

## 6. POTENTIAL FOR HUMAN EXPOSURE

### 6.1 OVERVIEW

Manganese has been identified in at least 869 of the 1,699 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 2008). However, the number of sites evaluated for manganese is not known. The frequency of these sites can be seen in Figure 6-1. Of these sites, 861 are located within the United States, 5 are located in the Commonwealth of Puerto Rico, 2 are located in the Virgin Islands, and 1 is located in Guam (not shown).

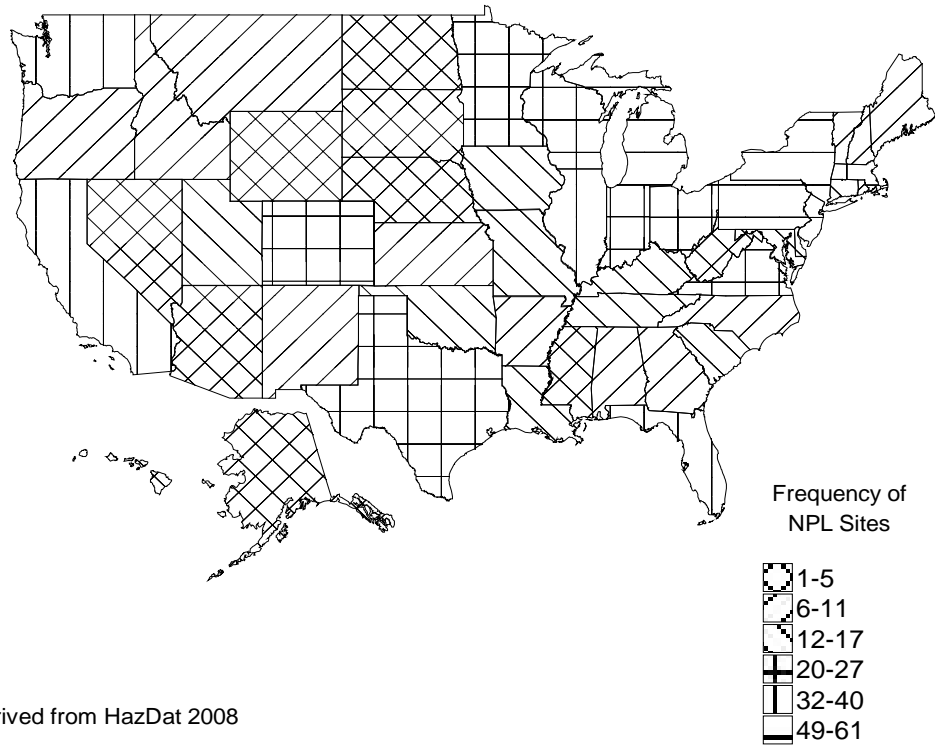
Manganese is ubiquitous in the environment, and human exposure arises from both natural and anthropogenic activities. It occurs naturally in more than 100 minerals with background levels in soil ranging from 40 to 900 mg/kg, with an estimated mean background concentration of 330 mg/kg (Barceloux 1999). Manganese is released to the environment from industrial emissions, fossil fuel combustion, and erosion of manganese-containing soils. Volcanic eruptions can also contribute to levels of manganese in air. Almost 80% of industrial emissions of manganese are attributable to iron and steel production facilities (EPA 2003a). Power plant and coke oven emissions contribute about 20% (EPA 2003a). Manganese may also be released to the environment through the use of MMT as a gasoline additive. Thus, all humans are exposed to manganese, and manganese is a normal component of the human body.

Background levels of manganese in the atmosphere vary widely depending on the proximity of point sources, such as ferroalloy production facilities, coke ovens, and power plants. The estimated average background concentration of manganese in urban areas is approximately 40 ng/m<sup>3</sup>, based on measurements obtained in 102 U.S. cities (EPA 2003a; WHO 2004b). Concentrations near source dominated areas were reported to range from 220 to 300 ng/m<sup>3</sup> (WHO 2004b) and rural/remote levels are typically under 10 ng/m<sup>3</sup> (Sweet et al. 1993). Manganese occurs naturally in surface water and groundwater. A median dissolved manganese concentration of 24 µg/L in samples from 286 U.S. rivers and streams was reported (Smith et al. 1987). Natural concentrations of manganese in seawater reportedly range from 0.4 to 10 µg/L (EPA 1984).

The general population is exposed to manganese primarily through food intake. The World Health Organization (WHO) estimates that adults consume between 0.7 and 10.9 mg of manganese per day in the diet, with higher intakes for vegetarians who may consume a larger proportion of manganese-rich nuts, grains, and legumes in their diet as compared to non-vegetarians in the general population (WHO 2004b).

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**Figure 6-1. Frequency of NPL Sites with Manganese Contamination**



Derived from HazDat 2008

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Manganese intake from drinking water is substantially lower than intake from food. Using a median drinking-water level of 10 µg/L and an assumption that the average adult drinks 2 L of water/day, an average intake of approximately 0.020 mg/day was estimated (WHO 2004b). Exposure to manganese from air is considered negligible as compared to intake from diet; however, persons in certain occupations may be exposed to much higher levels than the general public (see Section 6.7).

Manganese adsorbed to particulate matter in air can be classified by the size of the particles. Air concentrations can be reported as total suspended particulate matter (TSP), respirable particulates, and fine particulates. In this document, manganese adsorbed to particulate matter <10 microns in aerodynamic diameter is referred to as PM<sub>10</sub>. The EPA has further divided these tiny particles into "fine" particles of ≤2.5 microns (PM<sub>2.5</sub>) and "coarse" particles of between 2.5 and 10 microns.

## 6.2 RELEASES TO THE ENVIRONMENT

The Toxics Release Inventory (TRI) data should be used with caution because only certain types of facilities are required to report (EPA 2005). This is not an exhaustive list. Manufacturing and processing facilities are required to report information to the TRI only if they employ 10 or more full-time employees; if their facility is included in Standard Industrial Classification (SIC) Codes 10 (except 1011, 1081, and 1094), 12 (except 1241), 20–39, 4911 (limited to facilities that combust coal and/or oil for the purpose of generating electricity for distribution in commerce), 4931 (limited to facilities that combust coal and/or oil for the purpose of generating electricity for distribution in commerce), 4939 (limited to facilities that combust coal and/or oil for the purpose of generating electricity for distribution in commerce), 4953 (limited to facilities regulated under RCRA Subtitle C, 42 U.S.C. section 6921 et seq.), 5169, 5171, and 7389 (limited S.C. section 6921 et seq.), 5169, 5171, and 7389 (limited to facilities primarily engaged in solvents recovery services on a contract or fee basis); and if their facility produces, imports, or processes ≥25,000 pounds of any TRI chemical or otherwise uses >10,000 pounds of a TRI chemical in a calendar year (EPA 2005).

According to the Toxics Release Inventory (TRI), in 2006, a total of 27,094,361 pounds (12,290 metric tons) of manganese was released to the environment from 2,040 large processing facilities (TRI06 2008). An additional 199,804,760 pounds (90,630 metric tons) of manganese compounds were released from 1,748 facilities. Tables 6-1, and 6-2 list the amount of manganese and manganese related compounds, respectively, that were released from all of the facilities that manufacture or process manganese to each medium within each state in 2006 (TRI06 2008). The TRI data should be used with caution because only

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**Table 6-1. Releases to the Environment from Facilities that Produce, Process, or Use Manganese<sup>a</sup>**

State <sup>c</sup>	RF <sup>d</sup>	Reported amounts released in pounds per year <sup>b</sup>							
		Air <sup>e</sup>	Water <sup>f</sup>	UI <sup>g</sup>	Land <sup>h</sup>	Other <sup>i</sup>	Total release		
							On-site <sup>j</sup>	Off-site <sup>k</sup>	On- and off-site
AK	1	440	0	0	43,596	0	440	43,596	44,036
AL	46	14,003	2,594	0	311,137	138	39,354	288,519	327,873
AR	28	14,187	202	0	40,397	11,970	14,345	52,410	66,756
AZ	23	1,249	86	0	988,345	5	989,128	557	989,685
CA	52	5,905	446	0	266,849	508	190,728	82,980	273,708
CO	13	673	61	0	14,367	0	686	14,415	15,101
CT	9	6	7	0	550	76	6	633	639
DE	1	0	0	0	14	0	0	14	14
FL	28	1,387	304	0	19,193	10	1,390	19,504	20,894
GA	43	3,256	2,559	0	47,464	10	3,618	49,671	53,289
HI	1	0	0	3	33	0	3	33	36
IA	75	26,407	3,501	0	87,121	387,890	52,459	452,460	504,919
ID	2	206	0	0	1,159,780	0	1,159,986	0	1,159,986
IL	113	16,708	3,288	723	725,350	2,914	17,866	731,117	748,983
IN	142	31,353	5,835	0	487,103	23,334	32,009	515,617	547,626
KS	28	7,838	290	0	1,045,331	159,947	1,013,666	199,740	1,213,406
KY	57	8,743	1,431	0	93,655	16,068	10,660	109,238	119,898
LA	35	11,553	7,316	0	64,028	8,095	17,306	73,686	90,993
MA	16	453	108	0	8,497	737	465	9,329	9,795
MD	12	148	57	0	759	6	148	822	970
ME	5	15	68	0	81	420	20	564	584
MI	101	13,587	3,774	0	418,116	6,311	42,212	399,577	441,789
MN	43	4,752	184	0	86,817	394	4,757	87,390	92,148
MO	59	10,907	1,718	0	20,096	10,164	21,102	21,783	42,885
MS	30	4,955	345	0	67,256	0	8,037	64,518	72,555
MT	3	0	0	0	78,546	0	78,546	0	78,546
NC	58	2,225	140	0	30,064	2,830	2,250	33,009	35,260
ND	7	1,592	7	0	531	0	1,600	530	2,130
NE	17	1,723	307	0	49,079	635	1,770	49,974	51,744
NH	6	87	17,300	0	6,245	0	17,387	6,245	23,632
NJ	10	583	29	0	11,324	0	600	11,336	11,936
NM	1	500	0	0	1,699	0	2,199	0	2,199
NV	11	135	0	0	94,028	0	94,163	0	94,163
NY	43	5,555	8,766	0	320,476	1,182	7,932	328,047	335,979
OH	166	39,047	120,301	2	11,591,066	2,463,430	10,515,399	3,698,447	14,213,846
OK	77	4,384	325	0	62,595	0	10,352	56,952	67,304

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**Table 6-1. Releases to the Environment from Facilities that Produce, Process, or Use Manganese<sup>a</sup>**

State <sup>c</sup>	RF <sup>d</sup>	Reported amounts released in pounds per year <sup>b</sup>							
		Air <sup>e</sup>	Water <sup>f</sup>	UI <sup>g</sup>	Land <sup>h</sup>	Other <sup>i</sup>	Total release		
							On-site <sup>j</sup>	Off-site <sup>k</sup>	On- and off-site
OR	14	349	0	0	528	0	349	528	877
PA	152	43,170	11,360	0	1,000,943	59,278	375,255	739,495	1,114,751
PR	4	2	0	0	0	16	2	16	18
RI	2	0	0	0	0	0	0	0	0
SC	48	6,328	536	0	85,146	29,788	46,481	75,318	121,799
SD	19	8,727	5	0	26,330	0	16,460	18,602	35,062
TN	62	49,422	1,924	0	348,780	3,943	99,022	305,047	404,069
TX	121	53,231	22,104	5	171,602	624	155,306	92,260	247,566
UT	17	2,559	13	0	747,419	0	738,680	11,311	749,992
VA	29	2,033	608	0	161,608	22,211	2,356	184,105	186,461
WA	26	5,729	42,723	0	139,920	539	51,480	137,431	188,911
WI	171	20,115	8,948	0	2,137,917	12,870	58,935	2,120,915	2,179,850
WV	8	26	1	0	38,324	14	24,026	14,338	38,364
WY	5	193	0	0	71,139	5	71,332	5	71,337
Total	2,040	426,449	269,573	733	23,171,244	3,226,362	15,992,276	11,102,084	27,094,361

<sup>a</sup>The TRI data should be used with caution since only certain types of facilities are required to report. This is not an exhaustive list. Data are rounded to nearest whole number.

<sup>b</sup>Data in TRI are maximum amounts released by each facility.

<sup>c</sup>Post office state abbreviations are used.

<sup>d</sup>Number of reporting facilities.

<sup>e</sup>The sum of fugitive and point source releases are included in releases to air by a given facility.

<sup>f</sup>Surface water discharges, waste water treatment-(metals only), and publicly owned treatment works (POTWs) (metal and metal compounds).

<sup>g</sup>Class I wells, Class II-V wells, and underground injection.

<sup>h</sup>Resource Conservation and Recovery Act (RCRA) subtitle C landfills; other on-site landfills, land treatment, surface impoundments, other land disposal, other landfills.

<sup>i</sup>Storage only, solidification/stabilization (metals only), other off-site management, transfers to waste broker for disposal, unknown

<sup>j</sup>The sum of all releases of the chemical to air, land, water, and underground injection wells.

<sup>k</sup>Total amount of chemical transferred off-site, including to POTWs.

RF = reporting facilities; UI = underground injection

Source: TRI06 2008 (Data are from 2006)

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**Table 6-2. Releases to the Environment from Facilities that Produce, Process, or Use Manganese Compounds<sup>a</sup>**

State <sup>c</sup>	RF <sup>d</sup>	Reported amounts released in pounds per year <sup>b</sup>							
		Air <sup>e</sup>	Water <sup>f</sup>	UI <sup>g</sup>	Land <sup>h</sup>	Other <sup>i</sup>	Total release		On- and off-site
							On-site <sup>j</sup>	Off-site <sup>k</sup>	
AK	5	25,328	750	0	845,025	6,280	871,103	6,280	877,383
AL	68	178,265	875,746	0	5,663,753	2,104,302	5,163,681	3,658,386	8,822,066
AR	50	19,489	441,421	0	1,480,122	989,320	1,844,948	1,085,404	2,930,352
AZ	14	4,047	410,010	0	1,971,519	28,019	2,113,096	300,499	2,413,595
CA	37	7,828	1,059	0	200,104	193	124,029	85,154	209,183
CO	24	5,126	11,022	0	2,246,268	9,100	714,772	1,556,744	2,271,516
CT	10	4,362	1,883	0	60,665	7,840	5,865	68,885	74,750
DC	3	0	4,347	0	6,000	0	10,347	0	10,347
DE	11	4,334	39,657	0	4,092,502	28,305	116,524	4,048,274	4,164,798
FL	37	18,745	202,776	0	3,397,056	33,456	3,360,138	291,895	3,652,032
GA	61	47,377	773,805	0	2,836,912	7,944	3,330,409	335,629	3,666,038
HI	1	38	0	0	26,872	0	38	26,872	26,910
IA	42	41,786	22,496	0	840,340	364,964	227,129	1,042,457	1,269,586
ID	12	841	190,590	0	14,737,870	130	14,679,285	250,146	14,929,431
IL	84	96,010	35,152	0	9,033,549	259,391	5,687,449	3,736,654	9,424,103
IN	79	192,010	93,426	1,900	18,418,915	1,671,366	7,130,422	13,247,195	20,377,618
KS	28	13,257	652	250	244,236	250	220,445	38,200	258,645
KY	47	68,576	147,644	0	1,528,078	119,954	1,660,270	203,982	1,864,252
LA	29	20,544	387,475	0	6,371,936	1,408	6,476,794	304,569	6,781,363
MA	17	1,436	1,331	0	100,634	32,790	61,732	74,459	136,191
MD	26	21,618	90,703	25,571	2,166,200	125,137	2,233,047	196,182	2,429,229
ME	10	2,117	331,758	0	677,049	18,587	792,798	236,713	1,029,511
MI	67	81,060	99,058	2,000	2,612,249	43,241	1,138,887	1,698,721	2,837,608
MN	31	14,598	256,905	0	1,587,019	23,337	1,591,056	290,802	1,881,858
MO	46	13,796	51,704	0	708,194	3,865	418,616	358,943	777,558
MS	33	15,064	288,780	8,506,700	9,204,141	6,158	17,937,765	83,078	18,020,843
MT	9	9,747	29,912	0	1,981,375	46,473	1,849,854	217,652	2,067,506
NC	63	19,836	213,027	0	2,011,149	1,177,640	1,796,233	1,625,420	3,421,652
ND	9	15,474	16,333	0	2,480,954	15,612	1,479,091	1,049,282	2,528,373
NE	22	21,131	500	0	153,009	612,303	161,275	625,668	786,943
NH	1	134	0	0	4,770	0	4,034	870	4,904
NJ	15	3,032	10,005	0	472,763	13,484	31,191	468,093	499,284
NM	6	3,916	1,300	0	828,935	0	834,151	0	834,151
NV	10	12,998	3,611	0	10,169,735	0	9,260,544	925,800	10,186,344
NY	26	4,650	81,183	0	584,743	26,423	261,995	435,004	697,000
OH	138	430,169	642,313	30,514	11,063,512	597,498	6,580,637	6,183,369	12,764,006
OK	32	11,618	48,634	0	750,631	508,644	531,270	788,257	1,319,527

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**Table 6-2. Releases to the Environment from Facilities that Produce, Process, or Use Manganese Compounds<sup>a</sup>**

State <sup>c</sup>	RF <sup>d</sup>	Air <sup>e</sup>	Water <sup>f</sup>	UI <sup>g</sup>	Land <sup>h</sup>	Other <sup>i</sup>	Reported amounts released in pounds per year <sup>b</sup>		
							On-site <sup>j</sup>	Off-site <sup>k</sup>	On- and off-site
OR	23	8,314	144,618	0	2,142,046	323,198	2,004,367	613,809	2,618,176
PA	134	72,526	184,992	0	10,063,261	340,932	5,031,291	5,630,420	10,661,710
PR	6	8,490	401	0	282	0	8,886	287	9,173
RI	1	103	31	0	169	0	134	169	303
SC	44	27,819	378,152	0	6,760,363	6,148	2,292,815	4,879,668	7,172,483
SD	7	38	0	0	27,700	0	22,038	5,700	27,738
TN	53	50,882	293,774	0	16,727,879	38,873	16,041,491	1,069,917	17,111,408
TX	104	66,756	185,907	0	6,283,703	94,244	6,150,141	480,469	6,630,610
UT	20	6,687	761	0	1,271,573	13	1,264,871	14,163	1,279,034
VA	35	19,359	183,002	0	1,522,896	4,563	729,421	1,000,399	1,729,820
VT	1	0	0	0	0	0	0	0	0
WA	25	3,227	187,304	0	814,927	16,167	808,095	213,530	1,021,626
WI	60	23,609	116,184	0	2,533,313	95,152	364,876	2,403,382	2,768,258
WV	26	133,302	13,412	0	1,700,100	24,034	1,325,047	545,801	1,870,848
WY	6	8,493	1,327	0	647,297	0	617,518	39,599	657,117
<b>Total</b>	<b>1,748</b>	<b>1,859,959</b>	<b>7,496,834</b>	<b>8,566,935</b>	<b>172,054,292</b>	<b>9,826,740</b>	<b>137,361,909</b>	<b>62,442,851</b>	<b>199,804,760</b>

<sup>a</sup>The TRI data should be used with caution since only certain types of facilities are required to report. This is not an exhaustive list. Data are rounded to nearest whole number.

<sup>b</sup>Data in TRI are maximum amounts released by each facility.

<sup>c</sup>Post office state abbreviations are used.

<sup>d</sup>Number of reporting facilities.

<sup>e</sup>The sum of fugitive and point source releases are included in releases to air by a given facility.

<sup>f</sup>Surface water discharges, waste water treatment-(metals only), and publicly owned treatment works (POTWs) (metal and metal compounds).

<sup>g</sup>Class I wells, Class II-V wells, and underground injection.

<sup>h</sup>Resource Conservation and Recovery Act (RCRA) subtitle C landfills; other on-site landfills, land treatment, surface impoundments, other land disposal, other landfills.

<sup>i</sup>Storage only, solidification/stabilization (metals only), other off-site management, transfers to waste broker for disposal, unknown

<sup>j</sup>The sum of all releases of the chemical to air, land, water, and underground injection wells.

<sup>k</sup>Total amount of chemical transferred off-site, including to POTWs.

RF = reporting facilities; UI = underground injection

Source: TRI06 2008 (Data are from 2006)

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certain types of facilities are required to report. This is not an exhaustive list. Also, because these data reflect past releases, they may not be representative of current releases at these facilities.

Manganese may also be emitted to the environment through the use of gasoline that contains MMT; however, no data on the amount of MMT that is currently being used in gasoline in the United States were located. No data for releases of mangafodipir to the environment were found. Because mangafodipir is a compound used exclusively in a clinical environment, it is not expected to be released to the environment and will not be discussed in subsequent sections concerning fate and transport.

**6.2.1 Air**

Estimated releases of 426,449 pounds (193 metric tons) of manganese to the atmosphere from 2,040 domestic manufacturing and processing facilities in 2006, accounted for about 1.6% of the estimated total environmental releases from facilities required to report to the TRI (TRI06 2008).

Estimated releases of 1,859,959 pounds (844 metric tons) of manganese compounds to the atmosphere from 1,748 domestic manufacturing and processing facilities in 2006, accounted for about 1% of the estimated total environmental releases from facilities required to report to the TRI (TRI06 2008). These releases are summarized in Tables 6-1 and 6-2.

According to data from the National Pollutant Release Inventory (NPRI) maintained by Environment Canada, approximately 273.9 metric tons of manganese were released to air in Canada in 2003 from various industrial sources (Health Canada 2008). The major industrial sources for manganese emissions in Canada were attributed to an iron-ore mine located in Labrador, iron- and steel-related industries, pulp/paper/newsprint mills, fossil fuel electric power generation, and the manufacturing of heating and commercial refrigeration equipment.

Manganese has been identified in air samples collected at 31 of the 869 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2008).

The main sources of manganese release to the air are industrial emissions, combustion of fossil fuels, and reentrainment of manganese-containing soils (EPA 1983c, 1984, 1985c, 1985d, 1987a; Liroy 1983). The principal sources of industrial emissions are ferroalloy production and iron and steel foundries, and the principal sources of combustion emissions are power plants and coke ovens (EPA 1983c, 1985c, 1985d). Atmospheric emissions of manganese and other trace metals from these industrial sources have declined



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over the last 2 decades due to the use of advanced pollution control devices and increased government regulations regarding these emissions (EPA 1984, 1985d).

Windblown erosion of dusts and soils is also an important atmospheric source of manganese. Wallace and Slonecker (1997) estimated that the background contribution of windblown soil to fine particulate atmospheric manganese levels was 1–2 ng/m<sup>3</sup> in the United States and Canada. Volcanic eruptions may also release manganese to the atmosphere (Schroeder et al. 1987).

MMT is a manganese-containing compound used to enhance the octane rating in gasoline. MMT was used as an additive in leaded gasoline until the phase-out of leaded gas in the United States in 1995. It was also used in unleaded gasoline for a short period of time in the late 1970's, but was banned as an additive in unleaded gasoline by EPA in 1977 (EPA 1978, 1979a, 1981). In 1995, the ban on MMT use in unleaded gasoline was lifted, and a court decision ordered EPA to register the product for use as a fuel additive, although testing for health effects continues (EPA 1995a). Analysis of manganese levels in the air indicates that vehicular emissions from MMT containing fuels contributed an average of 13 ng manganese/m<sup>3</sup> in southern California, while vehicular emissions were only responsible for about 3 ng/m<sup>3</sup> in central and northern California (Davis et al. 1988). A survey of ambient air concentrations of fine (PM<sub>2.5</sub>) manganese in rural sites in U.S. national parks and in urban sites in California indicated that from 1988 to 1993, ambient concentrations of manganese ranged from 1 ng/m<sup>3</sup> in rural sites to 3 ng/m<sup>3</sup> in urban sites (Wallace and Slonecker 1997). Part of the increase in fine manganese during this period was considered to be the result of the use of MMT in leaded gasoline. It was estimated that automobile emissions from leaded gasoline were responsible for 37% of the fine manganese levels in California in 1992. In 1994, automobile emissions were estimated to contribute 12% of the fine manganese levels in the atmosphere, as the use of leaded gasoline declined. It has been estimated that if MMT were used in all gasoline, urban air manganese levels would be increased by about 50 ng/m<sup>3</sup> (Cooper 1984; Ter Haar et al. 1975). Other authors have estimated that the increase in atmospheric manganese levels would be <20 ng/m<sup>3</sup> (Lynam et al. 1994).

In Canada, where the use of MMT containing gasoline has been extensive, a 10% per year increase in manganese emission rates from MMT in gasoline since 1981 was estimated (Loranger and Zayed 1994). A positive correlation between atmospheric manganese concentration and traffic density has been observed (Loranger and Zayed 1997a; Loranger et al. 1994a). The principal emission product of MMT combustion is a fine particulate matter (0.1–0.4 µm diameter) consisting of manganese oxide (Egyed and Wood 1996; Ter Haar et al. 1975), manganese phosphate, and some manganese sulfate (Lynam et al.

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1999). The finding of soluble manganese (<0.4 µm) in snow samples obtained close to a highway in Montreal, Canada suggested a possible contamination from mobile sources (Loranger and Zayed 1997a; Loranger et al. 1995). However, it has been difficult to assess the exact contribution of mobile sources to overall contamination from natural and industrial sources because of the physico-chemical characteristics of manganese particulate, environmental factors affecting its dispersion, and the difficulties in distinguishing between mobile sources of manganese and background manganese levels (Loranger and Zayed 1997a; Veysseyre et al. 1998).

Despite the estimated 10% per year increase in manganese emission rates from the use of MMT in gasoline in Canada, atmospheric manganese concentrations in Montreal have remained fairly constant between 1981 and 1990, and have decreased markedly in 1991 and 1992 (Loranger and Zayed 1994). The decline in manganese concentration after 1990 may have been due to a shutdown in 1991 of a ferromanganese plant located near Montreal. Air concentrations are in general below the EPA reference concentration (RfC) of 0.05 µg/m<sup>3</sup> for respirable manganese. However, in 1998, it was observed that some atmospheric concentrations in specific microenvironments with important traffic density were higher than the RfC (Zayed et al. 1999a).

### 6.2.2 Water

Estimated releases of 269,573 pounds (122 metric tons) of manganese to water from 2,040 domestic manufacturing and processing facilities in 2006, accounted for about 1% of the estimated total environmental releases from facilities required to report to the TRI (TRI06 2008). Estimated releases of 7,496,834 pounds (3,401 metric tons) of manganese compounds to water from 1,748 domestic manufacturing and processing facilities in 2006, accounted for about 4% of the estimated total environmental releases from facilities required to report to the TRI (TRI06 2008). These releases are summarized in Tables 6-1 and 6-2.

Manganese has been identified in surface water and groundwater samples collected at 392 and 692, respectively, of the 869 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2008).

Based on comparison to typical background levels of manganese in surface water or groundwater (see Section 6.4.2), it seems likely that some waste sites where manganese is detected contain only natural levels. Although ambient manganese levels are about 200 µg/L in a number of cases, high levels (in

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excess of 1,000 µg/L) have been detected indicating that manganese wastes may lead to significant contamination of water at some sites. For example, at one site in Ohio where "heavy metals" had been disposed, manganese concentrations up to 1,900 µg/L were found in on-site wells (Cooper and Istok 1988). Levels in water at two NPL sites in Missouri ranged from 0.009 to 3.7 µg/L (MDNR 1990). No information is available on the method used to determine these values, so it is not clear whether the data refer to total or dissolved manganese.

**6.2.3 Soil**

Estimated releases of 23,171,244 pounds (10,510 metric tons) of manganese to soil from 2,040 domestic manufacturing and processing facilities in 2006, accounted for about 86% of the estimated total environmental releases from facilities required to report to the TRI (TRI06 2008). Estimated releases of 172,054,292 pounds (78,043 metric tons) of manganese compounds to the soil from 1,748 domestic manufacturing and processing facilities in 2006, accounted for about 86% of the estimated total environmental releases from facilities required to report to the TRI (TRI06 2008). An additional 8,566,935 pounds (3,886 metric tons) were injected underground. These releases are summarized in Tables 6-1 and 6-2.

Manganese deposition to soils from the use of MMT in gasoline was estimated for two sites in Toronto, Canada (Bhuie et al. 2005). Accounting for variables such as annual average daily traffic (AADT) density, fuel consumption, distance traveled by automobiles, and a manganese content of 10 mg/L of gasoline, the annual average manganese contribution to soils from MMT emissions were calculated as 5.73 and 2.47 mg/kg at two sites (Bhuie et al. 2005). These concentrations were considered insignificant when compared to natural background manganese levels (541 and 557 mg/kg ) in soil for these areas.

Manganese has been identified in soil and sediment, samples collected at 355 and 257, respectively, of the 869 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2008).

**6.3 ENVIRONMENTAL FATE****6.3.1 Transport and Partitioning**

Manganese compounds have negligible vapor pressures (see Table 4-2), but may exist in air as suspended particulate matter derived from industrial emissions or the erosion of soils. Manganese-containing particles are mainly removed from the atmosphere by gravitational settling, with large particles tending to

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fall out faster than small particles (EPA 1984). The half-life of airborne particles is usually on the order of days, depending on the size of the particle and atmospheric conditions (Nriagu 1979). Some removal by washout mechanisms such as rain may also occur, although it is of minor significance in comparison to dry deposition (EPA 1984; Turner et al. 1985).

In a study completed by Evans (1989), there were two mechanisms involved in explaining the retention of manganese and other metals in the environment by soil. First, through cation exchange reactions, manganese ions and the charged surface of soil particles form manganese oxides, hydroxides, and oxyhydroxides, which in turn form absorption sites for other metals. Secondly, manganese can be adsorbed to other oxides, hydroxides, and oxyhydroxides through ligand exchange reactions. When the soil solution becomes saturated, these manganese oxides, hydroxides, and oxyhydroxides can precipitate into a new mineral phase and act as a new surface to which other substances can absorb (Evans 1989).

The behavior of heavy metals in the combustion gases of urban waste incinerators was studied by Fernandez et al. (1992). Manganese was detected inside gaseous fly ash particles in the form of oxides and chlorides. When these soluble oxides and chlorides reach environmental media, they can leach out and become mobile (Fernandez et al. 1992).

The transport of manganese in air is largely determined by its particle size. About 80% of the manganese in suspended particulate matter is associated with particles having a mass median aerodynamic diameter (MMAD) of  $<5 \mu\text{m}$  (WHO 1981). The compound's small particle size (approximately 80% with a MMAD  $<5 \mu\text{m}$  and approximately 50% with an MMAD  $<2 \mu\text{m}$ ) favors widespread airborne distribution and is within the respirable range (WHO 1981).

The transport and partitioning of manganese in water is controlled by the solubility of the specific chemical form present, which in turn is determined by pH, Eh (oxidation-reduction potential), and the characteristics of the available anions. The metal may exist in water in any of four oxidation states; however, Mn(II) predominates in most waters (pH 4–7), but may become oxidized under alkaline conditions at pH  $>8$  (EPA 1984). The principal anion associated with Mn(II) in water is usually carbonate ( $\text{CO}_3^{-2}$ ), and the concentration of manganese is limited by the relatively low solubility (65 mg/L) of manganese carbonate (Schaanning et al. 1988). Under oxidizing conditions, the solubility of Mn(II) may be controlled by manganese oxide equilibria (Ponnamperuma et al. 1969), with manganese being converted to the Mn(II) or Mn(IV) oxidation states (Rai et al. 1986). In extremely reduced water, the fate of manganese tends to be controlled by formation of a poorly soluble sulfide (EPA 1984).

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Manganese is often transported in rivers as suspended sediments. It has been reported that most of the manganese in a South American river came from industrial sources and was bound to suspended particles in the water (Malm et al. 1988).

In an aquifer studied in France, manganese was shown to originate from within the aquifer itself (Jaudon et al. 1989). In the presence of decreased dissolved oxygen in the groundwater, Mn(IV) has been shown to be reduced both chemically and bacterially into the Mn(II) form (Jaudon et al. 1989). This oxidation state is water soluble and easily released into the groundwater.

Manganese in water may be significantly bioconcentrated at lower trophic levels. A bioconcentration factor (BCF) relates the concentration of a chemical in plant and animal tissues to the concentration of the chemical in the water in which they live. Folsom et al. (1963) estimated that the BCFs of manganese were 2,500–6,300 for phytoplankton, 300–5,500 for marine algae, 800–830 for intertidal mussels, and 35–930 for coastal fish. Similarly, Thompson et al. (1972) estimated that the BCFs of manganese were 10,000–20,000 for marine and freshwater plants, 10,000–40,000 for invertebrates, and 100–600 for fish. In general, these data indicate that lower organisms such as algae have larger BCFs than higher organisms. In order to protect consumers from the risk of manganese bioaccumulation in marine mollusks, EPA has set a criterion for manganese at 0.1 mg/L for marine waters (EPA 1993b).

The tendency of soluble manganese compounds to adsorb to soils and is dependent upon the cation exchange capacity and the organic composition of the soil (Curtin et al. 1980; Hemstock and Low 1953; Kabata-Pendias and Pendias 1984; McBride 1979; Schnitzer 1969). Baes and Sharp (1983) noted that soil adsorption constants (the ratio of the concentration in soil to the concentration in water) for Mn(II) span five orders of magnitude, ranging from 0.2 to 10,000 mL/g, increasing as a function of the organic content and the ion exchange capacity of the soil; thus, adsorption may be highly variable. In some cases, adsorption of manganese to soils may not be a readily reversible process. At low concentrations, manganese may be "fixed" by clays and will not be released into solution readily (Reddy and Perkins 1976). At higher concentrations, manganese may be desorbed by ion exchange mechanisms with other ions in solution (Rai et al. 1986). For example, the discharge of waste water effluent into estuarine environments resulted in the mobilization of manganese from the bottom sediments (Helz et al. 1975; Paulson et al. 1984). The metals in the effluent may have been preferentially adsorbed resulting in the release of manganese.

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**6.3.2 Transformation and Degradation****6.3.2.1 Air**

Very little information is available on atmospheric reactions of manganese (EPA 1984). Manganese can react with sulfur dioxide and nitrogen dioxide, but the occurrence of such reactions in the atmosphere has not been demonstrated.

MMT undergoes photolysis rapidly by sunlight in the atmosphere or in aqueous solutions with a very short half-life (i.e., <2 minutes) (Ter Haar et al. 1975; Garrison et al. 1995). The photodegradation products tentatively identified in aqueous photolysis experiments were methylcyclopentadiene, cyclopentadiene, carbon monoxide, manganese carbonyl, and trimanganese tetroxide (Garrison et al. 1995). Undegraded MMT is not likely to be released directly to the atmosphere in significant quantities from its intended use as a gasoline additive. Spectroscopic studies of the tailpipe emissions of MMT-containing gasoline indicated that the manganese in MMT is converted to a mixture of solid manganese oxides, sulfates, and phosphates. The manganese containing particulates were determined to be  $Mn_3O_4$ ,  $MnSO_4 \cdot H_2O$  and a divalent manganese phosphate,  $Mn_5(PO_4)[PO_3(OH)]_2 \cdot 4H_2O$  (Mölders et al. 2001; Ressler et al. 2000).

**6.3.2.2 Water**

Manganese in water may undergo oxidation at high pH or Eh (see Section 6.3.1) and is also subject to microbial activity. For example, Mn(II) in a lake was oxidized during the summer months, but this was inhibited by a microbial poison, indicating that the oxidation was mediated by bacteria (Johnston and Kipphut 1988). The microbial metabolism of manganese is presumed to be a function of pH, temperature, and other factors, but no data were located on this.

The rate of MMT degradation in natural aquifer and sediment systems was determined to be very slow under anaerobic conditions (Garrison et al. 1995). Calculated half-lives ranged from approximately 0.2 to 1.5 years at 25 °C. However, MMT photolyzed rapidly in purified, distilled water exposed to sunlight. The disappearance of MMT followed first-order kinetics, with a calculated half-life of 0.93 minutes. Reaction products included methylcyclopentadiene, cyclopentadiene, carbon monoxide, and a manganese carbonyl that readily oxidized to trimanganese tetroxide.

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**6.3.2.3 Sediment and Soil**

The oxidation state of manganese in soils and sediments may be altered by microbial activity. Geering et al. (1969) observed that Mn(II) in suspensions of silt or clay loams from several areas of the United States was oxidized by microorganisms, leading to the precipitation of manganese minerals. Other studies (Francis 1985) have shown that bacteria and microflora can increase the mobility of manganese in coal-waste solids by increasing dissolution of manganese in subsurface environments.

MMT was found to be stable in a stream bottom sediment under anaerobic conditions. Photodegradation of MMT is not likely to occur in sediments, and it may equilibrate between the sediment, sediment porewater, and water column manganese (Garrison et al. 1995).

**6.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT**

Reliable evaluation of the potential for human exposure to manganese depends in part on the reliability of supporting analytical data from environmental samples and biological specimens. Concentrations of manganese in unpolluted atmospheres and in pristine surface waters are often so low as to be near the limits of current analytical methods. In reviewing data on manganese levels monitored or estimated in the environment, it should also be noted that the amount of chemical identified analytically is not necessarily equivalent to the amount that is bioavailable. The analytical methods available for monitoring manganese in a variety of environmental media are detailed in Chapter 7.

**6.4.1 Air**

Table 6-3 summarizes historic manganese air level data collected over a period of nearly 30 years from numerous urban, nonurban, and source-dominated areas of the United States. Direct comparisons of data from different time periods are complicated because of changes in sample collection and analytical methodology. However, it is clear that manganese levels tend to be higher in source-dominated and urban areas than in nonurban areas. These data also indicate that concentrations in all areas have tended to decrease over the past three decades (EPA 1984; Kleinman et al. 1980). This decrease came as the result of the installation of emission controls in the metals industry (EPA 1984, 1985d). A concurrent decrease in total suspended particulates (TSP) was observed in most areas. Ambient air levels of manganese (PM<sub>10</sub> and PM<sub>2.5</sub>) in Canadian locations monitored from the late 1980s through the early 2000s were reported to have a 13–77% reduction over that time period (Health Canada 2008). Annual averages of manganese in urban and rural areas without significant manganese pollution are in the range of 10–70 ng/m<sup>3</sup> (0.01–

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**Table 6-3. Average Levels of Manganese in Ambient Air<sup>a</sup>**

Sampling location	Concentration (ng/m <sup>3</sup> )		
	1953–1957	1965–1967	1982
Nonurban	60	12	5
Urban	110	73	33
Source dominated	No data	250–8,300	130–140

<sup>a</sup>Adapted from EPA 1984



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0.07  $\mu\text{g}/\text{m}^3$ ) (WHO 1997). The daily intake of manganese in the air by the general population in areas without manganese emitting industries was estimated to be  $<2$   $\mu\text{g}/\text{day}$  (WHO 1981). In areas with major foundry facilities, intake may rise to 4–6  $\mu\text{g}/\text{day}$ , and in areas associated with ferro- or silicomanganese industries, it may be as high as 10  $\mu\text{g}$ , with 24-hour peak values exceeding 200  $\mu\text{g}/\text{day}$  (WHO 1981). Data compiled for 2006 under the EPA Urban Air Toxics Monitoring Program, studied ambient air levels of manganese and several other metals at 20 urban locations across the United States. Manganese ( $\text{PM}_{10}$ ) was detected in 415 samples of urban air at levels ranging from 0.24 to 89.10  $\text{ng}/\text{m}^3$  (EPA 2007b). The arithmetic mean, geometric mean, and median concentrations were 10.13, 6.68, and 6.29  $\text{ng}/\text{m}^3$ , respectively. Manganese levels ranged from 0.85 to 614.00  $\text{ng}/\text{m}^3$  in 114 samples of total suspended particulates (TSP) at these 20 urban locations. The arithmetic mean, geometric mean, and median concentrations of manganese in TSP were 47.89, 22.39, and 23.98  $\text{ng}/\text{m}^3$ , respectively.

During 1988–1993, ambient concentration of fine ( $\text{PM}_{2.5}$ ) manganese ranged from 1  $\text{ng}/\text{m}^3$  (0.001  $\mu\text{g}/\text{m}^3$ ) in rural sites in U.S. National Parks to 3  $\text{ng}/\text{m}^3$  (0.003  $\mu\text{g}/\text{m}^3$ ) in urban sites in California (Wallace and Slonecker 1997). There is concern in Canada regarding the combustion of MMT as an important source of manganese contamination in the urban environment, especially in areas of high traffic density. For instance, Loranger and Zayed (1997a) reported significantly higher levels of both respirable and total manganese levels at a high traffic density site (24 and 50  $\text{ng}/\text{m}^3$ , respectively) in Montreal in contrast to a low traffic density site (15 and 27  $\text{ng}/\text{m}^3$ , respectively). Temporal variation of respirable and total manganese was similar for both sites, and atmospheric manganese concentrations reflected a positive relationship with the traffic density. However, as discussed in Section 6.2.1, it has been difficult to assess the exact contribution of the combustion of MMT by vehicles to manganese levels in the environment.

In Montreal, Canada, ambient atmospheric concentrations of MMT, and respirable and total manganese, were measured in five microenvironments including a gas station, an underground car park, downtown Montreal, near an expressway, and near an oil refinery (Zayed et al. 1999a). The overall mean concentrations of respirable manganese, total manganese, and MMT measured for all the microenvironments were 36, 103, and 5  $\text{ng}/\text{m}^3$ , respectively (0.036, 0.103, and 0.005  $\mu\text{g}/\text{m}^3$ ); however, the mean respirable manganese concentration 53  $\text{ng}/\text{m}^3$  (0.053  $\mu\text{g}/\text{m}^3$ ) near the expressway was greater than the EPA Reference Concentration (RfC) of 0.05  $\mu\text{g}/\text{m}^3$ .

The Canadian National Air Pollution Surveillance (NAPS) Program reported that average fine ( $\text{PM}_{2.5}$ ) manganese levels from 2003 to 2005 in cities with industrial sources (Windsor and Hamilton) were 9–15  $\text{ng}/\text{m}^3$  (Health Canada 2008). In Vancouver, Winnipeg, Montreal, Quebec, Toronto, and Edmonton,

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the average levels were 4–14 ng/m<sup>3</sup>. In Saskatoon, Ottawa, Victoria, St. John, and background sites, levels were <5 ng/m<sup>3</sup>. NAPS also reported manganese PM<sub>10</sub> levels were: 20–60 ng/m<sup>3</sup> in Hamilton and Windsor; 8–25 ng/m<sup>3</sup> in Montreal, Toronto, Edmonton, Winnipeg, Quebec, Calgary, Vancouver, and Victoria; and generally <10 ng/m<sup>3</sup> in Saskatoon, Ottawa, St. John, Yellowknife, and background sites (Health Canada 2008).

Studies were conducted in Indianapolis, Indiana and Toronto, Canada to assess levels of PM<sub>2.5</sub> and PM<sub>10</sub> manganese in indoor, outdoor, and personal air samples (Pellizzari et al. 1999, 2001). The levels observed in Toronto, where MMT had been used in gasoline for over 20 years, were approximately 2 times greater in indoor and outdoor air than in Indianapolis, where MMT was not being used as a gasoline additive. The monitoring data from these studies are summarized in Table 6-4.

#### 6.4.2 Water

Many factors, both environmental (e.g., the presence of high or low levels of other inorganics in drinking water) and biological or host-related (e.g., age, nutritional status, and alcohol consumption) can significantly influence the uptake of manganese by an individual (EPA 1993b). The determination of a single concentration of manganese in drinking water, then, must be recognized as a process that is limited in its ability to reflect the variable nature of manganese toxicity (EPA 1993b).

Concentrations of manganese in surface water are usually reported as dissolved manganese. Although total manganese may be a better indicator, since manganese adsorbed to suspended solids may exceed dissolved manganese in many systems, the bioavailability of manganese in this form has not been established (EPA 1984; NAS 1977). In a 1962–1967 survey of U.S. surface waters, dissolved manganese was detected in 51% of 1,577 samples, at a mean concentration of 59 µg/L. Individual values ranged from 0.3 to 3,230 µg/L. Mean concentrations for 15 different drainage basins in the United States ranged from 2.3 µg/L in the western Great Lakes to 232 µg/L in the Ohio River drainage basin (Kopp and Kroner 1967). A later (1974–1981) survey of U.S. river waters reported a median dissolved manganese concentration of 24 µg/L in samples from 286 locations, with values ranging from <11 µg/L (25th percentile) to >51 µg/L (75th percentile) (Smith et al. 1987). Analyzing data available from the USGS National Water Quality Assessment (NAWQA) database, the EPA reported that the median concentration of manganese was 16 µg/L for surface water and 5 µg/L for groundwater from 20 watersheds and 16 drainage basins in the United States (EPA 2003a). The results of this analysis for all sites are reproduced in Table 6-5. Reported mean groundwater concentrations of manganese were 20 and 90 µg/L

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**Table 6-4. Levels of PM<sub>2.5</sub> and PM<sub>10</sub> in Indoor and Outdoor Air in Toronto, Canada and Indianapolis, Indiana**

Location	Number	Median concentration (ng/m <sup>3</sup> )	90 <sup>th</sup> concentration (ng/m <sup>3</sup> )
PM <sub>10</sub> Manganese			
Toronto (indoor)	203	6.7	14
Indianapolis (indoor)	59	3.9	8.7
Toronto (outdoor)	203	17	28
Indianapolis (outdoor)	59	8.8	14
PM <sub>2.5</sub> Manganese			
Toronto (indoor)	187	4.7	9.9
Indianapolis (indoor)	58	2.2	4.6
Toronto (outdoor)	185	8.6	16
Indianapolis (outdoor)	57	3.2	5.8

Sources: Pellizzari et al. 1999, 2001

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**Table 6-5. Manganese Detections and Concentrations in Surface Water and Groundwater in the United States**

	Detection frequency				Concentration ( $\mu\text{g/L}$ )	
	Above the minimal reporting level (1 $\mu\text{g/L}$ )		Above the health reference level <sup>a</sup> (300 $\mu\text{g/L}$ )		Median	99 <sup>th</sup>
	Samples	Sites	Samples	Sites		
<b>Surface water</b>						
Urban	99.1%	99.6%	4.6%	13.0%	36	700
Mixed	92.4%	98.5%	1.3%	6.4%	12	400
Agricultural	96.3%	97.2%	3.7%	12.3%	19	700
Forest/rangeland	90.9%	96.4%	5.0%	6.6%	11	800
All sites	94.0%	96.9%	3.0%	10.2%	16	700
<b>Groundwater</b>						
Urban	74.7%	85.3%	17.2%	21.0%	15	5,600
Mixed	56.9%	62.9%	8.9%	9.0%	2	1,300
Agricultural	61.4%	64.0%	11.9%	12.8%	4	1,600
Forest/rangeland	75.3%	81.3%	10.9%	13.8%	12	2,900
All sites	64.1%	70.1%	12.8%	13.8%	5	2,900

<sup>a</sup>The Health Reference Level (HRL) is based on the dietary reference dose (RfD) and application of a modifying factor (MF) of 3 for drinking water, and on an allocation of an assumed 20% relative source contribution from water ingestion as opposed to total manganese exposure.

Source: EPA 2003a

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in an analysis of California shallow groundwater from two geologic zones (Deverel and Millard 1988). Values up to 1,300 and 9,600 µg/L have been reported in neutral and acidic groundwater, respectively (EPA 1984). Manganese levels of 9,500–18,600 µg/L have been reported in four private wells in Connecticut (CDHS 1990). Natural concentrations of manganese in seawater reportedly range from 0.4 to 10 µg/L (EPA 1984).

A 1962 survey of public drinking water supplies in 100 large U.S. cities reported that 97% contained <100 µg/L of manganese (USGS 1964). Similarly, a 1969 survey of 969 systems reported that 91% contained <50 µg/L, with a mean concentration of 22 µg/L (U.S. DHEW 1970). Several other studies reported similar manganese concentrations, with mean values ranging from 4 to 32 µg/L (EPA 1984; NAS 1980a; WHO 1981). The EPA analyzed drinking water statistics from Alabama, California, Illinois, New Jersey, and Oregon for occurrence and concentration data for manganese in public water supplies. The data used contained >37,000 analytical results from about 4,000 public water supplies from 1993 to 1997, although some prior monitoring data were also employed in the analysis. The median manganese level for all detections was 10 µg/L and the 99<sup>th</sup> percentile of the detections was 720 µg/L (EPA 2003a).

#### 6.4.3 Sediment and Soil

Manganese comprises about 0.1% of the earth's crust (Graedel 1978; NAS 1973), and manganese occurs naturally in virtually all soils. Average natural ("background") levels of manganese in soils range from around 40 to 900 mg/kg, with an estimated mean background concentration of 330 mg/kg (Barceloux 1999; Cooper 1984; Eckel and Langley 1988; EPA 1985c; Rope et al. 1988; Schroeder et al. 1987). The maximum value reported was 7,000 mg/kg (Eckel and Langley 1988). Using data from 20 watersheds and 16 drainage basins in the United States, manganese was detected at 100% of the National Water-Quality Assessment Program (NAWQA) stream bed sediment sampling sites. The median and 99<sup>th</sup> percentile concentrations in bed sediments were reported as 1.1 mg/kg (dry weight) and 9.4 mg/kg (dry weight), respectively (EPA 2003a). Manganese levels as high as 1,900 mg/kg were detected in sediment samples obtained from the Tar Creek Superfund site (a site heavily contaminated with mining wastes) in Ottawa County, Oklahoma (Wright et al. 2006).

Accumulation of manganese in soil usually occurs in the subsoil and not on the soil surface; 60–90% of manganese is found in the sand fraction of the soil (WHO 1981). A preliminary survey was conducted in Utah to provide an initial field measurement of the contamination by manganese oxides from exhaust in roadside soil and plant species due to the addition of MMT to motor vehicle fuels. Soil (0–5 cm)

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manganese concentrations were strongly correlated with distance from roadways with moderate and moderately high traffic volumes (Lytle et al. 1994). In addition, exchangeable manganese was found to be significantly higher in an organic soil located at stations with a high traffic density comparing to another one with a low traffic density (Brault et al. 1994). The average soil manganese concentration measured at 1 meter from a moderate to moderately-high traffic volume roadside was 3,046 µg/g dry weight. At 15m, the average soil manganese concentration decreased to 254 µg/g dry weight.

#### 6.4.4 Other Environmental Media

Manganese is a natural component of most foods. A summary of mean manganese concentrations in foods analyzed by the Food and Drug Administration (FDA) Total Diet Study (TDS) 1991–1996 is summarized in Table 6-6. TDS sampling is conducted 4 times annually, once in each of the major geographical regions of the country (west, north central, south, and northeast). Each round of sampling is referred to as an individual market basket survey and for each market basket survey, samples of 260 selected food and beverages were obtained from three cities within the region. The mean and median concentration of manganese in all foods were 2.4 and 1.0 mg/kg, respectively (Capar and Cunningham 2000). The TDS results concluded that detectable levels of manganese were present in roughly 75% of all foods, although approximately 24% of these detections were below the quantification limits used in the study (Capar and Cunningham 2000). The highest manganese level was observed in a sample of shredded wheat cereal (44.4 mg/kg). The five foods with the highest mean manganese levels were oat ring cereal (33.8 mg/kg), raisin bran cereal (28.8 mg/kg), shredded wheat cereal (25.0 mg/kg), mixed nuts (23.2 mg/kg), and granola cereal (20.1 mg/kg). These levels are similar to levels found in previous market basket surveys (Pennington et al. 1986). Tea and leafy green vegetables were the major dietary sources of manganese for young women taking part in a dietary study in Wisconsin (Davis et al. 1992a).

Bioaccumulation of manganese by plants was examined using oats (*Avena nova*) and beans (*Phaseolus vulgaris*) (Brault et al. 1994). These plants were grown in sandy and organic soil at a control site (greenhouse) and at two outdoor sites near <20,000 and 132,000 vehicles/day respectively. The highest manganese accumulation was found in the fruits and stems of oats grown in the organic and sandy soils at the station with the highest traffic density. Lönnerdal (1997) reported that infant formulas contain 30–75 ppb (0.03–0.075 ppm) manganese, as compared to concentrations of 3–10 ppb (0.003–0.01 ppm) in breast milk and 30 ppb (0.03 ppm) in cow's milk.

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**Table 6-6. Mean Concentrations of Manganese for FDA's Total Diet Study Market Baskets 1991 through 1997<sup>a</sup>**

Food product	Range (mg/kg)
Milk and cheese	Not detected–<2
Eggs	<1
Meat, poultry, and fish	Not detected–3.7
Legumes and nuts	3.4–23.2
Grain products	<1–33.8
Fruit	<1–10.0
Vegetables	<1–5.9
Mixed dishes and meals	<1–3.4
Desserts	Not detected–4.9
Snacks	3.4–9.3
Condiments and sweeteners	Not detected–4.1
Fats and dressings	Not detected–<1
Beverages	Not detected–2.9
Infant and junior foods	Not detected–7.5

<sup>a</sup>A < symbol indicates that manganese was detected, but at a level lower than the limit of quantification.

Source: Capar and Cunningham 2000

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During a 1992 survey conducted by Canada's Department of Fisheries and Oceans, concentrations of manganese were detected in the muscle samples of bluefin tuna (*Thunnus thynnus*) (Hellou et al. 1992). Concentrations of manganese in 14 samples of fish muscle ranged from 0.16 to 0.31 µg manganese/g dry weight, with a mean of 0.22 µg/g. Although the analysis was administered with a high accuracy of 94% using inductively coupled plasma-mass spectrometry (ICP-MS), the sample population was small.

In the field survey conducted by Lytle et al. (1994), terrestrial and aquatic plant samples were collected along motorways and local urban roadways throughout Utah during 1992 and 1993. Manganese was detected in the plant samples, with manganese concentrations ranging from 30.2 to 13,680 µg/g dry weight. Manganese was detected in plants found nearest to the motorway. Loranger et al. (1994b) evaluated the use of the pigeon as a monitor for manganese contamination from motor vehicles in urban and rural areas of Canada, a country in which MMT has been used to replace lead in gasoline. Manganese concentrations were similar in the two groups of pigeons for all tissues except the liver and feces; urban pigeons had about 35% more manganese than rural ones. Loranger et al. (1994b) suggested that although pigeon feces and liver may be good biomarkers of manganese contamination, it is premature to associate the excess manganese with the combustion of MMT. Toxicokinetic studies of manganese in both male and female rats suggested that MMT-derived manganese administered in oral doses resulted in higher and more prolonged plasma concentration versus time profiles than inorganic ( $MnCl_2$ ) complexes, leading to the conclusion that MMT-derived manganese was likely to accumulate following repeated exposures (Zheng et al. 2000).

### 6.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

Since manganese is ubiquitous in the environment, the general population is exposed to manganese from both natural and anthropogenic sources. The manganese concentration in blood of healthy adults is reported to range from 4 to 15 µg/L with an average value of about 9 µg/L (Barceloux 1999). Typical daily human exposure levels to manganese from water, air, and food are summarized in Table 6-7 (EPA 1984). As the table illustrates, the most significant exposure for the general population is from food, with an average ingestion rate of 3,800 µg/day (EPA 1984). Other estimates of daily intake for adults range from 2,000 to 8,800 µg (EPA 1984; NAS 1977; Patterson et al. 1984; Pennington et al. 1986; WHO 1984a) and 700–10,900 µg/day (WHO 2004b). Even though gastrointestinal absorption of manganese is low (3–5%), oral exposure is the primary source of absorbed manganese.



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**Table 6-7. Summary of Typical Human Exposure to Manganese<sup>a</sup>**

Parameter	Exposure medium		
	Water	Air	Food
Typical concentration in medium	4 µg/L	0.023 µg/m <sup>3</sup>	1.28 µg/calories
Assumed daily intake of medium by 70-kg adult	2 L	20 m <sup>3</sup>	3,000 calories
Estimated average daily intake by 70-kg adult	8 µg	0.46 µg <sup>b</sup>	3,800 µg
Assumed absorption fraction	0.03 <sup>c</sup>	1 <sup>c</sup>	0.03 <sup>d</sup>
Approximate absorbed dose	0.24 µg	0.46 µg	114 µg

<sup>a</sup>Adapted from EPA 1984

<sup>b</sup>Assumes 100% deposition in the lungs

<sup>c</sup>No data; assumed value

<sup>d</sup>Vitarella et al. 2000

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Manganese intake among individuals varies greatly, depending upon dietary habits. For example, an average cup of tea may contain 0.4–1.3 mg of manganese (Pennington et al. 1986; Schroeder et al. 1966). Thus, an individual consuming three cups of tea per day might receive up to 4 mg/day from this source alone, increasing the average intake from all dietary sources.

As part of the Third National Health and Nutrition Examination Survey (NHANES) conducted by the Centers of Disease Control and Prevention (CDC), manganese was detected at quantifiable levels in urine samples from 73% of 496 participants of the monitoring study (Paschal et al. 1998). The mean urinary manganese concentration in these 496 individuals (aged 6–88 years of age) was 1.19 µg/L (Paschal et al. 1998).

The EPA Reference Dose (RfD)/RfC workgroup in June 1990 set an RfD for manganese in food of 0.14 mg manganese/kg/day, equivalent to about 10 mg/day for a 70-kg man based on chronic manganese uptake (EPA 1993b). The Food and Nutrition Board of the National Research Council (NRC) estimated the adequate and safe intake of manganese for adults at 2–5 mg/day (NAS 1980b). This level was chosen because it includes an "extra margin of safety" of 5 mg/day below the level of 10 mg/day, which the NRC considered to be safe for occasional intake (IRIS 2008).

In the workplace, exposure to manganese is most likely to occur by inhalation of manganese fumes or manganese-containing dusts. This is a concern mainly in the ferromanganese, iron and steel, dry-cell battery, and welding industries (WHO 1986). Exposure may also occur during manganese mining and ore processing; however, the most recent data indicate that only a very small amount of manganese is still mined in the United States (USGS 2007). Excluding insignificant quantities of similar low-grade manganiferous ore, the United States has not mined significant amounts of manganese since 1978 and now relies on imports to fill its needs (USGS 2007). In 1980, it was estimated that in the United States about 300 workers were exposed to pure manganese and about 630,000 workers were exposed to other forms of manganese (NOES 1989). Concentrations as large as 1.5–450 mg manganese/m<sup>3</sup> have been reported in U.S. manganese mines (EPA 1984), 0.30–20 mg manganese/m<sup>3</sup> in ferroalloy production facilities (Saric et al. 1977), and 3–18 mg manganese/m<sup>3</sup> in a dry-cell battery facility (Emara et al. 1971). Steel-manufacturing facilities are significant employers in the United States. There is a potential for manganese exposure to workers in these facilities. Airborne manganese levels in a metal-producing plant in the United States were reported as 0.066 mg/m<sup>3</sup> (mean), 0.051 mg/m<sup>3</sup> (median) as respirable dust, and 0.18 mg/m<sup>3</sup> in total dust (Gibbs et al. 1999). Exposure levels should not exceed the Occupational Safety and Health Administration (OSHA) time-weighted average Permissible Exposure Limit (PEL) of 1 mg

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total manganese/m<sup>3</sup> (see Table 8-1). Average airborne manganese levels during welding operations of two factories located in China were 0.24 and 2.21 mg/m<sup>3</sup> (Wang et al. 2008). Manganese levels in workplace air at a smelting facility in China ranged from 0.30 to 2.9 mg/m<sup>3</sup> in the furnace smelting area and from about 0.2 to 0.8 mg/m<sup>3</sup> in a power control room (Jiang et al. 2007). The workplace air at this facility contained mainly MnO (20%) and SiO<sub>2</sub> (22%), in addition to other trace metals including Fe<sub>2</sub>O<sub>3</sub> (4%), CaO (4.5%), MgO (4%), and Al<sub>2</sub>O<sub>3</sub> (5%). Thus, for workers in industries using manganese, the major route of exposure may be inhalation from workplace air rather than from ingestion of food.

Occupational exposure to manganese resulting from the combustion of MMT in Montreal, Canada has been studied. Sierra et al. (1995) conducted a study of Montreal automotive workers (garage mechanics) and nonautomotive workers (control group). Exposure to manganese was measured for 5 consecutive working days. In addition, their environmental exposure (at home) was measured on 2 days of the same week. Air sampling was performed by portable pumps; for sampling at homes, workers were asked to wear the pumps as much as possible. At the workplace, the mechanics were exposed to manganese concentrations ranging from 0.010 to 6.673 µg/m<sup>3</sup> (mean of 0.45 µg/m<sup>3</sup>), while nonautomotive workers were exposed to manganese concentrations ranging from 0.011 to 1.862 µg/m<sup>3</sup> (mean of 0.04 µg/m<sup>3</sup>). The average manganese concentrations in the indoor air of the homes were 0.012 µg/m<sup>3</sup> for the mechanics and were 0.008 µg/m<sup>3</sup> for the nonautomotive workers (Sierra et al. 1995). Based on measurements of manganese particle size distributions, Sierra et al. (1995) estimated that <10% of the manganese exposure of the garage mechanics was due to MMT; however, the exact contribution of MMT could not be determined.

A similar study conducted in Montreal by these investigators, but involving taxi drivers and garage mechanics revealed that garage mechanics at work were exposed to an average of 0.250 µg/m<sup>3</sup> and taxi drivers to 0.024 µg/m<sup>3</sup> (Zayed et al. 1994). In another study, exposure of office workers and taxi drivers to both respirable and total manganese was evaluated (Zayed et al. 1996). Manganese concentrations measured for the office workers ranged from 0.001 to 0.034 µg/m<sup>3</sup> (respirable manganese) and from 0.002 to 0.044 µg/m<sup>3</sup> (total manganese). For the taxi drivers, the manganese concentrations ranged from 0.007 to 0.032 µg/m<sup>3</sup> (respirable manganese) and from 0.008 to 0.073 µg/m<sup>3</sup> (total manganese). Zayed et al. (1996) concluded that the higher exposure to atmospheric manganese in the outdoor urban environment may be at least partly due to the use of MMT in cars. Nevertheless, these investigators indicated that the exposures of taxi drivers to manganese were well below existing exposure and health guidelines.

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In order to assess the potential health risks from MMT combustion, Loranger and Zayed (1995) conducted a multi-media assessment (i.e., food, water, and ambient air) of manganese exposure in two groups of workers (garage mechanics and blue-collar workers) potentially exposed to different levels of manganese from MMT. Garage mechanics were exposed to higher air manganese concentrations ( $0.42 \mu\text{g}/\text{m}^3$ ) than blue-collar workers ( $0.04 \mu\text{g}/\text{m}^3$ ). However, for the garage workers, exposure to atmospheric manganese represented only approximately 4% of the total absorbed dose, while ingestion of food represented 95.7% of the total multi-media dose. For the blue collar workers, atmospheric manganese contributed only 0.3% to the total absorbed dose, whereas ingestion of food represented 99.2% of the total multi-media dose. These results were consistent with values of multi-media doses predicted by GADUS, an environmental fate/exposure model (Loranger and Zayed 1997b). Based on governmental standards or criteria for occupational and environmental exposures, Loranger and Zayed (1995) concluded that the manganese levels in food and air may not cause any problems for these workers.

Based on an analysis of data obtained from a large, continuous personal exposure study in Toronto, Canada, a city with widespread use of MMT, it was determined that the general population was exposed to low concentrations (median concentration was  $0.008 \mu\text{g}/\text{m}^3$ ) of  $\text{PM}_{2.5}$  manganese in personal air samples (Lynam et al. 1999; Pellizzari et al. 1999). A similar study, which collected 925 personal exposure samples for residents of Toronto, also concluded that MMT was not a significant source of  $\text{PM}_{2.5}$  manganese inhalation exposure for the general population (Crump 2000). However, personal exposure levels of fine manganese in Toronto were nearly 3 times greater when compared to data obtained from Indianapolis, Indiana where MMT was not being used as a gasoline additive. The median concentration of  $\text{PM}_{2.5}$  manganese in personal exposure samples from Indianapolis was  $0.0028 \mu\text{g}/\text{m}^3$  (Pellizzari et al. 2001). These data are summarized in Table 6-8. Certain activities such as time spent in subways, metal working, and smoking were associated with higher personal manganese exposure.

## 6.6 EXPOSURES OF CHILDREN

This section focuses on exposures from conception to maturity at 18 years in humans. Differences from adults in susceptibility to hazardous substances are discussed in Section 3.7, Children's Susceptibility.

Children are not small adults. A child's exposure may differ from an adult's exposure in many ways. Children drink more fluids, eat more food, breathe more air per kilogram of body weight, and have a larger skin surface in proportion to their body volume. A child's diet often differs from that of adults.

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**Table 6-8. Levels of PM<sub>2.5</sub> in Personal Air Samples Collected in Toronto, Canada and Indianapolis, Indiana**

Location	Number	Median concentration (µg/m <sup>3</sup> )	90 <sup>th</sup> concentration (µg/m <sup>3</sup> )
PM <sub>2.5</sub> Manganese in personal air			
Toronto	272	0.008	0.016
Indianapolis	240	0.0028	0.006

Source: Pellizzari et al. 1999, 2001

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The developing human's source of nutrition changes with age: from placental nourishment to breast milk or formula to the diet of older children who eat more of certain types of foods than adults. A child's behavior and lifestyle also influence exposure. Children crawl on the floor, put things in their mouths, sometimes eat inappropriate things (such as dirt or paint chips), and spend more time outdoors. Children also are closer to the ground, and they do not use the judgment of adults to avoid hazards (NRC 1993).

Children would be exposed to manganese in the same manner as adults. The main source of exposure of children to manganese is through food. Infants and young toddlers who are formula-fed may receive higher daily intakes of manganese than breast-fed infants because of the increased levels of the element in infant formulas as compared to breast milk (Collipp et al. 1983; Cook 1997; Dorner et al. 1989; Keen et al. 1986; Lönnerdal et al. 1983, 1994). For example, a study of 2,339 breast milk samples obtained from nursing mothers in Germany had a mean manganese level of 6.2 µg/L, while two different brands of formula had levels of 77 and 99 µg/L (Dorner et al. 1989). It was concluded that the mean daily manganese intake of formula-fed infants was approximately 13 times greater than that of breast-fed infants.

Manganese concentrations in blood serum of children of different ages are provided in Section 3.4.2. The data indicate that manganese concentrations decrease slightly from the time the infant is 5 days of age until he or she is 12 months of age (Alarcón et al. 1996; Rügauer et al. 1997). Manganese concentrations increase after this time, and they have been measured as an average of  $1.4 \pm 1.25$  µg/L in children aged 1 month to 18 years (Rügauer et al. 1997).

Children are exposed *in utero* because manganese in maternal blood crosses the placenta to satisfy the fetus's need for manganese. The compound has been measured in cord blood plasma of premature and full-term infants and their mothers (Wilson et al. 1991). Full-term babies had higher (but not statistically significantly different) blood concentrations of manganese than premature babies, and pregnant women had higher blood concentrations than nonpregnant women. The average manganese concentration in the cord blood of full term babies was 5.5 µg/L, as compared to 5.0 µg/L for preterm babies (Wilson et al. 1991). No correlations were observed between maternal and infant concentrations of manganese. Manganese in breast milk has been found to range from 3.4 to 10 µg/L (Arnaud and Favier 1995; Collipp et al. 1983) depending on the maturity of the milk. The Food and Nutrition Board of the NRC based the recommended manganese intake of infants on the analyses of pooled human milk samples. As discussed above, manganese intakes of infants fed some formulas appear high, but no signs of toxicity have been observed (Dorner et al. 1989; Lönnerdal et al. 1983). Differences in weight-adjusted intake are likely to

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be caused by the type of diet that infants and small children receive. It is unknown whether nursing mothers exposed to higher-than-average concentrations of manganese would excrete increased concentrations of the metal in their breast milk.

Young children often eat dirt (exhibiting what is called soil pica, the ingestion of a material unfit for food) and exhibit frequent hand-to-mouth activity; they can be exposed to manganese through this unique pathway if the soils contain the metal. Current estimates indicate that soil pica may be more prevalent in the general population than previously thought and that most children periodically ingest soil to varying degrees; this may be a potential health concern (EPA 1986d; Stanek and Calabrese 1995). The predicted oral average daily intake of manganese for children from soils in the vicinity of a municipal solid waste incinerator was estimated to range from approximately 0.0021 to 0.0032 mg/kg/day (Mari et al. 2007). However, no information was found concerning the bioavailability of manganese from soil and, therefore, determining the actual risk posed to children from this exposure pathway is difficult. This behavior should not pose an increased risk of exposure to manganese in most residential situations where the manganese levels are in the normal or background range. If the soils are from a hazardous waste site that contains high concentrations of manganese, then increased exposure to the compound may occur. Manganese levels in hair samples of 32 children residing near a hazardous waste site (former mining facility) in Northeast Oklahoma ranged from 89.1 to 2,145.3 ppb (471.5 ppb mean) (Wright et al. 2006). The authors found that in school-aged children, higher manganese and arsenic levels in hair samples were associated with significantly lower scores on a standardized test, as well as on tests of verbal learning and memory.

Children who suffer from cholestatic liver disease or who have gastrointestinal disorders that mandate they be given parenteral nutrition may be at increased risk from overexposure to manganese. Increased manganese concentrations in blood and brain, and symptoms of neuromotor dysfunction were observed in an 8-year-old girl with cholestatic liver failure (Devenyi et al. 1994). Children with or without chronic liver disease and a 5-year-old boy who had gastrointestinal disorders, all of whom were administered parenteral nutrition, had abnormal MRI scans indicative of manganese accumulation (Fell et al. 1996; Ono et al. 1995) accompanied by motor disorders (Fell et al. 1996).

Because manganese is a trace element that is essential for normal human health and is predominantly obtained from food, it is unlikely that toxic amounts of manganese will be absorbed from food. However, diets vary and some are higher in manganese than others (diets high in grains and tea, for instance). One case study suggested that a 59-year-old man developed manganism-like symptoms from abusing vitamins

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and minerals. This man had very high manganese concentrations in blood, urine, feces, hair, and brain (Banta and Markesbery 1977). Both manganese and iron are bound by transferrin and these elements compete for the binding protein in the body. Therefore, diets that are low in iron allow transferrin to bind more manganese. For this reason, it is important to provide children with a balanced diet to maintain optimal iron and manganese stores in the body. Studies show that adults absorb only 3–5% of manganese ingested from the diet (Davidsson et al. 1988, 1989a; Mena et al. 1969); infants have increased absorption relative to adults (Dorner et al. 1989). Neonatal animals also exhibit increased absorption relative to older animals (Ballatori et al. 1987; Miller et al. 1975; Rehnberg et al. 1981).

Children may be exposed to organic manganese compounds through a variety of routes. They may be exposed to MMT combustion products via inhalation of these products in air, or ingestion of them after deposition on the soil.

### 6.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

As discussed in Section 6.5, workers in industries using or producing manganese are mostly likely to have higher exposures to manganese, primarily by inhalation of manganese dusts in workplace air as compared to the general population. In a year-long investigation of personal exposure to manganese fine particulate matter (PM<sub>2.5</sub>) for residents of Toronto, Canada, it was determined that workers in the metal industry had the highest personal exposures as compared to other groups. The mean concentration of manganese PM<sub>2.5</sub> in personal samples for 39 workers engaged in welding, soldering, or other metal working practices was 105 ng/m<sup>3</sup>, which was more than 10 times greater than the mean concentration (10 ng/m<sup>3</sup>) for 886 non-metal workers (Crump 2000). Smokers and those nearby second-hand smoke were also shown to be exposed to higher levels of fine particulate matter manganese as compared to nonsmokers. The mean concentration of PM<sub>2.5</sub> manganese in 702 personal air samples of nonsmokers in Toronto, Canada was 10 ng/m<sup>3</sup>, while the mean concentration calculated from 223 personal samples obtained from smokers was 27 ng/m<sup>3</sup> (Crump 2000).

Average airborne manganese levels (total dust) in the breathing zone of two factories located in China were 0.24 and 2.21 mg/m<sup>3</sup> (Wang et al. 2008). The greatest levels were observed during welding operations in enclosed spaces. The workers at these two factories had higher measurable manganese levels in their saliva (3.47±1.42 and 5.55±2.31 µg/L), as compared to a control group of non-occupationally exposed individuals (3.04±1.40 µg/L).



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Workers in three manganese alloy production plants located in Norway were found to have slightly higher manganese blood and urine levels when compared to a group of non-occupationally exposed individuals. The arithmetic mean manganese level in the blood of workers at these plants was 189 nmol/L (10.3 µg/L) versus 166 nmol/L (9.1 µg/L) for the reference group (Ellingsen et al. 2003c). The urinary arithmetic mean concentrations were 3.9 nmol/mmol creatinine for the occupationally exposed workers and 0.9 nmol/mmol creatinine for the reference group (Ellingsen et al. 2003c). The arithmetic mean inhalable and respirable concentrations of manganese in the air of these production plants were 0.769 and 0.064 mg/m<sup>3</sup>, respectively (Ellingsen et al. 2003c). Section 3.2.1.4 summarizes other studies that compared noted health effects with urinary and blood manganese levels of occupationally exposed individuals and reference populations. It has been demonstrated that levels in the blood and urine may not be adequate biomarkers for high level manganese exposure since free manganese usually does not accumulate within the circulatory system (Josephs et al. 2005).

Populations living in the vicinity of ferromanganese or iron and steel manufacturing facilities, coal-fired power plants, or hazardous waste sites may also be exposed to elevated manganese particulate matter in air or water, although this exposure is likely to be much lower than in the workplace. Populations living in regions of natural manganese ore deposits may be exposed to above-average levels in soil, water, or air.

People ingesting large amounts of foods high in manganese also have a potential for higher-than-usual exposure. Included in this group would be vegetarians, who ingest a larger proportion of grains, legumes, and nuts in their diets than the average U.S. population, and heavy tea drinkers. While the intake of manganese from vegetarians may exceed the estimates of daily dietary intake, the bioavailability of manganese from vegetable sources is substantially decreased by dietary components such as fiber and phytates (EPA 1993b). In addition to the population with these dietary habits, individuals with iron deficiency show increased rates of manganese absorption (Mena et al. 1969, 1974); iron deficiency leads to increased brain manganese concentrations in experimental animals (Aschner and Aschner 1990).

Manganese is eliminated from the body primarily through the bile. Interruption of the manufacture or flow of bile can impair the body's ability to clear manganese. Several studies have shown that adults and children (Devenyi et al. 1994; Fell et al. 1996; Hauser et al. 1994, 1996; Pomier-Layrargues et al. 1998; Rose et al. 1999; Spahr et al. 1996), as well as experimental animals (Rose et al. 1999), with cholestatic liver disorders have increased manganese levels in their blood and brain and are at risk from potentially increased exposure to manganese due to their decreased homeostatic control of the compound.

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In addition to oral diets, people on partial and total parenteral nutrition may be exposed to increased amounts of manganese. Forbes and Forbes (1997) found that of 32 patients receiving home parenteral nutrition due to digestive problems, 31 had elevated serum manganese levels (0.5–2.4 mg/L). It is unclear whether these levels reflected steady-state conditions due to the time the samples were taken. However, these levels are much higher than other studies involving patients on TPN; thus, it is unlikely that these levels represent steady-state conditions. Further, the normal range reported by these authors (0.275–0.825 mg/L) is elevated compared to other studies, suggesting the possibility that the blood samples were contaminated with exogenous manganese. The authors observed no clinical evidence of toxicity in the patients. Fourteen of the patients suffered iron deficiency anemia; because low iron concentrations are associated with increased manganese uptake, the anemia may have exacerbated the increased blood manganese concentrations. Increased blood manganese levels and MRI scans indicative of increased manganese in brains have been reported in children fed entirely on parenteral nutrition (Fell et al. 1996; Ono et al. 1995). Only in the Fell et al. (1996) study were neurotoxic effects reported. Whole-blood manganese in the children from this study ranged from 9.9 to 110 µg/L. Devenyi et al. (1994) found hyperintense signals in the brain of an 8-year-old child who had cholestatic liver disease and exhibited dystonia and other motor dysfunctions. Nagatomo et al. (1999) reported that two elderly patients who had been administered TPN for 3–4 months exhibited clinical signs of manganism (including masked facies, marked rigidity, hypokinesia) with associated elevated blood manganese levels and hyperintense signals on MRI, localized to the basal ganglia, especially the globus pallidus. Signs of manganism abated upon levodopa treatment and the administration of Ca-EDTA; the high intensity signals on MRI abated when manganese supplementation ceased. In addition to patients on parenteral nutrition, uremic patients on hemodialysis have been found to have increased manganese levels due to increased concentrations of manganese in the dialysis solution (Lin et al. 1996). These studies indicate that while increased levels of manganese in blood and brain are often associated with TPN administration, adverse neurological effects are not always reported. Nagatomo et al. (1999) found increased serum concentrations of manganese and brain abnormalities in two patients who showed Parkinsonism with psychiatric symptoms after 3–4 months of total parenteral nutrition. Discontinuation of manganese supplementation in the parenteral diet, coupled with levodopa treatment, gradually improved both the symptoms and brain abnormalities in the patients.

In comparison to other groups within the general population, persons living close to high density traffic areas, automotive workers, gas station attendants, and taxi drivers may be exposed to higher concentrations of manganese arising from the combustion of MMT. Levels of respirable manganese, in both indoor and outdoor air near an expressway with high traffic density were shown to be greater than

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corresponding air samples obtained from a rural location in Montreal, Canada (Bolte et al. 2004). The average concentration of respirable manganese (defined in this study as  $<5 \mu\text{m}$  diameter) in outdoor air from the urban location of Montreal was  $0.025 \mu\text{g}/\text{m}^3$ , which is 5 times greater than the average of  $0.005 \mu\text{g}/\text{m}^3$  found in the rural location. The average indoor respirable manganese concentration was also greater for the urban area ( $0.017 \mu\text{g}/\text{m}^3$ ) as compared to the rural area ( $0.007 \mu\text{g}/\text{m}^3$ ). However, differences in exposure levels did not lead to significantly greater levels of manganese in blood for residents of these areas. The mean manganese concentration in blood samples obtained from female residents in the urban location ( $8.4 \mu\text{g}/\text{L}$ ) was only slightly greater than the average level observed for females living in the rural location ( $7.8 \mu\text{g}/\text{L}$ ).

It is possible that medical workers may be exposed to higher concentrations of manganese than the general population, although exposure routes other than intravenous are not expected to pose a significant risk.

## 6.8 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of manganese is available. Where adequate information is not available, ATSDR, in conjunction with NTP, is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of manganese.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

### 6.8.1 Identification of Data Needs

**Physical and Chemical Properties.** The fundamental physical and chemical properties of manganese and manganese compounds are known (see Table 4-2), and additional research does not appear necessary.

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**Production, Import/Export, Use, Release, and Disposal.** According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit substance release and off-site transfer information to the EPA. The TRI, which contains this information for 2006, became available in March of 2008. This database is updated yearly and should provide a list of industrial production facilities and emissions.

Information is available on U.S. import, export and production of manganese ore and related materials (USGS 2007, 2008). It is clear that most manganese is used in steel production. Information regarding the import, export, and use of MMT in U.S. fuels is a data need.

Data from the TRI database provide valuable information on the amounts of manganese released to different environmental media (e.g., air, soil, and water) each year, although details on the chemical form and physical state of the waste materials are not included. These disposal practices are not regulated under current federal law. TRI data may not be complete estimates of total release. Also, because these data reflect past releases, they may not be representative of current releases at these facilities.

**Environmental Fate.** The partitioning of manganese between water and soil can be fairly well predicted using thermodynamic equilibrium concepts, if soil-specific information is available (Baes and Sharp 1983; Rai et al. 1986). The fate of manganese particles released into the air is determined by the particle size, and the direction and distance of particle transport at a site can be predicted from meteorological data and particle size data (EPA 1984; Nriagu 1979). Transport of manganese in water is determined mainly by the solubility of the manganese compounds present, although suspended particles may also be transported in flowing waters (EPA 1984; Schaanning et al. 1988).

The primary transformations that manganese undergoes in the environment are oxidation/reduction reactions (EPA 1984; Rai et al. 1986). Reactions of manganese with airborne oxidants have not been studied. Information on the rate and extent of such reactions would be helpful in understanding the fate of atmospheric releases. The transformation of manganese in water or soil is dependent mainly on Eh, pH, and available counter ions (EPA 1984). In some soils, manganese may also be oxidized by bacteria (Geering et al. 1969; Johnston and Kipphut 1988). More work is needed on the environmental factors, such as soil composition and pH, which may determine the form in which manganese will appear and thus impact manganese availability and absorption.

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Modeling has also provided interesting insight into the contribution of the combustion of MMT to atmospheric manganese (Loranger et al. 1995). According to the model estimations, the contribution of direct emissions from motor vehicles to the atmospheric background manganese (as measured from sampling stations) would be about 50% at <25 m and <8% at 250 m. These results are confirmed with an *in situ* study using snow as the environmental indicator where the average deposition rates of manganese for the top and bottom layers ranged from 0.01 to 0.21 mg/m<sup>2</sup>/day (Loranger et al. 1996). The average concentrations of manganese decreased with distance from the road. However, it was impossible to distinguish between directly-emitted manganese from automobiles, manganese enriched road dust, and the naturally-occurring manganese in crustal materials. No study to date has provided the complete answer to this question and this constitutes one of the major remaining data needs regarding the environmental significance of manganese from MMT and the resulting potential for exposure.

**Bioavailability from Environmental Media.** Manganese is known to be absorbed following inhalation or oral exposure (Mena et al. 1969; Pollack et al. 1965; Zheng et al. 2000), but dermal exposure is not considered to be significant. The uptake of manganese from air, food, milk, and water has been studied (Davidsson et al. 1988, 1989a). However, absorption from soil has not been investigated. In view of the potential for tight binding of manganese to some soil types, studies on this subject would be valuable in evaluating risk to humans, especially children who may ingest contaminated soils near hazardous waste sites. Additional information would also be valuable on the relative bioavailability of different manganese compounds across various environmental media.

**Food Chain Bioaccumulation.** It has been established that while lower organisms (e.g., plankton, aquatic plants, and some fish) can significantly bioconcentrate manganese, higher organisms (including humans) tend to maintain manganese homeostasis (EPA 1984; Folsom et al. 1963; Thompson et al. 1972). This indicates that the potential for biomagnification of manganese from lower trophic levels to higher ones is low, and it does not appear that additional research in this area is essential at this time.

**Exposure Levels in Environmental Media.** Reliable monitoring data for the levels of manganese in contaminated media at hazardous waste sites are needed so that the information obtained on levels of manganese in the environment can be used in combination with the known body burden of manganese to assess the potential risk of adverse health effects in populations living in the vicinity of hazardous waste sites.

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Manganese levels have been monitored in all environmental media, including air, water, soil, and food (Capar and Cunningham 2000; EPA 1984; NAS 1980a; Pennington et al. 1986). Estimates are available for the average human intake levels of manganese from water, air, and food (EPA 1984; WHO 2004b).

More specific data on levels in the environment around those particular sites where manganese is believed to have been dumped would be helpful in determining the extent of exposure levels around such waste sites. In particular, data on the concentration of manganese in the air around hazardous waste sites would be valuable in assessing the potential significance of this exposure pathway.

**Exposure Levels in Humans.** This information is necessary for assessing the need to conduct health studies on these populations. Manganese is a normal component of human tissues and fluids (Sumino et al. 1975; Tipton and Cook 1963). Increased average levels of manganese have been detected in blood and urine of populations exposed to high concentrations of manganese in the workplace (Roels et al. 1987b). Manganese has been measured in hair samples of children residing near a hazardous waste site (Wright et al. 2006); however, the absence of data on levels of manganese in the hair of U.S. children in the general population makes it difficult to draw conclusions about whether the exposures of the children at this site are unusually high. Surveys of manganese levels in the blood or urine of populations living near waste sites could be useful in identifying groups with above-average levels of manganese exposure. More information is also needed to determine whether iron-deficient populations have a higher manganese body burden. Manganese and iron have many physico/chemical similarities and there is a possibility of competition between these elements. Increased manganese concentrations have been shown to inhibit the metabolic function of the iron-dependent enzyme, aconitase (Zheng et al. 1998). Iron deficiency is the single most prevalent nutritional deficiency in the world, and so the potential health risk associated with iron deficiencies exacerbating the brain manganese burden may represent a crucial issue of exposure and susceptibility, and has yet to be evaluated. Air concentrations in areas with high traffic density are sometimes higher than the guide level (Zayed et al. 1999a); therefore, some individuals could be at risk. Research focusing on the environmental level of exposure of certain groups of the population, such as those living near a major highway, is needed.

This information is necessary for assessing the need to conduct health studies on these populations.

**Exposures of Children.** Children are exposed daily to manganese. The compound is an essential trace element vital for the body to function properly and body burden studies are available (Alarcón et al. 1996; Rügauer et al. 1997). Although the primary pathway for exposure is the diet, studies involving

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exposures to airborne manganese (e.g., in dust that may be present at a nearby hazardous waste site or manganese-processing plant) would aid in understanding other pathways that may contribute significantly to children's total body burden of manganese

Soil ingestion is likely the only unique exposure pathway for children. Additional studies concerning bioavailability of manganese from soil would provide important information concerning the proportion of the total daily manganese intake that could originate from ingested soils.

Although infants differ in their weight-adjusted intake of manganese, it is unknown whether older children differ in this parameter. Studies concerning this end point would be very valuable.

Studies involving inhalation or ingestion exposure to MMT in the young are very few (Komura and Sakamoto 1992b, 1994). Although these studies indicate that MMT had very little measurable effect on development, only one dose level was used. Although analytical data indicate that environmental MMT is unlikely to persist (Lynam et al. 1999), it is unknown what typical body burdens of manganese might be in children following long-term exposure to MMT combustion products. Additional studies measuring these end points in the young would be helpful.

Child health data needs relating to susceptibility are discussed in Section 3.12.2, Identification of Data Needs: Children's Susceptibility.

**Exposure Registries.** No exposure registries for manganese were located. This substance is not currently one of the compounds for which a sub-registry has been established in the National Exposure Registry. The substance will be considered in the future when chemical selection is made for sub-registries to be established. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to exposure to this substance.

### 6.8.2 Ongoing Studies

The Federal Research in Progress (FEDRIP 2008) database provides additional information obtainable from a few ongoing studies that may fill in some of the data needs identified in Section 6.8.1.

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Researchers at the University of Delaware (D.M. Di Toro, principal investigator) are conducting research to develop models for predicting the toxicity and mobilization of individual metals (including manganese) and metal mixtures in sediments. These predictions are critical in evaluating the risk associated with contaminated sediments at Superfund sites.

Thomas R. Guilarte and co-workers at Johns Hopkins University are studying the behavioral and neuropathological changes that occur as a result of chronic exposure to low levels of manganese. The findings from the proposed studies will be used to aid in understanding the mechanism(s) of chronic, low-level manganese neurotoxicity. Moreover, these data will identify sensitive markers for the early detection of manganese neurotoxicity that can be used *in vivo* in humans.

Wei Zheng and co-workers at Purdue University are studying the biomarkers for early diagnosis of manganese toxicity among Chinese smelting workers. They plan to combine exposure indices and biological effects into one parameter for quick clinical assessment of manganese toxicity. They are also conducting clinical trials to investigate the efficacy of para-aminosalicylic acid in treatment of severe manganese. Advanced MRI and MRS techniques along with molecular biotechnology have been used in these studies.

Donald Smith and co-workers at the University of California, Santa Cruz are studying the effect that early manganese exposure in neonatal rats has on neurobehavioral and neurocognitive deficits and comparing these data with epidemiological studies in children.