hour yearly refresher
2. Duties
   - Water sampling of water wells and surface sites in and around the mine site
   - Operation of water sampling pump truck including use of static water level indicator, maintenance of submersible pump, raising and lowering pump and electric generator, and operate air compressor for pumping
   - Sample preparation for shipment to outside lab including filling out chain of custody form, marking sample bottles, package and seal samples in cooler
   - Field water analysis for pH, SC and flows using field meters, flumes, and flow calculations
   - Lab analyses for total suspended solids including SC, pH, filtering samples, and weighing filters
   - Ambient and groundwater field data collection
   - Biological treatment plant water field data collection
   - Surveying with use of Trimble GPS survey instruments, recording data, and electronic data transfer to engineering staff
   - Purchasing/Accounting/Payroll including placing orders for operation and maintenance of all aspects of reclamation and water treatment, filing of purchase orders and purchase logs, organization of time sheets, and forwarding time sheets to headquarters
   - Copier and fax machine maintenance and cleaning, office cleaning including bathrooms
   - Maintenance of roads, minor reclamation, stormwater controls, pipe networks, snow removal, and freight delivery using motor grader, bulldozer, and other maintenance equipment
   - Fencing and fence repair, plumbing, and containment liner repairs
   - Assist water treatment plant operators with plant maintenance

Site Manager

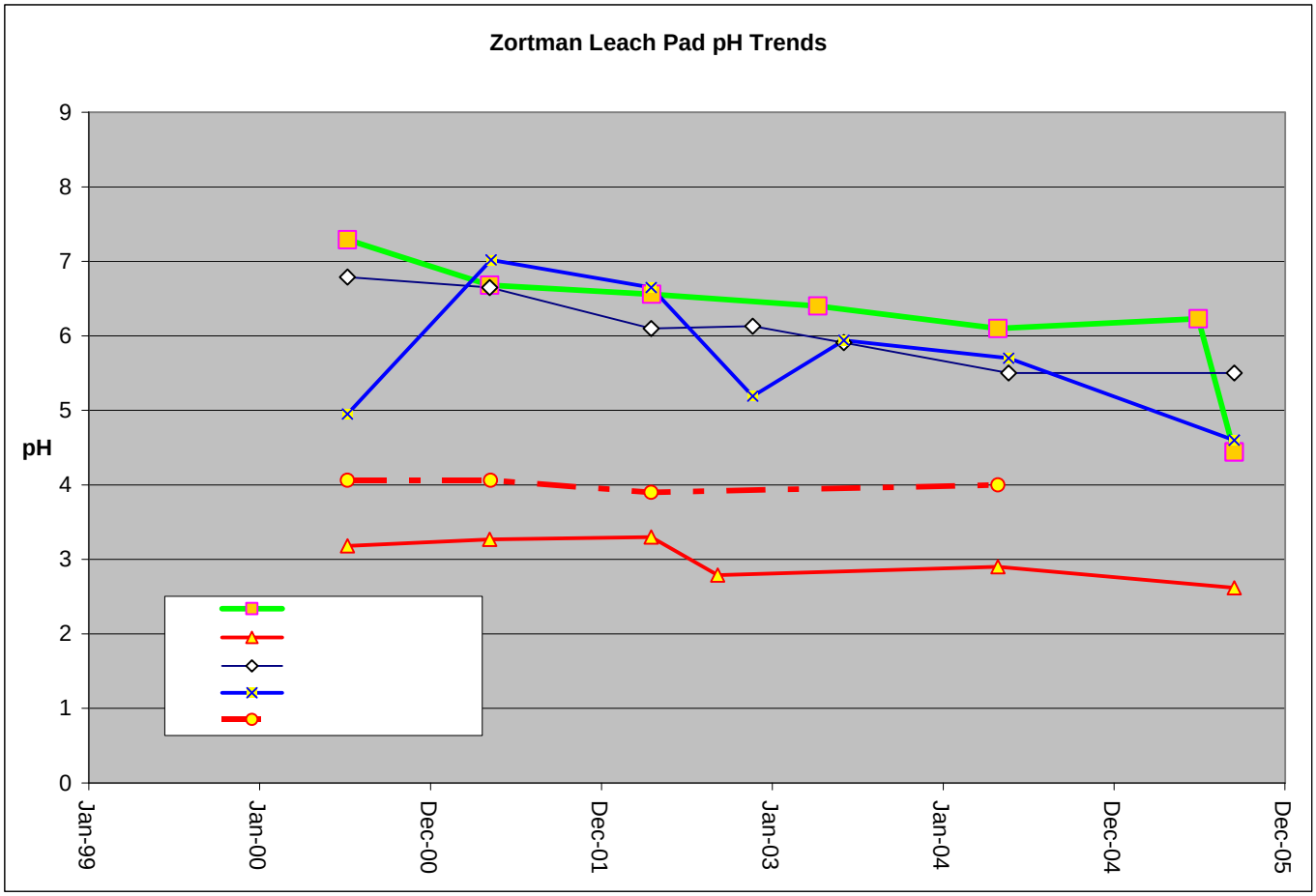
1. Certification and Training:
   - Must have 40-hour OSHA training with 8-hour yearly refresher
2. Duties
   - Oversee all aspects of mine reclamation, vegetation, annual reports, and maintenance
   - Oversee all aspects of two conventional lime water treatment plants and one biological treatment system and all capture systems
   - Water sampling of water wells and surface sites in and around the mine site
   - Operation of water sampling pump truck including use of static water level indicator, maintenance of submersible pump, raising and lowering pump and electric generator, and operate air compressor for pumping

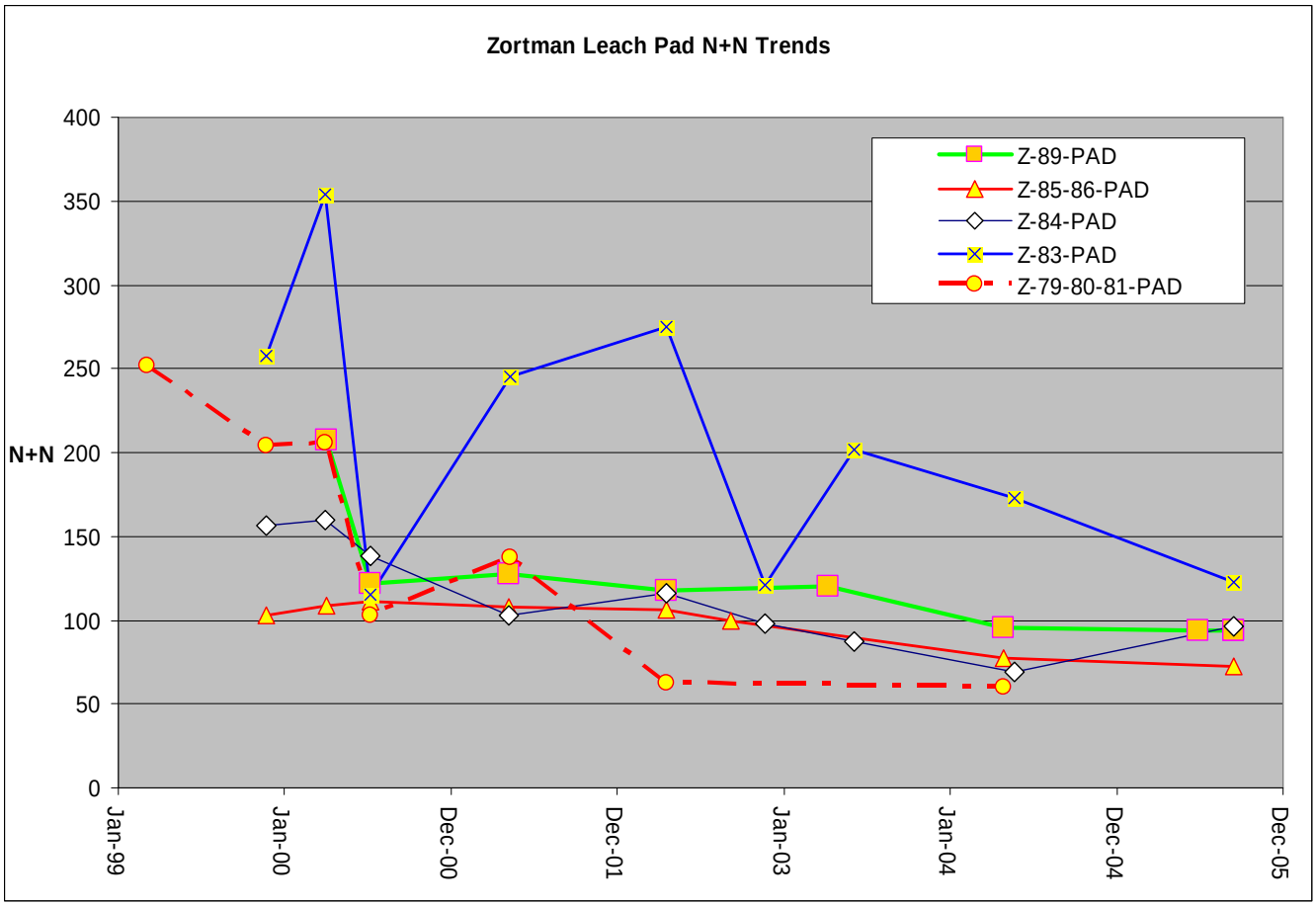
- Sample preparation for shipment to outside lab including filling out chain of custody form, marking sample bottles, package and seal samples in cooler
- Field water analysis for pH, SC and flows using field meters, flumes, and flow calculations
- Lab analyses for total suspended solids including SC, pH, filtering samples, and weighing filters
- Surveying with use of Trimble GPS survey instruments, recording data, electronic data transfer to engineering staff, construct and analyze survey data
- Control mine access including security and safety
- Placing orders for operation and maintenance of all aspects of reclamation and water treatment
- Interaction with engineering department
- Maintain all water lab analyses in Access database
- Knowledge of AutoCAD, Word, Excel, Access
- Maintenance of roads, minor reclamation, stormwater controls, pipe networks, snow removal, and freight delivery using motor grader, bulldozer, and other maintenance equipment
- Assist water treatment plant operators with plant maintenance
- Submit quarterly monitoring reports for water treatment plant effluent and monthly Access database CD's are submitted to the DEQ, the BLM, and the Fort Belknap Environmental Department.

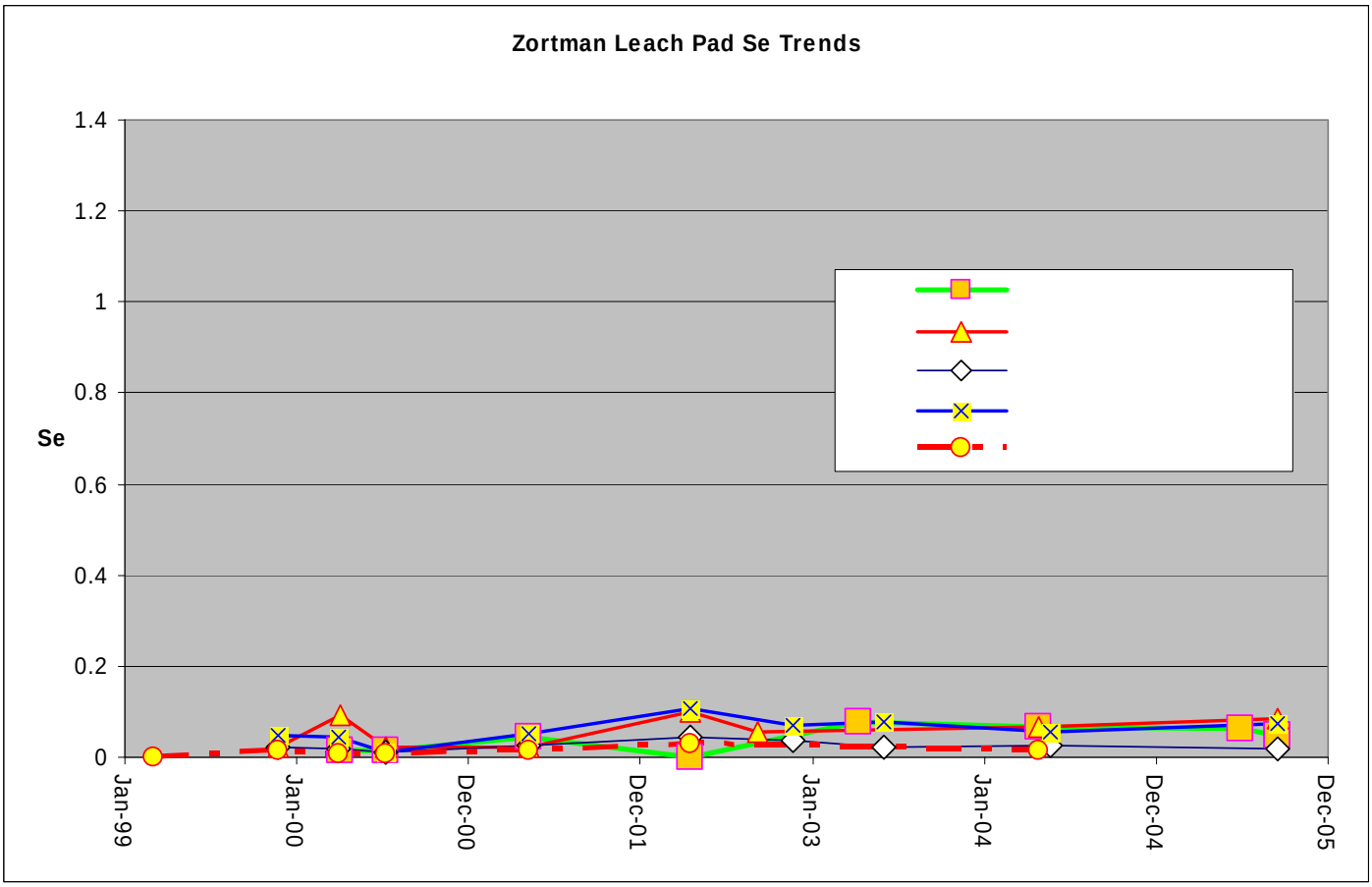
3.0 LEACH PAD CHEMISTRY TRENDS

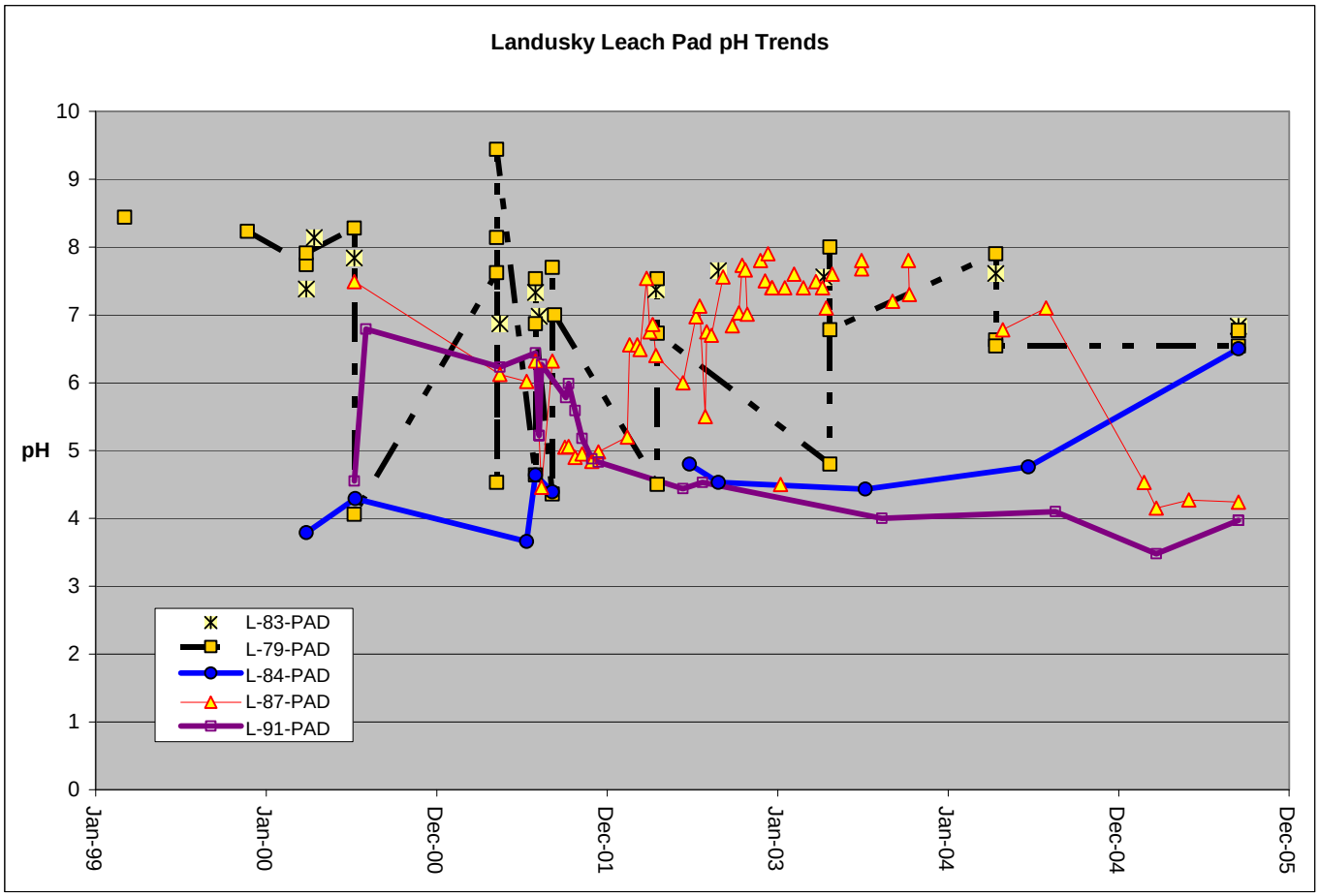
This appendix contains the various graphs showing the trends in the water quality for the leach pads.

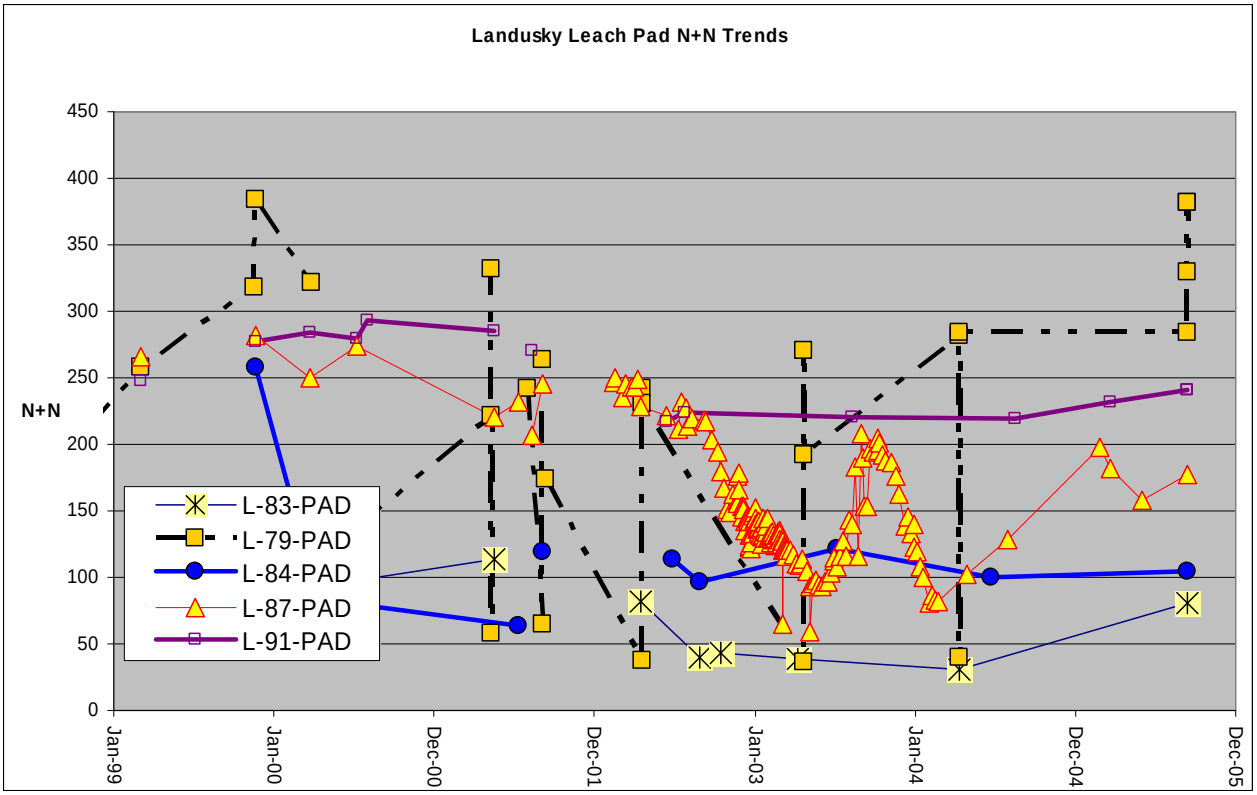
Note that the pH of all the Zortman Mine leach pads is acidic and trending towards more acidic, whereas, at the Landusky Mine, only the L87, L91 and L84 pads are acidic, and the L84 pH trend is from acidic to neutral. The L87 and L91 Pads are trending more acidic.




Zortman Leach Pad N+N Trends
N+N
400
350
300
250
200
150
100
50
0
Jan-99
Jan-00
Dec-00
Dec-01
Jan-03
Jan-04
Dec-04
Dec-05
Z-89-PAD
Z-85-86-PAD
Z-84-PAD
Z-83-PAD
Z-79-80-81-PAD


Zortman Leach Pad Se Trends
Se
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Jan-99
Jan-00
Dec-00
Dec-01
Jan-03
Jan-04
Dec-04
Dec-05


Landusky Leach Pad pH Trends
pH
10
9
8
7
6
5
4
3
2
1
0
Jan-99
Jan-00
Dec-00
Dec-01
Jan-03
Jan-04
Dec-04
Dec-05
L-83-PAD
L-79-PAD
L-84-PAD
L-87-PAD
L-91-PAD


Landusky Leach Pad N+N Trends
N+N
450
400
350
300
250
200
150
100
50
0
L-83-PAD
L-79-PAD
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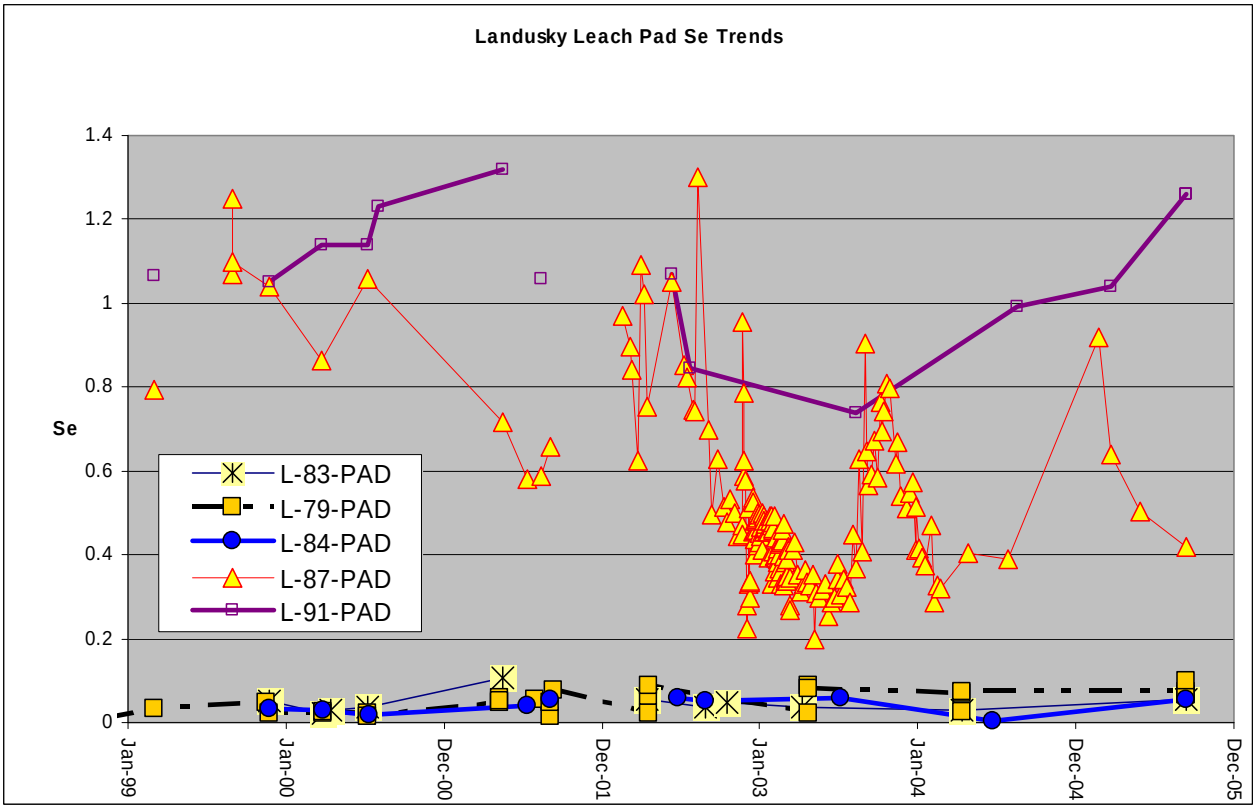
Dec-00
Dec-01
Jan-03
Jan-04
Dec-04
Dec-05


Landusky Leach Pad Se Trends
Se
1.4
1.2
1
0.8
0.6
0.4
0.2
0
L-83-PAD
L-79-PAD
L-84-PAD
L-87-PAD
L-91-PAD
Jan-99
Jan-00
Dec-00
Dec-01
Jan-03
Jan-04
Dec-04
Dec-05

4.0 IDENTIFICATION OF APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARs)

Zortman and Landusky Mines Non-Time-Critical Removal Action			
Standard, Requirement Criteria Or Limitation	**Citation**	**Description**	**ARAR Status**
Federal Contaminant Specific:			
Safe Drinking Water Act National Primary Drinking Water Regulation National Secondary Drinking Water Regulations	40 USC § 300 40 CFR Part 141 40 CFR Part 143	Establishes health-based standards (MCLs) for public water systems. Establishes welfare-based standards (secondary MCLs) for public water systems.	Not an ARAR. Defer to State Standards Not an ARAR, not enforceable standards.
Clean Water Act Water Quality Standards	33 USC 1313(c) 40 CFR Part 131	Water Pollution Prevention & Control EPA's requirements for state water quality standards.	Not an ARAR since the State has primary responsibility over this program and has promulgated water quality standards for the designated beneficial uses.
Federal Location Specific:			
National Historic Preservation Act	16 USC § 470; 36 CFR Part 800; 40 CFR Part 6.310(b)	Requires Federal Agencies to take into account the effect of any Federally-assisted undertaking or licensing on any district, site, building, structure, or object that is included in or eligible for inclusion in the National Register of Historic Places and to minimize harm to any National Historic Landmark adversely or directly affected by an undertaking.	Applicable

Zortman and Landusky Mines Non-Time-Critical Removal Action			
Standard, Requirement Criteria Or Limitation	**Citation**	**Description**	**ARAR Status**
Archaeological and Historic Preservation Act	16 USC § 469; 40 CFR ' 6.301(c)	Establishes procedures to provide for preservation of historical and archaeological data, which might be destroyed through alteration of terrain as a result of a Federal construction project or a Federally licensed activity or program.	Applicable
Historic Sites, Buildings and Antiquities Act	36 CFR § 62.6(d)	Requires Federal agencies to consider the existence and location of landmarks on the National Registry of Natural Landmarks to avoid undesirable impacts on such landmarks.	Applicable
Protection of Wetlands Order	40 CFR Part 6	Avoid adverse impacts to wetlands.	Applicable
Migratory Bird Treaty Act	16 USC § 703 et seq.	Establishes a federal responsibility for the protection of international migratory bird resource.	Applicable
Fish and Wildlife Coordination Act	16 USC § 661 et seq.; 40 CFR Part 6.302(g)	Requires consultation when Federal department or agency proposes or authorizes any modification of any stream or other water body and adequate provision for protection of fish and wildlife resources.	Applicable
Floodplain Management Order	40 CFR Part 6	Requires Federal agencies to evaluate the potential effects of actions they may take in a floodplain to avoid the adverse impacts associated with direct and indirect development of a floodplain, to the extent possible.	Relevant and Appropriate
Bald Eagle Protection Act	16 USC §§ 668 et seq.	Establishes a federal responsibility for protection of bald and golden eagles. Requires consultation with the FWS.	Applicable
Endangered Species Act	16 USC §§ 1531-1543; 40 CFR Part 6.302(h); 50 CFR Part 402	Requires action to conserve endangered species within critical habitat upon which species depend. Includes consultation with FWS.	Applicable

Zortman and Landusky Mines Non-Time-Critical Removal Action			
Standard, Requirement Criteria Or Limitation	**Citation**	**Description**	**ARAR Status**
Federal Action Specific:			
Federal Land Management and Policy Act Prevent unnecessary or undue degradation of federal lands	43 CFR 3809	Requires approved reclamation plan for mining operations including plans for post-closure management (includes water treatment when necessary)	Relevant and Appropriate
Clean Water Act National Pollutant Discharge Elimination System	33 USC §§ 1342 40 CFR Parts 122	Requires permits for the discharge of pollutants from any point source into waters of the United States.	Relevant and -Appropriate
Resource Conservation and Recovery Act	40 CFR Part 264	Provisions regarding run-on and run-off controls	Not an ARAR. Removal action will not create a RCRA TSD facility.
Occupational Safety & Health Act Hazardous Waste Operations And Emergency Response	29 USC § 655 29 CFR 1910.120	Defines standards for employee protection during initial site characterization and analysis, monitoring activities, materials handling activities, training & ER.	Applicable

Zortman and Landusky Mines Non-Time-Critical Removal Action			
Standard, Requirement Criteria Or Limitation	**Citation**	**Description**	**ARAR Status**
State Contaminant Specific:			
Montana Water Quality Act Regulations Establishing Ambient Surface Water Quality Standards	75-5-101 et seq., MCA	Establishes Montana's laws to prevent, abate and control the pollution of state waters	Applicable
	ARM 17.30.601 et seq.	Provides the water use classification for various streams and imposes specific water quality standards per classification.	Applicable
	ARM 17.30.637	Provides that surface waters must be free of substances attributable to industrial practices or other discharges that will: (a) settle to form objectionable sludge deposits or emulsions beneath the surface of the water or upon adjoining shorelines; (b) create floating debris, scum, a visible oil film or globules of grease or other floating materials; (c) produce odors, colors, or other conditions which create a nuisance or render undesirable tastes to fish or make fish inedible; (d) create concentrations or combinations of materials which are toxic or harmful to human, animal, plant or aquatic life; (e) create conditions which produce undesirable aquatic life.	Applicable
Montana Groundwater Pollution Control System Regulations	ARM 17.30.1011	Applies nondegradation requirements to any activity which could cause a new or increased source of pollution to state water	Applicable
	ARM 17.30.1006	Classifies groundwater into Classes I through IV based on the present and future most beneficial uses of the groundwater and states groundwater is to be classified to actual quality of actual use, whichever places the groundwater in a higher class.	Applicable

Zortman and Landusky Mines Non-Time-Critical Removal Action			
Standard, Requirement Criteria Or Limitation	**Citation**	**Description**	**ARAR Status**
Clean Air Act Of Montana	75-2-101, MCA	Montana policy is to achieve and maintain such levels of air quality as will protect human health and safety and, to the greatest degree practicable, prevent injury to plant and animal life and property.	Applicable
Air Quality Regulations	ARM 17.8.206	Establishes sampling, data collection, and analytical requirements to ensure compliance with ambient air quality standards.	Applicable
	ARM 17.8.222	No person shall cause or contribute to concentrations of lead in the ambient air which exceed the following 90-day average: 1.5 micrograms per cubic meter of air.	Applicable
	ARM 17.8.220	No person shall cause or contribute to concentrations of particulate matter in the ambient air such that the mass of settled particulate matter exceeds the following 30-day average: 10 grams per square meter.	Applicable
	ARM 17.8.223	No person may cause or contribute to concentrations of PM-10 in the ambient air which exceed the following standards: 1) 24-hr. avg.: 150 micrograms per cubic meter of air, with no more than one expected exceedance per year; 2) Annual avg.: 50 micrograms per cubic meter of air.	Applicable
Occupational Health Act of Montana	50-70-101, et. seq., MCA	The purpose of this act is to achieve and maintain such conditions of the work place as will protect human health and safety	Applicable
Occupational Air Contaminants Regulations	ARM 17.42.102	Establishes maximum threshold limit values for air contaminants believed that nearly all workers may be repeatedly exposed day after day without adverse health effects.	Applicable
Occupational Noise Regulations	ARM 17.42.101	Addresses occupational noise levels and provides that no worker should be exposed to noise levels in excess of the specified levels.	Applicable

Zortman and Landusky Mines Non-Time-Critical Removal Action			
Standard, Requirement Criteria Or Limitation	**Citation**	**Description**	**ARAR Status**
State Location Specific:			
Floodplain and Floodway Management Act	76-5-401, MCA	Lists the uses permissible in a floodway and generally prohibits permanent structures, fill, or permanent storage of materials or equipment.	Applicable
Floodplain Management Regulations	76-5-402 MCA	Lists the permissible permanent structures that are allowed in the floodplain excluding the floodway, if they are permitted and meet certain minimum standards.	Applicable
Floodplain Management Regulations (cont'd)	76-5-403, MCA	Lists certain uses which are prohibited in a designated floodway, including any change that will cause water to be diverted from the established floodway, cause erosion, obstruct the natural flow of water, or reduce the carrying capacity of the floodway, or the concentration or permanent storage of an object subject to flotation or movement during flood level periods.	Applicable
	ARM 36.15.216	The factors to consider in determining whether a permit should be issued to establish or alter an artificial obstruction or nonconforming use in the floodplain or floodway are set forth in this section.	Applicable
	ARM 36.15.602	Specifies uses requiring permits for allowing obstructions in the floodway.	Applicable
	ARM 36.15.603	Proposed diversions or changes in place of diversions must be evaluated by the DNRC to determine whether they may significantly affect flood flows and, therefore, require a permit.	Applicable
	ARM 36.15.604	Prohibits new artificial obstructions or nonconforming uses that will increase the upstream elevation of the base flood 0.5 of a foot or significantly increase flood velocities.	Applicable

Zortman and Landusky Mines Non-Time-Critical Removal Action			
Standard, Requirement Criteria Or Limitation	**Citation**	**Description**	**ARAR Status**
Floodplain Management Regulations (continued)	ARM 36.15.605	Identifies artificial obstructions and nonconforming uses that are prohibited within the designated floodway except as allowed by permit and includes "a structure or excavation that will cause water to be diverted from the established floodway, cause erosion, obstruct the natural flow of water, or reduce the carrying capacity of the floodway..." Solid waste disposal and storage of highly toxic, flammable, or explosive materials are also prohibited.	Applicable
	ARM 36.15.701 and 703	Describes allowed uses in the flood fringe. Prohibited uses within the flood fringe (i.e., areas in the floodplain, but outside of the designated floodway) areas including solid waste disposal and storage of highly toxic, flammable or explosive material.	Applicable
	ARM 36.15.801	Allowed uses where floodway is not designated.	Applicable
Montana Solid Waste Management Act and Regulations	75-10-201, MCA ARM 17.50.505	Specifies the requirements that apply to the location of any solid waste management facility	Applicable
Endangered Species	87-5-106, 107,111, MCA ARM 12.5.201	Fish and wildlife resources are to be protected and no construction project or hydraulic project shall adversely affect game or fish habitat.	Applicable

Zortman and Landusky Mines Non-Time-Critical Removal Action			
Standard, Requirement Criteria Or Limitation	**Citation**	**Description**	**ARAR Status**
State Action Specific:			
Montana Water Quality Act	75-5-605, MCA	It is unlawful to cause pollution of any state waters, to place any wastes in a location where they are likely to cause pollution of any state waters, to violate any permit provision, to violate any provision of the Montana Water Quality Act, to construct, modify, or operate a system for disposing of waste (including sediment, solid waste and other substances that may pollute state waters) which discharges into any state waters without a permit or discharges waste into any state waters.	Applicable
MPDES Permit Requirements	ARM17.30.1342-1344	Sets forth the substantive requirements applicable to all MPDES and NPDES permits. Includes the requirement to properly operate and maintain all facilities and systems of treatment and control.	Relevant and Appropriate
	ARM 17.30.1203 and 1344	Technology-based treatment for MPDES permits.	Relevant and Appropriate
Nondegradation of Water Quality	75-5-303, MCA	Existing uses of state waters and the level of water quality necessary to protect the uses must be maintained and protected.	Relevant
	75-5-317, MCA	Provides exemption that allows changes of existing water quality resulting from emergency or remedial activity designed to protect the public health or the environment.	Relevant
	ARM 17.30.705(2)(a)	Provides that for any surface water, existing and anticipated uses and the water quality necessary to protect these uses must be maintained and protected.	Relevant
Clean Air Act Of Montana	75-2-101, MCA	Montana's policy is to achieve and maintain such levels of air quality as will protect human health and safety and, to the greatest degree practicable, prevent injury to plant and animal life and property.	Applicable

Zortman and Landusky Mines Non-Time-Critical Removal Action			
Standard, Requirement Criteria Or Limitation	**Citation**	**Description**	**ARAR Status**
Air Quality Requirements	ARM 17.8.308	No person shall cause or authorize the production, handling, transportation or storage of any material unless reasonable precautions to control emissions of airborne particulate matter are taken.	Applicable
	ARM 17.8.604	Lists certain wastes that may not be disposed of by open burning.	Applicable
	ARM 17.8.1401-1404	Sets forth emission standards for hazardous air pollutants	Applicable
Montana Solid Waste Management Act	75-10-201, et seq, MCA	Public policy is to control solid waste management systems to protect the public health and safety and to conserve natural resources whenever possible.	Relevant and Appropriate
Solid Waste Management Regulations	ARM 17.50.523	Solid waste must be transported in such a manner as to prevent its discharge, dumping, spilling or leaking from the transport vehicle.	Relevant and Appropriate
Montana Metal Mine Reclamation Act	82-4-301 et seq. MCA	Describes reclamation plan requirements and access rights. Disturbed areas must be reclaimed to utility and stability comparable to areas adjacent, except existing facilities may be left in place if they are valuable for an approved post-mining use.	Relevant and Appropriate Relevant and Appropriate
Montana Strip and Underground Mine Reclamation Act	82-4-231, MCA	Sets forth objectives that require the operator to prepare and carry out a method of operations plan to reclaim and revegetate the land affected by his operation	Relevant and Appropriate
	82-4-233, MCA	Requires that after the operation has been backfilled, graded, topsoiled and approved, the operator shall establish a vegetative cover on all impacted lands. Specifications for the vegetative cover and performance are provided.	Relevant and Appropriate

Zortman and Landusky Mines Non-Time-Critical Removal Action			
Standard, Requirement Criteria Or Limitation	**Citation**	**Description**	**ARAR Status**
Hydrology Requirements	ARM 17.24.631	Reclamation operations must be planned and conducted to minimize disturbance and prevent damage to the prevailing hydrologic balance.	Relevant and Appropriate
	ARM 17.24.633	Specifies that sediment controls must be maintained until the disturbed area has been restored and revegetated.	Relevant and Appropriate
	ARM 17.24.634	Drainage design shall emphasize premining channel and floodplain configurations that blend with the undisturbed drainage system above and below; will meander naturally; remain in dynamic equilibrium with the system; improve unstable premining conditions, provide for floods, provide for long term stability of the landscape; and establish a premining diversity of aquatic habitats and riparian vegetation.	Relevant and Appropriate
	ARM 17.24.635-637	Sets forth requirements for temporary and permanent diversions.	Relevant and Appropriate
	ARM 17.24.641	Sets methods for preventing drainage from acid-and toxic-forming wastes into ground and surface waters.	Relevant and Appropriate
Top Soiling, Revegetation, and Protection of Wildlife and Air Resource Regulations	ARM 17.24.703	Materials other than, or along with, soil for final surfacing of spoils or other disturbances must be capable of supporting the approved vegetation and postmining land use.	Relevant and Appropriate
Top Soiling, Revegetation, and Protection of Wildlife and Air Resource Regulations (cont'd)	ARM 17.24.713	Specifies that seeding and planting of disturbed areas must be conducted during the first appropriate period for favorable planting after final seedbed preparation; but not longer than 90 days after topsoil placement.	Relevant and Appropriate
	ARM 17.24.714	According to this section, as soon as practical, a mulch or cover crop must be used on all regraded and resoiled areas to control erosion, to promote germination of seeds, and to increase moisture retention of soil until permanent cover is established.	Relevant and Appropriate

Zortman and Landusky Mines Non-Time-Critical Removal Action			
Standard, Requirement Criteria Or Limitation	**Citation**	**Description**	**ARAR Status**
	ARM 17.24.716	Establishes methods of revegetation	Relevant and Appropriate
	ARM 17.24.718	Soil amendments must be used as necessary to aid in the establishment of permanent vegetation; irrigation, management, fencing, or other measures may also be used after review and approval by the dep't.	Relevant and Appropriate
	ARM 17.24.751	Required site activities must be conducted so as to avoid or minimize impacts to important fish and wildlife species, including critical habitat and any threatened or endangered species identified at the site.	Relevant and Appropriate
	ARM 17.24.761	Section requires fugitive dust control measures for site preparation and reclamation operations.	Relevant and Appropriate

5.0 TOXICITY PROFILE SUMMARIES

Arsenic

The toxicity of inorganic arsenic (As) depends on its valence state (-3, +3, or +5), and also on the physical and chemical properties of the compound in which it occurs. Trivalent (As+3) compounds are generally more toxic than pentavalent (As+5) compounds, and the more water soluble compounds are usually more toxic and more likely to have systemic effects than the less soluble compounds, which are more likely to cause chronic pulmonary effects if inhaled. One of the most toxic inorganic arsenic compounds is arsine gas (AsH3). It should be noted that laboratory animals are generally less sensitive than humans to the toxic effects of inorganic arsenic. In addition, in rodents the critical effects appear to be immunosuppression and hepato-renal dysfunction, whereas in humans the skin, vascular system, and peripheral nervous system are the primary target organs.

Water soluble inorganic arsenic compounds are absorbed through the G.I. tract (>90%) and lungs; distributed primarily to the liver, kidney, lung, spleen, aorta, and skin; and excreted mainly in the urine at rates as high as 80% in 61 hours following oral dosing (EPA, 1984; ATSDR, 1989; Crecelius, 1977). Pentavalent arsenic is reduced to the trivalent form and then methylated in the liver to less toxic methylarsinic acids (ATSDR, 1989).

Symptoms of acute inorganic arsenic poisoning in humans are nausea, anorexia, vomiting, epigastric and abdominal pain, and diarrhea. Dermatitis (exfoliative erythroderma), muscle cramps, cardiac abnormalities, hepatotoxicity, bone marrow suppression and hematologic abnormalities (anemia), vascular lesions, and peripheral neuropathy (motor dysfunction, paresthesia) have also been reported (U.S. Air Force, 1990; ATSDR, 1989; Franzblau and Lilis, 1989; EPA, 1984; Armstrong et al., 1984; Hayes, 1982; Mizuta et al., 1956). Oral doses as low as 20-60 g/kg/day have been reported to cause toxic effects in some individuals (ATSDR, 1989). Severe exposures can result in acute encephalopathy, congestive heart failure, stupor, convulsions, paralysis, coma, and death. The acute lethal dose to humans has been estimated to be about 0.6 mg/kg/day (ATSDR, 1989). General symptoms of chronic arsenic poisoning in humans are weakness, general debility and lassitude, loss of appetite and energy, loss of hair, hoarseness of voice, loss of weight, and mental disorders (Hindmarsh & McCurdy, 1986). Primary target organs are the skin (hyperpigmentation and hyperkeratosis) [Terada et al. 1960; Tseng et al., 1968; Zaldivar 1974; Cebrian et al., 1983; Huang et al., 1985], nervous system (peripheral neuropathy) [Hindmarsh et al., 1977, 1986; Valentine et al., 1982; Heyman et al., 1956; Mizuta et al., 1956; Tay & Seah, 1975], and vascular system [Tseng et al., 1968; Borgano & Greiber, 1972; Salcedo et al., 1984; Wu et al., 1989; Hansen, 1990]. Anemia, leukopenia, hepatomegaly, and portal hypertension have also been reported (Terada et al., 1960; Viallet et al., 1972; Morris et al., 1974; Datta, 1976). In addition, possible reproductive effects include a high male to female birth ratio (Lyster, 1977).

In animals, acute oral exposures can cause gastrointestinal and neurological effects (Heywood and Sortwell, 1979). Oral LD50 values range from about 10 to 300 mg/kg (ASTDR, 1989; U.S. Air Force, 1990). Low subchronic doses can result in immunosuppression, (Blakely et al., 1980) and hepato-renal effects (Mahaffey et al., 1981; Brown et al., 1976; Woods & Fowler, 1977, 1978; Fowler & Woods, 1979; Fowler et al., 1979). Chronic exposures have also resulted in mild hyperkeratosis and bile duct enlargement with hyperplasia, focal necrosis, and fibrosis (Baroni et al., 1963; Byron et al., 1967). Reduction in litter size, high male/female birth ratios, and fetotoxicity without significant fetal abnormalities occur following oral exposures (Schroeder & Mitchener, 1971; Hood et al., 1977; Baxley et al., 1981); however, parenteral dosing has resulted in exencephaly, encephaloceles, skeletal defects, and urogenital system abnormalities (Ferm & Carpenter, 1968; Hood & Bishop, 1972; Beaudoin, 1974; Burk & Beandoin, 1977).

The Reference Dose for chronic oral exposures, 0.0003 mg/kg/day, is based on a NOAEL of 0.0008 mg/kg/day and a LOAEL of 0.014 mg/kg/day for hyperpigmentation, keratosis, and possible vascular complications in a human population consuming arsenic-contaminated drinking water (EPA, 1991a). Because of uncertainties in the data, EPA (1991a) states that "strong scientific arguments can be made for various values within a factor of 2 or 3 of the currently recommended RfD value." The subchronic

Reference Dose is the same as the chronic RfD, 0.0003 mg/kg/day (EPA, 1992).

Acute inhalation exposures to inorganic arsenic can damage mucous membranes, cause rhinitis, pharyngitis and laryngitis, and result in nasal septum perforation (EPA, 1984). Chronic inhalation exposures, as occurring in the workplace, can lead to rhino-pharyno-laryngitis, tracheobronchitis, (Lundgren, 1954); dermatitis, hyperpigmentation, and hyperkeratosis (Perry et al., 1948; Pinto & McGill, 1955); leukopenia (Kyle & Pease, 1965; Hine et al., 1977); peripheral nerve dysfunction as indicated by abnormal nerve conduction velocities (Feldman et al., 1979; Blom et al., 1985; Landau et al., 1977); and peripheral vascular disorders as indicated by Raynaud's syndrome and increased vasospastic reactivity in fingers exposed to low temperatures (Lagerkvist et al., 1986). Higher rates of cardiovascular disease have also been reported in some arsenic-exposed workers (Lee & Fraumeni, 1969; Axelson et al., 1978; Wingren & Axelson, 1985). Possible reproductive effects include a high frequency of spontaneous abortions and reduced birth weights (Nordström et al., 1978a,b). Arsine gas (AsH3), at concentrations as low as 3-10 ppm for several hours, can cause toxic effects. Hemolysis, hemoglobinuria, jaundice, hemolytic anemia, and necrosis of the renal tubules have been reported in exposed workers (ACGIH, 1986; Fowler & Weissberg, 1974).

Animal studies have shown that inorganic arsenic, by intratracheal instillation, can cause pulmonary inflammation and hyperplasia (Webb et al., 1986, 1987), lung lesions (Pershagen et al., 1982), and immunosuppression (Hatch et al. (1985). Long-term inhalation exposures have resulted in altered conditioned reflexes and CNS damage (Rozenshstein, 1970). Reductions in fetal weight and in the number of live fetuses, and increases in fetal abnormalities due to retarded osteogenesis have been observed following inhalation exposures (Nagymajtenyi et al., 1985).

Subchronic and chronic RfCs for inorganic arsenic have not been derived.

Epidemiological studies have revealed an association between arsenic concentrations in drinking water and increased incidences of skin cancers (including squamous cell carcinomas and multiple basal cell carcinomas), as well as cancers of the liver, bladder, respiratory and gastrointestinal tracts (U.S. EPA, 1987; IARC, 1987; Sommers et al., 1953; Reymann et al., 1978; Dobson et al., 1965; Chen et al., 1985, 1986). Occupational exposure studies have shown a clear correlation between exposure to arsenic and lung cancer mortality (IARC, 1987; U.S. EPA, 1991a). U.S. EPA (1991a) has placed inorganic arsenic in weight-of-evidence group A, human carcinogen. A drinking water unit risk of 5E-5(ug/L)-1 has been proposed (U.S. EPA, 1991a); derived from drinking water unit risks for females and males that are equivalent to slope factors of 1.0E-3 (ug/kg/day)-1 (females) and 2.0E-3 (ug/kg/day)-1 (males) (U.S. EPA, 1987). For inhalation exposures, a unit risk of 4.3E-3 (ug/m3)-1 (U.S. EPA, 1991a) and a slope factor of 5.0E+1 (mg/kg/day)-1 have been derived (U.S. EPA, 1992).

Aluminum

Aluminum is a silver-white flexible metal with a vast number of uses. It is poorly absorbed and efficiently eliminated; however, when absorption does occur, aluminum is distributed mainly in bone, liver, testes, kidneys, and brain (ATSDR, 1990).

Aluminum may be involved in Alzheimer's disease (dialysis dementia) and in Amyotrophic Lateral Sclerosis and Parkinsonism-Dementia Syndromes of Guam (Guam ALS-PD complex) (ATSDR, 1990; Goyer, 1991). Aluminum content of brain, muscle, and bone increases in Alzheimer's patients. Neurofibrillary tangles (NFTs) are found in patients suffering from aluminum encephalopathy and Alzheimer's disease. Symptoms of "dialysis dementia" include speech disorders, dementia, convulsions, and myoclonus. People of Guam and Rota have an unusually high incidence of neurodegenerative diseases. The volcanic soil in the region of Guam where the high incidence of ALS-PD occurs contains high levels of aluminum and manganese. Neurological effects have also been observed in rats orally exposed to aluminum compounds.

The respiratory system appears to be the primary target following inhalation exposure to aluminum. Alveolar proteinosis has been observed in guinea pigs, rats, and hamsters exposed to aluminum powders (Gross et al., 1973). Rats and guinea pigs exposed to aluminum chlorohydrate exhibited an increase in alveolar macrophages, increased relative lung weight, and multifocal granulomatous pneumonia (Cavender et al., 1978).

No decrease in reproductive capacity, hormonal abnormalities, or testicular histopathology was observed in male rats exposed to aluminum in drinking water for 90 days (Dixon et al., 1979). However, male rats exposed to aluminum (as aluminum chloride) via gavage for 6 months exhibited decreased spermatozoa counts and sperm motility, and testicular histological and histochemical changes (Krasovskii et al., 1979).

Subchronic and chronic reference doses and reference concentrations have not been derived for aluminum.

Male rats exposed to drinking water containing aluminum (as aluminum potassium sulfate) for a lifetime exhibited increases in unspecified malignant and nonmalignant tumors (Schroeder and Mitchener, 1975a), and similarly exposed female mice exhibited an increased incidence of leukemia (Schroeder and Mitchener, 1975b). Rats and guinea pigs exposed via inhalation to aluminum chlorohydrate developed lung granulomas (Cavender et al., 1978), while granulomatous foci developed in similarly exposed male hamsters (Drew et al., 1974).

The EPA has not evaluated aluminum or aluminum compounds for carcinogenicity, and a weight-of-evidence classification is currently not assigned.

Cadmium

Cadmium is a naturally occurring metal that is used in various chemical forms in metallurgical and other industrial processes, and in the production of pigments. Environmental exposure can occur via the diet and drinking water (ATSDR, 1989).

Cadmium is absorbed more efficiently by the lungs (30 to 60%) than by the gastrointestinal tract, the latter being a saturable process (Nordberg et al., 1985). Cadmium is transported in the blood and widely distributed in the body but accumulates primarily in the liver and kidneys (Goyer, 1991). Cadmium burden (especially in the kidneys and liver) tends to increase in a linear fashion up to about 50 or 60 years of age after which the body burden remains somewhat constant. Metabolic transformations of cadmium are limited to its binding to protein and nonprotein sulfhydryl groups, and various macromolecules, such as metallothionein, which is especially important in the kidneys and liver (ATSDR, 1989). Cadmium is excreted primarily in the urine.

Acute oral exposure to 20-30 g have caused fatalities in humans. Exposure to lower amounts may cause gastrointestinal irritation, vomiting, abdominal pain, and diarrhea (ATSDR, 1989). An asymptomatic period of one-half to one hour may precede the onset of clinical signs. Oral LD50 values in animals range from 63 to 1125 mg/kg, depending on the cadmium compound (USAF, 1990). Longer term exposure to cadmium primarily affects the kidneys, resulting in tubular proteinosis although other conditions such as "itai-itai" disease may involve the skeletal system. Cadmium involvement in hypertension is not fully understood (Goyer, 1991).

Inhalation exposure to cadmium and cadmium compounds may result in effects including headache, chest pains, muscular weakness, pulmonary edema, and death (USAF, 1990). The 1-minute and 10-minute lethal concentration of cadmium for humans has been estimated to be about 2,500 and 250 mg/m3, respectively (Barrett et al., 1947; Beton et al., 1966). An 8-hour TWA (time-weighted-average) exposure level of 5 mg/m3 has been estimated for lethal effects of inhalation exposure to cadmium, and exposure to 1 mg/m3 is considered to be immediately dangerous to human health (Friberg, 1950). Renal toxicity (tubular proteinosis) may also result from inhalation exposure to cadmium (Goyer, 1991).

Chronic oral RfDs of 5E-4 and 1E-3 mg/kg/day have been established for cadmium exposure via drinking water and food, respectively (EPA, 1991). Both values reflect incorporation of an uncertainty factor of 10. The RfDs are based on an extensive data base regarding toxicokinetics and toxicity in both human and animals, the critical effect being renal tubular proteinuria. Confidence in the RfD and data base is high. Inhalation RfC values are currently not available.

The target organ for cadmium toxicity via oral exposure is the kidney (Goyer, 1991). For inhalation exposure, both the lungs and kidneys are target organs for cadmium-induced toxicity (ATSDR, 1989; Goyer, 1991).

There is limited evidence from epidemiologic studies for cadmium-related respiratory tract cancer (ATSDR, 1989). An inhalation unit risk of 1.8E-3 (µg/m3)-1 and an inhalation slope factor of 6.1E+0

(mg/kg/day)-1 are based on respiratory tract cancer associated with occupational exposure (EPA, 1985). Based on limited evidence from multiple occupational exposure studies and adequate animal data, cadmium is placed in weight-of-evidence group B1 - probable human carcinogen.

Copper

Copper occurs naturally in elemental form and as a component of many minerals. Because of its high electrical and thermal conductivity, it is widely used in the manufacture of electrical equipment. Common copper salts, such as the sulfate, carbonate, cyanide, oxide, and sulfide are used as fungicides, as components of ceramics and pyrotechnics, for electroplating, and for numerous other industrial applications (ACGIH, 1986). Copper can be absorbed by the oral, inhalation, and dermal routes of exposure. It is an essential nutrient that is normally present in a wide variety of tissues (ATSDR, 1990; U.S. EPA, 1987).

In humans, ingestion of gram quantities of copper salts may cause gastrointestinal, hepatic, and renal effects with symptoms such as severe abdominal pain, vomiting, diarrhea, hemolysis, hepatic necrosis, hematuria, proteinuria, hypotension, tachycardia, convulsions, coma, and death (U.S. AF, 1990). Gastrointestinal disturbances and liver toxicity have also resulted from long-term exposure to drinking water containing 2.2-7.8 mg Cu/L (Mueller-Hoecker et al., 1988; Spitalny et al., 1984). The chronic toxicity of copper has been characterized in patients with Wilson's disease, a genetic disorder causing copper accumulation in tissues. The clinical manifestations of Wilson's disease include cirrhosis of the liver, hemolytic anemia, neurologic abnormalities, and corneal opacities (Goyer, 1991; ATSDR, 1990; U.S. EPA, 1987). In animal studies, oral exposure to copper caused hepatic and renal accumulation of copper, liver and kidney necrosis at doses of >=100 mg/kg/day; and hematological effects at doses of 40 mg/kg/day (EPA, 1986; Haywood, 1985; 1980; Rana & Kumar, 1978; Gopinath et al., 1974; Kline et al., 1971).

Acute inhalation exposure to copper dust or fumes at concentrations of 0.075-0.12 mg Cu/m3 may cause metal fume fever with symptoms such as cough, chills and muscle ache (U.S. AF, 1990). Among the reported effects in workers exposed to copper dust are gastrointestinal disturbances, headache, vertigo, drowsiness, and hepatomegaly (Suciu et al., 1981). Vineyard workers chronically exposed to Bordeaux mixture (copper sulfate and lime) exhibit degenerative changes of the lungs and liver. Dermal exposure to copper may cause contact dermatitis in some individuals (ATSDR, 1990).

Oral or intravenous administration of copper sulfate increased fetal mortality and developmental abnormalities in experimental animals (Lecyk, 1980; Ferm and Hanlon, 1974). Evidence also indicates that copper compounds are spermicidal (ATSDR, 1990; Battersby et al., 1982).

A Reference Dose (RfD) for elemental copper is not available (U.S. EPA, 1992). However, EPA established an action level of 1300 ug/L for drinking water (56 FR 26460, June 7, 1991). Data were insufficient to derive a Reference concentration (RfC) for copper.

No suitable bioassays or epidemiological studies are available to assess the carcinogenicity of copper. Therefore, U.S. EPA (1991a) has placed copper in weight-of-evidence group D, not classifiable as to human carcinogenicity.

Cyanide

Cyanide most commonly occurs as hydrogen cyanide and its salts--sodium and potassium cyanide. Cyanides are both man-made and naturally occurring substances. They are found in several plant species as cyanogenic glycosides and are produced by certain bacteria, fungi, and algae. In very small amounts, cyanide is a necessary requirement in the human diet. Cyanides are released to the environment from industrial sources and car emissions (ATSDR, 1989).

Cyanides are readily absorbed by the inhalation, oral, and dermal routes of exposure. The central nervous system (CNS) is the primary target organ for cyanide toxicity. Neurotoxicity has been observed in humans and animals following ingestion and inhalation of cyanides. Cardiac and respiratory effects, possibly CNS-mediated, have also been reported. Short-term exposure to high concentrations produces

almost immediate collapse, respiratory arrest, and death (Hartung, 1982; EPA, 1985). Symptoms resulting from occupational exposure to lower concentrations include breathing difficulties, nervousness, vertigo, headache, nausea, vomiting, precordial pain, and electrocardiogram (EKG) abnormalities (Carmelo, 1955; El Ghawabi et al., 1975; Sandberg, 1967; Wuthrich, 1954). Thyroid toxicity has been observed in humans and animals following oral and inhalation exposure to cyanides (Philbrick et al.,1979; EPA, 1984). In animal studies, cyanides have produced fetotoxicity and teratogenic effects, including exencephaly, encephalocele, and rib abnormalities (Doherty et al., 1982; Frakes et al., 1986; Tewe and Maner, 1981b; Willhite, 1982).

Reference doses (RfDs) have been calculated for subchronic and chronic oral exposure to cyanide and several cyanide compounds (EPA, 1990a-e; 1991a-e). The values, derived from a single study, are based on a no-observed-adverse-effect level (NOAEL) of 10.8 mg/kg/day for cyanide in a 2-year dietary study with rats (Howard and Hanzal, 1955). The subchronic and chronic oral RfDs are 0.02 mg/kg/day for cyanide; 0.04 mg/kg/day for sodium cyanide, calcium cyanide, and cyanogen; 0.05 mg/kg/day for potassium cyanide, chlorine cyanide, and zinc cyanide; 0.1 mg/kg/day for silver cyanide; and 0.2 mg/kg/day for potassium silver cyanide. Data were insufficient to derive a reference concentration (RfC) for cyanide.

No suitable cancer bioassays or epidemiological studies are available to assess the carcinogenicity of cyanide. Therefore, EPA (1991b) has placed cyanide in weight-of-evidence group D, not classifiable as to human carcinogenicity.

Nitrates

Nitrates are produced by natural biological and physical oxidations and therefore are ubiquitous in the environment (Ridder and Oehme 1974). Most of the excess nitrates in the environment originate from inorganic chemicals manufactured for agriculture. Organic molecules containing nitrate groups are manufactured primarily for explosives or for their pharmacological effects (Stokinger 1982). Exposure to inorganic nitrates is primarily through food and drinking water, whereas exposure to organic nitrates can occur orally, dermally, or by respiration (Stokinger 1978). The primary toxic effects of the inorganic nitrate ion (NO_3^-) result from its reduction to nitrite (NO_2^-) by microorganisms in the upper gastrointestinal tract (Johnson and Kross 1990, Bouchard et al. 1992). Nitrite ions can also be produced with organic nitrate exposure; however, the primary effect of organic nitrate intake is thought to be dependent on the production of an active nitric oxide (NO-) radical (Waldman and Murad 1987). Organic nitrates are metabolized in the liver resulting in an increase in blood nitrites (Murad 1990). Nitrates and nitrites are excreted primarily in the urine as nitrates (Hartman 1982).

The primary toxic effect of inorganic nitrates is the oxidation of the iron in hemoglobin by excess nitrites forming methemoglobin. Infants less than 6 months old comprise the most sensitive population (Hartman 1982, Bouchard et al. 1992). Epidemiological studies have shown that baby formula made with drinking water containing nitrate nitrogen levels over 10 mg/L can result in methemoglobinemia, especially in infants less than 2 months of age. No cases of methemoglobinemia were reported with drinking water nitrate nitrogen levels of 10 mg/L or less (Bosch et al. 1950, Walton 1951, Shuval and Gruener 1972). A secondary target for inorganic nitrate toxicity is the cardiovascular system. Nitrate intake can also result in a vasodilatory effect, which can complicate the anoxia resulting from methemoglobinemia (Ridder and Oehme 1974). Decreased motor activity was reported in mice given up to 2000 mg nitrite/L in drinking water, and persistent changes in EEG recordings were observed in rats exposed to 100 to 2000 mg nitrite/L in drinking water. However, exposure of rats to 3000 mg nitrite/L in drinking water for 2 years did not result in any gross or microscopic changes in brain tissue. The data indicate that these central nervous system effects are not related to methemoglobin levels (Shuval and Gruener 1972).

The importance of the primary and secondary targets are reversed with organic nitrates, several of which have long been used for their vasodilatory effects in the treatment of angina pectoris in humans (Murad 1990). Large doses of organic nitrates, however, can also produce methemoglobinemia (Andersen and Mehl 1973). Epidemiological studies have shown that chronic or subchronic exposure to organic nitrates results in the development of tolerance to the cardiovascular effects of these compounds. This apparent biocompensation has caused serious cardiac problems in munitions workers exposed to organic nitrates when they are suddenly removed from the source of exposure (Carmichael & Lieben 1963).

An epidemiological study correlated the number of congenital malformations of the central nervous system and musculoskeletal system of babies with the amount of inorganic nitrate in the mother's drinking water (Dorsch et al. 1984). Other studies, however, do not support these associations, and the presence of unidentified teratogenic factors in the environment could not be ruled out. Inorganic nitrate and nitrite have been tested for teratogenicity in rats, guinea pigs, mice, hamsters, and rabbits. No teratogenic responses were reported; however, fetotoxicity attributed to maternal methemoglobinemia was observed at high doses (4000 mg nitrate/L in drinking water) (Sleight and Atallah 1968, Shuval and Gruener 1972, FDA 1972a, b, c).

A Reference Dose (RfD) of 1.60 mg/kg/day (nitrate nitrogen) for chronic oral exposure was calculated from a NOAEL of 10 mg/L and a LOAEL of 11-20 mg/L in drinking water, based on clinical signs of methemoglobinemia in 0-3-month-old infants (Bosch et al. 1950, Walton 1951). It is important to note, however, that the effect was documented in the most sensitive human population so no uncertainty or modifying factors were used (EPA 1994).

The possible carcinogenicity of nitrate depends on the conversion of nitrate to nitrite and the reaction of nitrite with secondary amines, amides, and carbamates to form N-nitroso compounds that are carcinogenic (Bouchard et al. 1992). Experiments with rats have shown that when given both components, nitrite and heptamethyleneimine, in drinking water, an increase in the incidence of tumors occurs (Taylor and Lijinsky 1975). Human epidemiological studies, however, have yielded conflicting evidence. Positive correlations between the concentration of nitrate in drinking water and the incidence of stomach cancer were reported in Columbia and Denmark (Cuello et al. 1976, Fraser et al. 1980). However, studies in the United Kingdom and other countries have failed to show any correlation between nitrate levels and cancer incidence (Forman 1985, Al-Dabbagh et al. 1986, Croll and Hayes 1988). Nitrate has not been classified as to its carcinogenicity by the EPA, although it is under review (EPA 1994).

Selenium

Selenium is an essential trace element important in many biochemical and physiological processes including the biosynthesis of coenzyme Q (a component of mitochondrial electron transport systems), regulation of ion fluxes across membranes, maintenance of the integrity of keratins, stimulation of antibody synthesis, and activation of glutathione peroxidase (an enzyme involved in preventing oxidative damage to cells). Recommended human dietary allowances (average daily intake) for selenium are as follows: infants up to 1 year, 10-15 µg; children 1-10 years, 20-30 µg; adult males 11-51+ years, 40-70 µg; adult females 11-51+ years, 45-55 µg; pregnant or lactating women, 65-75 µg. There appears to be a relatively narrow range between levels of selenium intake resulting in deficiency and those causing toxicity.

Selenium occurs in several valence states: -2 (hydrogen selenide, sodium selenide, dimethyl selenium, trimethyl selenium, and selenoamino acids such as selenomethionine; 0 (elemental selenium); +4 (selenium dioxide, selenious acid, and sodium selenite); and +6 (selenic acid and sodium selenate). Toxicity of selenium varies with valence state and water solubility of the compound in which it occurs. The latter can affect gastrointestinal absorption rates.

Gastrointestinal absorption in animals and humans for various selenium compounds ranges from about 44% to 95% of the ingested dose (Thomson and Stewart, 1974; Bopp et al., 1982; Thomson, 1974). Respiratory tract absorption rates of 97% and 94% for aerosols of selenious acid have been reported for dogs and rats, respectively (Weissman et al., 1983; Medinsky et al., 1981). Selenium is found in all tissues of the body; highest concentrations occur in the kidney, liver, spleen, and pancreas (Schroeder and Mitchener, 1971a; Schroeder and Mitchener, 1972; Jacobs and Forst, 1981a; Julius et al., 1983; Shamberger, 1984; Echevarria et al., 1988). Excretion is primarily via the urine (0-15 g/L); however, excretory products can also be found in the feces, sweat, and in expired air.

In humans, acute oral exposures can result in excessive salivation, garlic odor to the breath, shallow breathing, diarrhea, pulmonary edema, and death (Civil and McDonald, 1978; Carter, 1966; Koppel et al., 1986). Other reported signs and symptoms of acute selenosis include tachycardia, nausea, vomiting, abdominal pain, abnormal liver function, muscle aches and pains, irritability, chills, and tremors. Acute toxic effects observed in animals include pulmonary congestion, hemorrhages and edema, convulsions, altered blood chemistry (increased hemoglobin and hematocrit); liver congestion; and congestion and

hemorrhage of the kidneys (Smith et al., 1937; Anderson and Moxon, 1942; Hopper et al., 1985).

General signs and symptoms of chronic selenosis in humans include loss of hair and nails, acropachia (clubbing of the fingers), skin lesions (redness, swelling, blistering, and ulcerations), tooth decay (mottling, erosion and pitting), and nervous system abnormalities attributed to polyneuritis (peripheral anesthesia, acroparaethesia, pain in the extremities, hyperreflexia of the tendon, numbness, convulsions, paralysis, motor disturbances, and hemiplegia). In domesticated animals, subchronic and chronic oral exposures can result in loss of hair, malformed hooves, rough hair coat, and nervous system abnormalities (impaired vision and paralysis). Damage to the liver and kidneys and impaired immune responses have been reported to occur in rodents following subchronic and/or chronic oral exposures (Ganther & Baumann, 1962; Beems & van Beek, 1985; NCI, 1980a; Tinsley et al., 1967; Harr et al., 1967; Schroeder, 1967).

Selenium is teratogenic in birds and possibly also in domesticated animals (pigs, sheep, and cattle), but evidence of teratogenicity in humans and laboratory animals is lacking (ASTDR, 1989). However, adverse reproductive and developmental effects (decreased rates of conception, increased rates of fetal resorption, and reduced fetal body weights) have been reported for domesticated and laboratory animals (Harr & Muth, 1972: Wahlstrom & Olson, 1959; Schroeder & Mitchener, 1971b).

The Reference Dose (RfD) for chronic oral exposures is 0.005 mg/kg/day for both selenium and selenious acid (EPA, 1992a, 1992b). The subchronic RfDs for these compounds are the same as the chronic RfDs (EPA, 1992c).

In humans, inhalation of selenium or selenium compounds primarily affects the respiratory system. Dusts of elemental selenium and selenium dioxide can cause irritation of the skin and mucous membranes of the nose and throat, coughing, nosebleed, loss of sense of smell, dyspnea, bronchial spasms, bronchitis, and chemical pneumonia (Clinton, 1947; Hamilton, 1949). Other signs and symptoms following acute inhalation exposures include lacrimation, irritation and redness of the eyes, gastrointestinal distress (nausea and vomiting), depressed blood pressure, elevated pulse rate, headaches, dizziness, and malaise (ATSDR, 1989). In animals, acute inhalation exposures also result in severe respiratory effects including edema, hemorrhage, and interstitial pneumonitis (Hall et al., 1951; Dudley and Miller, 1937) as well as in splenic damage (congestion, fissuring red pulp, and increased polymorphonuclear leukocytes) and liver congestion and mild central atrophy (Hall et al., 1951). Information on toxicity of selenium in humans and animals following chronic inhalation exposures is not available, and subchronic and chronic inhalation Reference Concentrations have not been derived.

Epidemiologic studies in humans havation between chronic oral exposures to selenium and an increased incidence of death due to neoplasms. Some studies have indicated that selenium may have anti-neoplastic properties (see Whanger, 1983; Hocman, 1988). In studies on laboratory animals, selenites or selenates have not been found to be carcinogenic; however, selenium sulfide produced a significant increase in the incidence of hepatocellular carcinomas in male and female rats and in female mice and a significant increase in alveolar/bronchiolar carcinomas and adenomas in female mice following chronic oral exposures (NCI, 1980c). EPA has placed selenium and selenious acid in Group D, not classifiable as to carcinogenicity in humans (U.S. EPA, 1992a and 1992b), while selenium sulfide is placed in Group B2, probable human carcinogen (U.S. EPA, 1992d). Quantitative data are, however, insufficient to derive a slope factor for selenium sulfide. Pertinent data regarding the potential carcinogenicity of selenium by the inhalation route in humans or animals were not located in the available literature.

Zinc

Zinc is used primarily in galvanized metals and metal alloys, but zinc compounds also have wide commercial applications as chemical intermediates, catalysts, pigments, vulcanization activators and accelerators in the rubber industry, UV stabilizers, and supplements in animal feeds and fertilizers. They are also used in rayon manufacture, smoke bombs, soldering fluxes, mordants for printing and dyeing, wood preservatives, mildew inhibitors, deodorants, antiseptics, and astringents (Lloyd, 1984; ATSDR, 1989). In addition, zinc phosphide is used as a rodenticide.

Zinc is an essential element with recommended daily allowances ranging from 5 mg for infants to 15 mg for adult males (NRC, 1989).

Gastrointestinal absorption of zinc is variable (20-80%) and depends on the chemical compound as well as on zinc levels in the body and dietary concentrations of other nutrients (U.S. EPA, 1984). In individuals with normal zinc levels in the body, gastrointestinal absorption is 20-30% (ATSDR, 1989). Information on pulmonary absorption is limited and complicated by the potential for gastrointestinal absorption due to mucociliary clearance from the respiratory tract and subsequent swallowing. Zinc is present in all tissues with the highest concentrations in the prostate, kidney, liver, heart, and pancreas. Zinc is a vital component of many metalloenzymes such as carbonic anhydrase, which regulates CO2 exchange (Stokinger, 1981). Homeostatic mechanisms involving metallothionein in the mucosal cells of the gastrointestinal tract regulate zinc absorption and excretion (ATSDR, 1989).

In humans, acutely toxic oral doses of zinc cause nausea, vomiting, diarrhea, and abdominal cramps and in some cases gastric bleeding (Elinder, 1986; Moore, 1978; ATSDR, 1989). Ingestion of zinc chloride can cause burning in the mouth and throat, vomiting, pharyngitis, esophagitis, hypocalcemia, and elevated amylase activity indicative of pancreatitis (Chobanian, 1981). Zinc phosphide, which releases phosphine gas under acidic conditions in the stomach, can cause vomiting, anorexia, abdominal pain, lethargy, hypotension, cardiac arrhythmias, circulatory collapse, pulmonary edema, seizures, renal damage, leukopenia, and coma and death in days to weeks (Mack, 1989). The estimated fatal dose is 40 mg/kg. Animals dosed orally with zinc compounds develop pancreatitis, gastrointestinal and hepatic lesions, and diffuse nephrosis.

Gastrointestinal upset has also been reported in individuals taking daily dietary zinc supplements for up to 6 weeks (Samman and Roberts, 1987). There is also limited evidence that the human immune system may be impaired by subchronic exposures (Chandra, 1984). In animals, gastrointestinal and hepatic lesions, (Allen et al., 1983; Brink et al., 1959); pancreatic lesions (Maita et al., 1981; Drinker et al., 1927a); anemia (ATSDR, 1989; Fox and Jacobs, 1986; Maita et al., 1981); and diffuse nephrosis (Maita et al., 1981; Allen et al., 1983) have been observed following subchronic oral exposures.

Chronic oral exposures to zinc have resulted in hypochromic microcytic anemia associated with hypoceruloplasminemia, hypocupremia, and neutropenia in some individuals (Prasad et al., 1978; Porter et al., 1977). Anemia and pancreatitis were the major adverse effects observed in chronic animal studies (Aughey et al., 1977; Drinker et al., 1927a; Walters and Roe, 1965; Sutton and Nelson, 1937). Teratogenic effects have not been seen in animals exposed to zinc; however, high oral doses can affect reproduction and fetal growth (Ketcheson et al., 1969; Schlicker & Cox 1967, 1968; Sutton & Nelson, 1937).

The reference dose for chronic oral exposure to zinc is under review by EPA; the currently accepted RfD for both subchronic and chronic exposures is 0.2 mg/kg/day based on clinical data demonstrating zinc-induced copper deficiency and anemia in patients taking zinc sulfate for the treatment of sickle cell anemia (EPA, 1992). The chronic oral RfD for zinc phosphide is 0.0003 mg/kg/day (EPA, 1991a), and the subchronic RfD is 0.003 mg/kg/day (EPA, 1992).

Under occupational exposure conditions, inhalation of zinc compounds (mainly zinc oxide fumes) can result in a condition identified as "metal fume fever", which is characterized by nasal passage irritation, cough, rales, headache, altered taste, fever, weakness, hyperpnea, sweating, pains in the legs and chest, leukocytosis, reduced lung volume, and decreased diffusing capacity of carbon monoxide (ATSDR, 1989; Bertholf, 1988). Inhalation of zinc chloride can result in nose and throat irritation, dyspnea, cough, chest pain, headache, fever, nausea and vomiting, and respiratory disorders such as pneumonitis and pulmonary fibrosis (ITII, 1988; ATSDR, 1989; Nemery, 1990). Pulmonary inflammation and changes in lung function have also been observed in inhalation studies on animals (Amur et al.,1982; Lam et al., 1985; Drinker & Drinker, 1928).

Although "metal fume fever" occurs in occupationally exposed workers, it is primarily an acute and reversible effect that is unlikely to occur under chronic exposure conditions when zinc air concentrations are less than 8-12 mg/m3 (ATSDR, 1989). Gastrointestinal distress, as well as enzyme changes indicative of liver dysfunction, have also been reported in workers occupationally exposed to zinc (NRC, 1979; Stokinger, 1981; EPA, 1991a; Guja, 1973; Badawy et al., 1987a); however, it is unclear as to what extent these effects might have been caused by pulmonary clearance, and subsequent gastrointestinal absorption. Consequently, there are no clearly defined toxic effects that can be identified as resulting specifically from pulmonary absorption following chronic low level inhalation exposures. Animal data for chronic inhalation exposures are not available. An inhalation reference concentration has not been

derived for zinc or zinc compounds (EPA, 1992).

No case studies or epidemiologic evidence has been presented to suggest that zinc is carcinogenic in humans by the oral or inhalation route (EPA, 1991a). In animal studies, zinc sulfate in drinking water or zinc oleate in the diet of mice for a period of one year did not result in a statistically significant increase in hepatomas, malignant lymphomas, or lung adenomas (Walters & Roe, 1965); however, in a 3-year, 5-generation study on tumor-resistant and tumor-susceptible strains of mice, exposure to zinc in drinking water resulted in increased frequencies of tumors from the F0 to the F4 generation in the tumor-resistant strain (from 0.8 to 25.7%, vs. 0.0004% in the controls), and higher tumor frequencies in two tumor-susceptible strains (43.4% & 32.4% vs. 15% in the controls) (Halme, 1961).

Zinc is placed in weight-of-evidence Group D, not classifiable as to human carcinogenicity due to inadequate evidence in humans and animals (EPA, 1991a).