6. POTENTIAL FOR HUMAN EXPOSURE

6.1 OVERVIEW

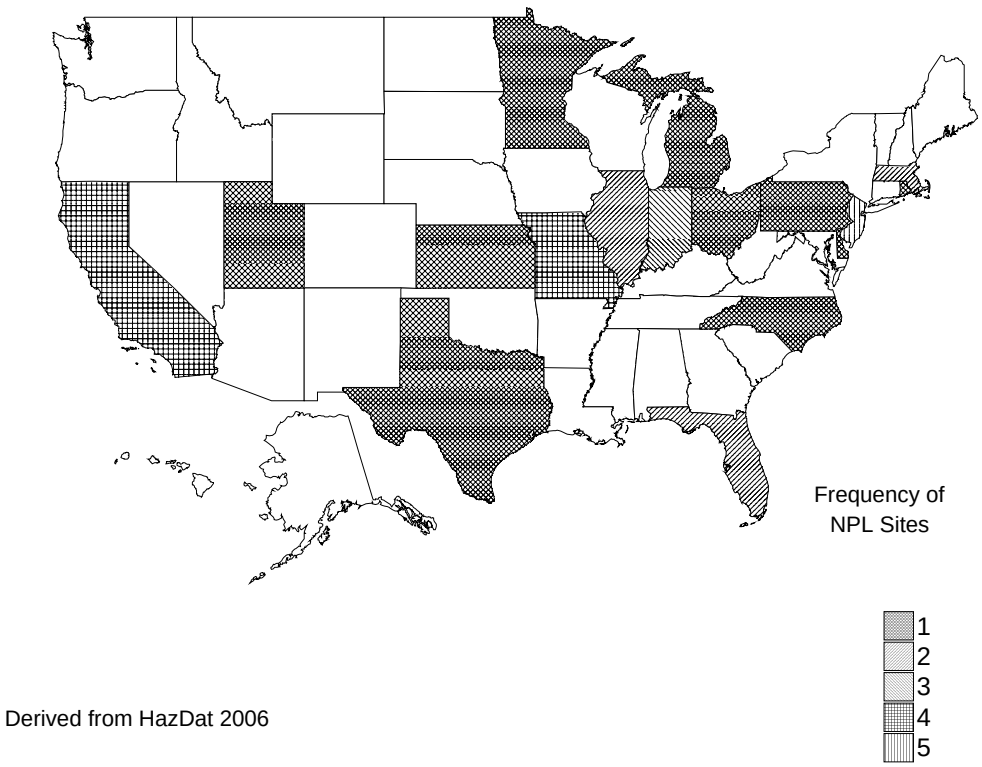
Acrolein has been identified in at least 32 of the 1,684 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 2006). However, the number of sites evaluated for acrolein is not known. The frequency of these sites can be seen in Figure 6-1.

Acrolein may be released to the environment in emissions and effluents from its manufacturing and use facilities, in emissions from combustion processes (including cigarette smoking and combustion of petrochemical fuels), from direct application to water and waste water as a slimicide and aquatic herbicide, as a photooxidation product of various hydrocarbon pollutants found in air (including propylene and 1,3-butadiene), and from land disposal of some organic waste materials. Acrolein is a reactive compound and is unstable in the environment.

In ambient air, the primary removal mechanism for acrolein is predicted to be reaction with photo-chemically generated hydroxyl radicals (half-life, 15–20 hours). Products of this reaction include carbon monoxide, formaldehyde, and glycolaldehyde. In the presence of nitrogen oxides, peroxynitrate and nitric acid are also formed. Small amounts of acrolein may also be removed from the atmosphere in precipitation. Insufficient data are available to predict the fate of acrolein in indoor air. In water, small amounts of acrolein may be removed by volatilization (half-life, 23 hours from a model river 1 m deep), aerobic biodegradation, or reversible hydration to β-hydroxypropionaldehyde, which subsequently biodegrades. Based on the reactivity of acrolein, it is expected that removal of acrolein from water through the binding of the chemical to dissolved and suspended organics will become increasingly important as the concentration of the organics in water increases. However, information on this removal process could not be located.

Half-lives of <1–3 days for small amounts of acrolein in surface water have been observed. When highly concentrated amounts of acrolein are released or spilled into water, this compound may polymerize by oxidation or hydration processes. In soil, acrolein is expected to be subject to removal through volatilization, abiotic and biotic degradation processes, and possibly irreversible binding to soil components. This compound can be highly mobile in soil; however, this movement is expected to be attenuated by the removal processes given above.

Figure 6-1. Frequency of NPL Sites with Acrolein Contamination



Data regarding the monitoring of acrolein are available for ambient and indoor air. Data from the EPA National Air Quality System show average acrolein concentrations in ambient air in the United States ranging between 0.5 and 3.186 ppbv (ppb based on volume) (EPA 2004a). For indoor air, acrolein concentrations range from <0.05 to 29 μg/m^3 (<0.02–12 ppb), with the higher concentrations in this range typically being obtained from indoor environments where the combustion of tobacco products occurs (CARB 1992; Highsmith and Zweidinger 1988; WHO 2002).

No data indicated that acrolein is a contaminant of drinking water supplies. The EPA STORET data indicate that acrolein occurs at a low frequency in waste water streams, ambient surface water, and groundwater in the United States. Acrolein has been detected in surface water and groundwater samples collected offsite at 4 and 15 of the 32 hazardous waste sites, respectively, where acrolein has been detected in some environmental medium (HazDat 2006). The HazDat information includes data from both NPL and other Superfund sites. Concentrations of acrolein in landfill leachate ranged from 0.07 to 2.1 ppm. In groundwater, the concentrations of acrolein ranged from 0.006 to 1.3 ppm (HazDat 2006). Acrolein is intentionally introduced into irrigation canal and other waterways to control underwater plants and other aquatic life. In 2001, 239,362 pounds (120 tons) of acrolein were used for this purpose in California (EPA 2003). Information on the quantities of acrolein that are released into waterways as a pesticide was not available for other U.S. states.

Acrolein is a gaseous constituent of cigarette smoke and has been detected at levels equivalent to 3–220 μg per cigarette. Acrolein is formed when fats are heated to high temperatures. It has also been found in foods and food products such as raw cocoa beans, volatiles from cooked mackerel and white bread, and vegetable oils, wine, whiskey, and lager beer. Acrolein concentrations in food are typically under 40 μg/g, with most concentrations at 1 μg/g or less (WHO 2002). Acrolein can be produced endogenously as a product of lipid peroxidation (Uchida et al. 1998a, 1998b) and can form protein adducts that have been implicated in atherosclerosis and Alzheimer's disease.

Monitoring data indicate that the general population may be exposed to acrolein through inhalation of contaminated air and through ingestion of certain foods. Other than exposures to acrolein through the inhalation of tobacco smoke, another important source of acrolein exposure may be via the overheating of fats contained in all living matter. Because of the lack of recent, comprehensive monitoring data the average daily intake of acrolein through the consumption of food and drinking water, and the relative importance of each of these sources of exposure, cannot be adequately determined. Estimating the typical level of exposure to acrolein is complicated because acrolein is a common component of tobacco smoke,

and there is wide variation among individuals regarding the frequency and level of exposure to tobacco smoke. Even so, estimates of acrolein exposure in both the general population and for nonsmokers living with a resident smoker are available. A study from Canada (Environment Canada 2000) suggests that the general population is exposed to an average acrolein concentration of 1.3 μg/m^3 with a median value of 0.6 μg/m^3. Based on this average acrolein exposure and an inhalation volume of 20 m^3, it can be estimated that the average adult inhales 26 μg acrolein/day. Nazaroff and Singer (2004) estimated that the daily average inhalation intake of acrolein through environmental tobacco smoke (ETS) over the lifetime of a nonsmoker is 22–50 μg/day for males and 16–36 μg/day for females. These exposure levels for nonsmokers in a household with ETS are approximately 2.2–3.7 times higher than residents living within a household without ETS.

There is potential for exposure to acrolein in many occupational settings as the result of its varied uses and its formation during the combustion and pyrolysis of materials such as wood, petrochemical fuels, and plastics. As a result, it would be difficult to list all the occupations in which work-related exposure to acrolein occurs. It appears that occupational exposure can occur via inhalation and dermal contact.

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 2005j). 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 2005j).

6.2.1 Air

Estimated releases of 152,201 pounds (69 metric tons) of acrolein to the atmosphere from 49 domestic manufacturing and processing facilities in 2004, accounted for about 54% of the estimated total environmental releases from facilities required to report to the TRI (TRI04 2006). These releases are summarized in Table 6-1.

Potential sources of atmospheric release of acrolein include: emissions from facilities involved in the manufacture or use of products containing acrolein; volatilization from treated waters and contaminated waste streams; formation as a photooxidation product of various hydrocarbon pollutants such as propylene, 1,3-butadiene, and other diolefins; emissions from combustion processes; and use in petroleum operations (Eisler 1994; Ghilarducci and Tjeerdema 1995; Graedel et al. 1978; Maldotti et al. 1980; WHO 1991, 2002).

Specific combustion sources include exhaust gas from engines powered by gasoline, diesel or other petrochemical fuels, power plants, burning vegetation (i.e., forest fires), combustion of cellulose materials such as wood, cotton, tobacco, and marijuana, and combustion of polyethylene plastics (EPA 1998a; 1998b; Hodgkin et al. 1982; Jonsson et al. 1985; Lipari et al. 1984; WHO 1991, 2002).

Acrolein is also a pyrolysis product of polyethylene, animal fats and vegetable oils, cellophane, plastics, and paraffin wax (Boettner and Ball 1980; EPA 1980; Potts et al. 1978; Tanne 1983; Wharton 1978). The concentrations of acrolein in emissions from various combustion and pyrolysis processes are listed in Table 6-2.

Recent estimates of the atmospheric loading rate of acrolein from a number of sources in the United States are available. Based on a report on national air pollutant emission trends for 1990–1993, it is estimated that total emissions of acrolein in the United States from all sources was 62,660 tons/year (EPA 1998a). The major sources of acrolein emissions were attributed to mobile (12,271 tons/year) and unspecified stationary (49,400 tons/year) combustion sources. The mobile source emission estimates for acrolein were subdivided into 5,541 tons/year for highway (e.g., automobiles, trucks, buses, and motorcycles) and 6,729 tons/year for off-highway (e.g., airplanes, boats, railway engines, lawnmowers, and off-road vehicles) combustion sources.

Table 6-1. Releases to the Environment from Facilities that Produce, Process, or Use Acrolein[a]

		Reported amounts released in pounds per year[b]						
						Total release		
State[c]	RF[d]	Air[e]	Water[f]	UI[g]	Land[h]	On-site[j]	Off-site[k]	On- and off-site
AL	1	95	No data	0	13	95	13	108
AR	1	0	No data	0	0	0	0	0
CA	1	138	0	0	0	138	0	138
GA	1	31,335	No data	0	0	31,335	0	31,335
IA	3	2,472	No data	0	0	2,472	0	2,472
IL	1	22,030	No data	0	0	22,030	0	22,030
KS	4	6,609	No data	0	0	6,609	0	6,609
LA	3	3,651	1	0	0	3,652	0	3,652
MN	4	3,493	No data	0	0	3,493	0	3,493
MS	1	25,000	No data	0	0	25,000	0	25,000
NC	2	250	No data	0	0	250	0	250
NE	4	2,476	No data	0	0	2,476	0	2,476
OH	1	990	No data	480	0	1,470	0	1,470
TX	17	26,639	0	131,784	1	158,423	1	158,424
VA	2	14,036	0	0	0	14,036	0	14,036
WI	2	12,966	No data	0	0	12,966	0	12,966
WV	1	22	No data	0	0	22	0	22
Total	49	152,201	1	132,264	14	284,466	14	284,480

[a]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.
[b]Data in TRI are maximum amounts released by each facility.
[c]Post office state abbreviations are used.
[d]Number of reporting facilities.
[e]The sum of fugitive and point source releases are included in releases to air by a given facility.
[f]Surface water discharges, waste water treatment-(metals only), and publicly owned treatment works (POTWs) (metal and metal compounds).
[g]Class I wells, Class II-V wells, and underground injection.
[h]Resource Conservation and Recovery Act (RCRA) subtitle C landfills; other on-site landfills, land treatment, surface impoundments, other land disposal, other landfills.
[i]Storage only, solidification/stabilization (metals only), other off-site management, transfers to waste broker for disposal, unknown
[j]The sum of all releases of the chemical to air, land, water, and underground injection wells.
[k]Total amount of chemical transferred off-site, including to POTWs.

RF = reporting facilities; UI = underground injection

Source: TRI04 2006 (Data are from 2004)

Table 6-2. Acrolein in Emissions from Combustion

Source	Concentration	References
Auto exhaust gas		
Gasoline engine	Not detected to 27.7 ppm (detection limit 0.01 ppm); 0–7.79% of total aldehydes, excluding acetone	IARC 1995; Lipari and Swarin 1982; Nishikawa et al. 1987a; Seizinger and Dimitriades 1972; Sigsby et al. 1987; Zweidinger et al. 1988
Gasoline engine	0.16 mg/L gasoline	Grosjean et al. 2001
Diesel engine	2.26 mg/L diesel fuel	
Diesel engine	0.05–0.3 ppm	IARC 1985, 1995; Seizinger and Dimitriades 1972
Ethanol engine	Not detected (detection limit 0.01 ppm)	Lipari and Swarin 1982
Cigarette smoke	3–220 µg/cigarette	Dong et al. 2000; Guerin et al. 1987; Hoffman et al. 1975; Horton and Guerin 1974; Lau et al. 1997; Magin 1980; Manning et al. 1983
Marijuana smoke	92–145 µg/cigarette	Hoffman et al. 1975; Horton and Guerin 1975
Smoke		
Wood	50 ppm	Einhorn 1975
Cotton	60 ppm	
Kerosene	<1 ppm	
Emissions from woodburning fireplaces	21–132 mg/kg wood 20–103 mg/kg wood	Lipari et al. 1984 EPA 1993
Softwood	46.90 mg/kg wood	McDonald et al. 2000
Hardwood	91.23 mg/kg wood	
Hardwood, wood stove	45.54 mg/kg wood	
Emissions from power plants:		
Coal-fueled	0.002 pounds of aldehydes/ 1,000 pounds of fuel	Natusch 1978
Gas-fueled	0.2 pounds of aldehydes/ 1,000 pounds of fuel	
Oil-fueled	0.1 pounds of aldehydes/ 1,000 pounds of fuel	
Pyrolysis of polyvinyl chloride food-wrap film during hot wire cutting	27–151 ng/cut	Boettner and Ball 1980
Emissions from the combustion of polyethylene foam	2–23	Potts et al. 1978
Pyrolysis of polyethylene foam	76–180 ppm	Potts et al. 1978
15 cm above heated cooking oil	2.5–30 mg/m^3	EPA 1980b
Emissions from burning candle	0.18 µg/kg	Lau et al. 1997

Mobile source emissions of acrolein into air for the 48 contiguous states were estimated to be 30,619 tons/year in 1996 (EPA 2001b). This estimate is based on data obtained from the 1996 National Toxics Inventory. The emissions were divided out into onroad (23,393 tons/year) and nonroad (7,226 tons/year) sources. Projected estimates of acrolein emissions in 2007 for onroad and nonroad sources were 11,203 and 5,019 tons/year, respectively. The projections assume a 30 ppm cap on sulfur in gasoline nationwide and implementation of Tier 2 mobile source exhaust emissions standards for light duty vehicles, but do not account for a phase-out of methyl tert-butyl ether (MTBE) in the reformulated gasoline program. Tables 6-3 and 6-4 provide specific release data based on the type of onroad and nonroad mobile sources, respectively. The major onroad acrolein emissions are generated from light duty gasoline vehicles and light duty gasoline trucks. The major generators of acrolein emissions from nonroad sources are nonroad diesel vehicles and airports.

Production of acrolein in air is known to occur through photochemical reactions of volatile organic compounds (VOCs) that are released from a number of differing source types, including solvent and fuel vapors and automobile exhaust (Ghilarducci and Tjeerdema 1995; Liu et al. 1999a; 1999b). Harley and Case (1994) estimated a total daily production of 4,600 kg/day for acrolein from both source emissions and photochemical production in air over the Los Angeles area for August 1987. The estimate is derived from a photochemical air quality model that tracks the transport and chemical reactions of selected VOCs and uses emission rates of carbon monoxide, nitrogen oxides, and VOCs from 800 source types prepared by the California Air Resources Board and the South Coast Air Quality Management District. Based on the modeling, the contribution of direct emissions and photochemical production to the total acrolein emissions are roughly similar. The model also shows that the total daily production rate of 4,600 kg/day results in a predicted range of 1.1–2.1 ppb carbon for the concentration of acrolein in air over the Los Angeles region.

Emissions of acrolein from coal-fired electric utility steam plants in the United States were estimated to be 27 tons/year in 1994 (EPA 1998b). This estimate was based on emission data obtained from 52 of 684 utility plants that were considered to be generally representative of the industry. Acrolein emissions of 34 tons/year were estimated for the year 2010 and are based on projected increases in electrical power usage and changes in fuel choices. However, the projections used to derive the 2010 estimate do not account for factors such as industry restructuring, new particulate and ozone standards, or global climate change programs.

Table 6-3. Estimated Acrolein Emissions from Onroad Mobile Sources in 1996 and 2007[a]

	Acrolein emissions (tons/year)							
Year	LDGV	LDGT	HDGV	MC	LDDV[b]	LDDT	HDDV	Total
1996	10,682	8,822	1,756	281	164	84	1,604	23,393
2007	3,044	5,444	687	363	0	16	1,649	11,203

[a]EPA 2001b
[b]LDDV usage is expected to be phased out by 2007.

HDDV = heavy duty diesel vehicles; HDGV = heavy duty gasoline vehicles; LDDT = light duty diesel trucks; LDDV = light duty diesel vehicles; LDGT = light duty gasoline trucks, categories 1 and 2; LDGV = light duty gasoline vehicles; MC=motorcycles

Table 6-4. Estimated Acrolein Emissions from Nonroad Mobile Sources in 1996 and 2007[a]

	Acrolein emissions (tons/year)						
Year	2-Stroke gasoline	4-Stroke gasoline	Nonroad diesel	Marine diesel	Railroad	Airports	Total
1996	511	470	4,996	77	167	1,006	7,226
2007	385	297	2,836	84	139	1,277	5,019

[a]EPA 2001b

Emissions of acrolein into air from paper and wood product manufacturing in Canada have been estimated for the year 1995. Acrolein emissions ranged from 3,208 to 25,664 kg/year (3.544–28.289 tons/year) for oriented strand board producers and from 3,747 to 18,735 kg/year (4.130–20.651 tons/year) for pulp and paper (kraft) mills (WHO 2002).

The intentional release of acrolein into irrigation channels as an herbicide and molluscicide also results in the volatilization of acrolein into air (Eisler 1994; EPA 2003; Ghilarducci and Tjeerdema 1995). In the San Joaquin Valley of California, it was reported that 194,668 pounds (97.3 tons) of acrolein were emitted into the air from agricultural uses of the pesticide in 2001, which amounted to 1.4% of the total pesticide emissions from this region (CEPA 2002).

EPA (1983) estimated the total loading rate of acrolein in 1978 for the United States to be 91,450 pounds (45.7 tons) from facilities involved in its production and use as a chemical intermediate. Loading rates of acrolein into the environment from various industrial sources were as follows: acrylic acid manufacturers, 15,175 pounds (7.59 tons); refined acrolein and glycerin manufacturers, 55,660 pounds (27.8 tons); methionine manufacturers, 18,150 pounds (9.08 tons); and miscellaneous intermediate uses, 2,420 pounds (1.21 tons). These loading rates were based on a total production volume of 350 million pounds (175,000 tons) for acrolein with 87% of this volume consumed in the production of acrylic acid and its derivatives.

6.2.2 Water

Estimated releases of 1 pound (<0.01 metric tons) of acrolein to surface water from 49 domestic manufacturing and processing facilities in 2004, accounted for <0.1% of the estimated total environmental releases from facilities required to report to the TRI (TRI04 2006). These releases are summarized in Table 6-1.

Acrolein may be released to water in effluents from its manufacturing plants and use facilities (see Section 5.3 for specific information regarding uses) and from its direct application to water as a broad-range biocide in irrigation canals, cooling towers, water treatment basins, and process water circuits (Eisler 1994; EPA 2003; Ghilarducci and Tjeerdema 1995; IARC 1985; Lue-Hing et al. 1981; Nordone et al. 1996a, 1996b; WHO 1991; WSSA 1983).

The amount of acrolein released from industrial operations to publicly owned treatment works (POTW) in the U.S. waters in 1986 was estimated to be 1,645,600 pounds/year (823 tons/year) (EPA 1991). However, it is reported that a large portion of the acrolein that is received by POTWs is removed before discharge in effluent streams, with 5% released to surface waters, 0–5% to air, and 10% to sludge (EPA 1991).

Data on the release of acrolein into water as a consequence of its use as a pesticide are available only for the state of California. It is reported that usage of acrolein in California declined from 328,238 pounds (164 tons) in 1999 to 290,180 pounds (145 tons) and 233,928 pounds (117 tons) in 2000 and 2001, respectively (EPA 2003). The predominant use of acrolein is as an aquatic herbicide with releases into rights-of-way (i.e., irrigation canals) and other water areas amounting to 326,767 pounds (163 tons), 297,320 pounds (149 tons), and 239,362 pounds (120 tons) in 1999, 2000, and 2001, respectively. The decrease in acrolein usage is due to changes in the permitting process required prior to acrolein treatment of irrigation canals instituted in 2001. Once irrigation districts in California work through the new permitting process, it is expected that future usage of acrolein will be comparable to acrolein usage reported for 1999–2000.

6.2.3 Soil

Estimated releases of 14 pounds (<0.01 metric tons) of acrolein to soils from 49 domestic manufacturing and processing facilities in 2004, accounted for <0.1% of the estimated total environmental releases from facilities required to report to the TRI (TRI04 2006). An additional 132,264 million pounds (~60 metric tons), constituting about 46% of the total environmental emissions, were released via underground injection (TRI04 2006). These releases are summarized in Table 6-1.

The occurrence of acrolein in soil at one hazardous waste site in the United States and leachate from several municipal landfills provides evidence that this compound has been released to soil as the result of land disposal of some organic wastes. No data were located regarding the amount of acrolein released to soil.

6.3 ENVIRONMENTAL FATE

6.3.1 Transport and Partitioning

Acrolein is relatively unstable in the atmosphere; therefore, transport within the atmosphere is expected to be limited. The relatively high vapor pressure of acrolein (274 mm Hg at 25 °C [Daubert and Danner 1987]) suggests that this compound will not partition from the vapor phase to particulates in the atmosphere. Occurrence of acrolein in rainwater (Grosjean and Wright 1983; Nishikawa et al. 1987b) indicates that this compound may be removed from the atmosphere by washout.

Volatilization is expected to be a significant removal process for any acrolein released to surface waters (Nordone et al. 1996a, 1996b). Based on a measured Henry's law constant of 1.22×10^{-4} atm-m^3/mol at 25 °C (Gaffney et al. 1987), the volatilization half-life from a model river 1 m deep, flowing 1 m/set with a wind speed of 3 m/second was estimated to be 23 hours using the method of Thomas (1982). Veith et al. (1980) measured a bioconcentration factor (BCF) of 344 for acrolein in bluegill sunfish; however, this may be an overestimate, since total 14C was measured in the fish, which may have resulted in the measurement of acrolein metabolites. A BCF of 0.6 was estimated for acrolein using a linear regression equation based on a log octanol/water partition coefficient (K_{ow}) of -0.01 (Bysshe 1982; Hansch and Leo 1995). These BCFs, as well as the relatively high water solubility of this compound, suggest that acrolein does not bioconcentrate significantly in aquatic organisms. Acrolein did not accumulate in leaf lettuce after both single and multiple applications in irrigation water at a concentration of 75 ppm (Nordone et al. 1997). Acrolein residues in the lettuce fell to 0% within 53 days following the initial application.

Using a linear regression equation based on log K_{ow} data (Lyman 1982), an adsorption coefficient (K_{oc}) of 24 was estimated, which suggests that adsorption of acrolein to suspended solids and sediments in water would not be significant. This does not take into account the reactivity of acrolein which could lead to the removal of acrolein from water through chemical binding of the compound to dissolved or suspended organics in water and sediments. The relatively low estimated K_{oc} value suggests that acrolein will be highly mobile in soil and that this compound has the potential to leach significantly (Swann et al. 1983).

The relatively high vapor pressure of acrolein and its volatility from water suggest that this compound will evaporate rapidly from soil surfaces and that volatilization is probably a major removal process from soil. The relatively low K_{oc} value for acrolein indicates high mobility in soil and suggests that this

compound has the potential to leach significantly (Swann et al. 1983). Degradation processes and volatilization, however, are expected to significantly retard movement of acrolein through soil.

6.3.2 Transformation and Degradation

6.3.2.1 Air

The dominant removal process for acrolein in ambient air is predicted to be reaction with photochemically generated hydroxyl radicals in the troposphere. The atmospheric half-life for acrolein is estimated to be 15–20 hours, based on experimentally determined hydroxyl radical reaction rate constants ranging between 1.90×10^{-11} and 2.53×10^{-11} cm^3/molecules-sec at 25–26 °C and an average ambient hydroxyl radical concentration of 5.0×10^5 molecules/cm^3 (Atkinson 1985). Acrolein reacts with hydroxyl radicals as both an olefin and an aldehyde (Grosjean 1990). Products of this reaction include carbon monoxide, formaldehyde, glyoxal, and glycolaldehyde. In the presence of nitrogen oxides, products include peroxynitrate, nitric acid, glycidaldehyde, malonaldehyde, and 3-hydroxypropanaldehyde (Edney et al. 1986; Grosjean 1990; Liu et al. 1999b).

Direct photolysis in the ambient atmosphere occurs but is expected to be of minor importance. Gardner et al. (1987) reported that the quantum yields for irradiation of acrolein at low air pressures were 0.0066 at 313 nm and 0.0044 at 334 nm. The authors used a computer analysis of their photodissociation data to estimate the half-life of acrolein to be 10 days in the lower troposphere and <5 days in the upper troposphere.

Experimental data indicate that reaction of acrolein with ozone ($k=2.8 \times 10^{-19}$ cm^3/molecules-sec at 25 °C; half-life, 59 days) or nitrate radicals ($k=5.9 \pm 2.8 \times 10^{-16}$ cm^3/molecules-sec at 25 °C; half-life, 16 days) in the troposphere would be too slow to be environmentally significant (Atkinson 1985; Atkinson et al. 1987). The fate of acrolein in indoor air is expected to be different from its fate in outdoor air because of differences in the concentrations of oxidants in indoor air compared to outdoor air and the possibility of other mechanisms of removal.

6.3.2.2 Water

Low concentrations of acrolein may degrade in natural water by either aerobic biodegradation or reversible hydration to β-hydroxypropionaldehyde, which subsequently undergoes aerobic biodegradation (Bowmer and Higgins 1976; EPA 1979; Ghilarducci and Tjeerdema 1995; Tabak et al. 1981). Acrolein at

a concentration of 5–10 mg/L was completely degraded in 7–10 days in a static culture flask screening procedure (Tabak et al. 1981). Acrolein applied to surface waters at application rates suggested for herbicidal use can persist up to 6 days (WSSA 1983). Bowmer and Higgins (1976) measured acrolein removal in both laboratory water and in field experiments using irrigation channels. Their studies suggested that the degradation of the hydration product of acrolein, 3-hydroxypropanal, occurs after the concentration of acrolein falls below 2–3 ppm. The degradation of 3-hydroxypropanal was also preceded by a 100-hour lag period, suggesting that biodegradation was occurring through the action of acclimated cultures.

In buffered laboratory water, acrolein reached its equilibrium apparently with β-hydroxypropionaldehyde in approximately 300 hours (92% β-hydroxypropionaldehyde, 8% acrolein); in irrigation channels, acrolein removal was complete. Half-lives were reportedly <1–3 days in surface water, but values were for the combined effect of degradation and volatilization (Bowmer and Higgins 1976; Bowmer et al. 1974). Kissel et al. (1978) measured acrolein removal in buffered laboratory water and natural river water using both chemical analysis methods and bioassays. Complete hydrolysis (which according to the authors includes hydration to 3-hydroxypropanal) occurred within 150, 120–180, and 5–40 hours in buffered solutions at 22 °C and pH 5, 7, and 9, respectively. Based on fish kill bioassays in natural river water at pH 8.1, >93% degradation of acrolein occurred within 6 days. The half-lives of acrolein in aerobic test systems that were treated at an application rate of 15 mg/L were 9.5 hours in water and 7.6 hours in sediment (Smith et al. 1995). The half-lives of acrolein in anaerobic test systems treated at the same rate were 10.3 hours in water and approximately 10 days in sediment. Degradation products included 3-hydroxypropanal, acrylic acid, and allyl alcohol, which indicate that both hydrolysis and biodegradation contributed to the degradation of acrolein during this study.

Jacobson and Smith (1990) studied the dissipation of acrolein, applied at the highest recommended rate according to the label, to achieve a 15 ppm concentration for a 2-hour duration in an irrigation canal and a lateral of the canal, which was infested with aquatic plants. The dissipation half-lives for acrolein in the irrigation and lateral canals were 275 and 64 minutes, respectively. No acrolein residues were detected (detection limit, 0.01 ppm). No residues of 3-hydroxypropanal were detected (detection limit, 2.0 ppm) in any of the water samples from either canal. These data suggest that acrolein will not persist for moderate or long periods of time in aerobic aquatic environments and that hydration of acrolein may not be an important degradation pathway for acrolein (Jacobson and Smith 1990). The decay rate constants for acrolein applied to irrigation canals have been reported to be similar (0.14–0.21) regardless of the difference in time-concentration regimens (100 μg/L for 48 hours to 15,000 μg/L for several hours)

(Eisler 1994). The half life of acrolein, applied at a flow rate of 3,964 L/second to achieve 15 ppm for 1 hour, was 10.2 hours in a weedy canal and 7.3 hours in a nonweedy canal (Nordone et al. 1996b; Rathbun 1998). The concentration of acrolein was 25 µg/L in samples from the Columbia River collected 65 km from where it was applied at a concentration of 125 µg/L (Eisler 1994). Nordone et al. (1996a) studied the dissipation of acrolein applied to agriculture canals with flow rates of 142, 283, and 453 L/second to achieve target concentrations of 7.5, 11.6, and 10.4 ppm, respectively. These authors concluded that typical application of acrolein as an aquatic herbicide in agricultural canals does not result in the introduction of acrolein into natural receiving waters 2.7 km downstream.

The ultraviolet (UV) spectrum of acrolein in hexane shows moderate absorption of UV light in the environmentally significant range (wavelengths >290), suggesting that acrolein might undergo photolysis in natural waters; however, hydration of acrolein destroys the chromophores that absorb UV light (EPA 1979), and the equilibrium appears to be far on the side of the hydration product. Thus, the potential for direct photolysis of acrolein in natural waters is probably slight. Oxidation of small amounts of acrolein in natural waters would not be environmentally significant; however, highly concentrated acrolein solutions (i.e., spills) may be polymerized by oxidation or hydration processes (EPA 1979). Insufficient data are available regarding anaerobic biodegradation to establish the significance of this process as a removal mechanism or to determine the rate at which such a process would proceed. This information would be particularly useful in determining the fate of acrolein under conditions frequently encountered in groundwater and in landfills.

Based on the reactivity and nucleophilicity of acrolein, it is expected that acrolein has the potential to react with dissolved and suspended organics in water. This removal process would become increasingly important for determining the fate of acrolein in water as the concentration of organics in water increased. However, no studies have been conducted to describe this possible route for removal of acrolein from water.

6.3.2.3 Sediment and Soil

Experimental data specifically pertaining to the degradation or transformation of acrolein in soil were not located. Results of studies in aquatic systems suggest that acrolein, at low concentrations, may be subject to aerobic biodegradation in soil or transformation via hydration followed by aerobic biodegradation of the hydrated product (see Section 6.3.2.2).

Since acrolein is a very reactive compound, abiotic processes, such as oxidation or conjugation with organic matter in soils, may be the most important degradation processes. However, no information could be located for these possible acrolein reaction pathways in soil.

6.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

Reliable evaluation of the potential for human exposure to acrolein depends in part on the reliability of supporting analytical data from environmental samples and biological specimens. Concentrations of acrolein 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 acrolein 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 acrolein in a variety of environmental media are detailed in Chapter 7.

6.4.1 Air

The atmospheric concentrations of acrolein have been measured in several locations, and the most comprehensive monitoring studies are discussed below. Data for 2004 obtained from EPA's Air Quality System (AQS) database show average concentrations of acrolein at various monitoring stations ranging from 0.3 to 2.048 ppb carbon (0.5–3.186 ppbv), with maximum values ranging from 0.3 to 3.6 ppb carbon (0.5–5.6 ppbv) (EPA 2004a). Data obtained for 1996 show similar average concentrations for acrolein, ranging from 0.05 to 3.2 ppb carbon (0.08–5.6 ppbv) with maximum values ranging from 0.5 to 11.46 ppb carbon (0.8–17.82 ppbv). Lower average concentrations of 0.05–0.64 ppb carbon (0.08–1.00 ppbv) for acrolein (maximum values ranging from 0.05 to 9.9 ppb carbon [0.08–15 ppbv]) were found for 2000. The National Air Toxics Monitoring Program (EPA) reported peak concentrations for acrolein of <1 ppbv at 12 monitoring sites, with one site reporting a peak concentration between 1 and 5 ppbv (Mohamed et al. 2002). These data were obtained in 1996 at 13 monitoring sites in New Jersey, Louisiana, Texas, and Vermont.

In the National Air Quality and Emissions Trend Report for 1998, the concentrations of acrolein in ambient air averaged 0.20 and 0.12 $\mu g/m^3$ (0.086 and 0.052 ppb) for urban and rural locations, respectively, based on emission and monitoring data obtained in 1996 (EPA 1998c). In the report submitted for 1999, it was noted that during the period of 1994–1999, the concentrations of acrolein are either showing no trend or are trending upwards in concentration in six urban areas (EPA 1999).

Information on trends in acrolein concentrations in rural areas was available for only one rural location, showing a downward trend within the same time period.

A concentration of acrolein in ambient air in California has been estimated to average 0.36 μg/m^3 (0.16 ppb) and is based on emissions and census tract data obtained in 1999 (Morello-Frosch et al. 2000). Estimated concentrations of acrolein in ambient air for the San Francisco Area in 1990–1991 ranged from 0.012 to 0.28 μg/m^3 (0.005–0.12 ppb) (Rosenbaum et al. 1999). Ambient air concentrations of acrolein at the Oakland-San Francisco Bay Bridge Toll Plaza obtained in April 2001 showed differing concentrations between morning and evening measurements. Acrolein concentrations ranged from 0.096 to 0.140 μg/m^3 (0.041–0.060 ppb) during the morning commute, which were lower than the concentrations of 0.031–0.047 and 0.058–0.079 μg/m^3 (0.013–0.020 and 0.025–0.034 ppb) during two evening monitoring periods taken on consecutive days (Destaillats et al. 2002). Altshuller and McPherson (1963) and Renzetti and Bryan (1961) determined that acrolein levels in air samples collected in Los Angeles, California, during 1960–1961 averaged between 5 and 8 ppb. Air samples collected in the Los Angeles Basin over a 12-week period during 1968 contained levels ranging between none detected to 18 ppb, although most values ranged between 0.9 and 9 ppb (IARC 1985).

Acrolein has been detected in indoor air and its concentrations are summarized in Table 6-5. The concentrations of acrolein range from <0.05 to 29 μg/m^3 (<0.02–12 ppb) in residential homes (CARB 1992; Highsmith and Zweidinger 1988; WHO 2002). Acrolein concentrations are found to be typically higher in indoor air when comparing paired indoor/outdoor samples taken at a site (CARB 1991; WHO 1991, 2002)

Acrolein has been detected in air samples collected at 6 of the 32 hazardous waste sites where acrolein has been detected in some environmental medium (HazDat 2006). The HazDat information includes data from both NPL and other Superfund sites. Concentrations of acrolein in outdoor air ranged from 0.01–9 to 0.013–106 ppbv in onsite and offsite sampling, respectively (HazDat 2006).

6.4.2 Water

Data from the EPA STORET Data Base indicate that acrolein has a low frequency of occurrence in waste water streams, ambient surface water, and groundwater in the United States (EPA 2007; Staples et al. 1985). Acrolein has not been found as a contaminant of drinking water (EPA 1980; Krill and Sonzogni 1986; Otson 1987; WHO 2002). Grosjean and Wright (1983) detected acrolein, in combination with

Table 6-5. Acrolein Concentrations in Indoor Air

Type of buiilding	Concentration	Location	References
Residential	0.36–1.95 ppbv[a] (0.85–4.62 µg/m^3)[b]	Raleigh, North Carolina	Highsmith and Zweidinger 1988
Residential	NQ–29 µg/m^3[c] 7.1 µg/m^3 (average)	Woodland, California	CARB 1992
Residential	0.4–8.1 µg/m^3	Windsor, Ontario	WHO 2002
Residential	<0.05–5.4 µg/m^3	Hamilton, Ontario	
Residential	16–23 µg/m^3	Toronto, Ontario	
Restaurants	8–18 ppb (19–43 µg/m^3)[b]	Zürich, Switzerland	Weber et al. 1979
Student lounge			
Non-smoking	0.8–1.6 µg/m^3	Bounds Green, United Kingdom	Williams et al. 1996
Smoking	6.4 µg/m^3		
Tavern	21–24 µg/m^3	Research Triangle Park, North Carolina	Löfroth et al. 1989

[a]ppbv = parts per billion by volume
[b]Converted measurement in ppbv to µg/m^3, assuming an ambient temperature of 20 °C and an atmospheric pressure of 1,013 mbars.
[c]NQ = not quantifiable below detection limit of 2.0 µg/m^3

acetone, at a concentration of 0.05 μg/mL (50 ppb) in rain water collected in Los Angeles, California; however, these compounds were not detected in rain water samples collected in four less densely populated sites in California.

More recently, acrolein has been detected in surface water and groundwater samples collected at 4 and 15 of the 32 hazardous waste sites, respectively, where acrolein has been detected in some environmental medium (HazDat 2006). The HazDat information includes data from both NPL and other Superfund sites. Concentrations of acrolein in landfill leachate ranged from 0.07 to 2.1 ppm (HazDat 2006). In groundwater, the concentrations of acrolein ranged from 0.006 to 1.3 ppm (HazDat 2006).

6.4.3 Sediment and Soil

Acrolein was identified in sediment/ soil/water samples collected from Love Canal in Niagara Falls, New York (Hauser and Bromberg 1982); however, no quantitative data were available.

More recently, acrolein has been detected in soil and sediment samples collected at 1 and 2 of the 32 hazardous waste sites, respectively, where acrolein has been detected in some environmental medium (HazDat 2006). The HazDat information includes data from both NPL and other Superfund sites. One soil sample site was found to have an acrolein concentration of 100 ppm (HazDat 2006).

6.4.4 Other Environmental Media

Acrolein can be produced in endogenously as a product of lipid peroxidation (Uchida et al. 1998a, 1998b) and can form protein adducts that have been implicated in atherosclerosis (Uchida et al. 1998b) and Alzheimer's disease (Calingasan et al. 1999). Acrolein has been identified in foods and food components such as raw cocoa beans, chocolate liquor, souring salted pork, fried potatoes and onions, raw and cooked turkey, and volatiles from cooked mackerel, white bread, raw chicken breast, ripe Arctic bramble berries, heated animal fats and vegetable oils, and roasted coffee (Cantoni et al. 1969; EPA 1980, 1985; Feron et al. 1991; IARC 1985; Umano and Shibamoto 1987). Feron et al. (1991) reported concentrations of acrolein of <0.01–0.05 ppm in various fruits and up to 0.59 ppm in cabbage, carrots, potatoes, and tomatoes. The concentration in food is <40 μg/g and, in most instances, is <1 μg/g (WHO 2002). The acrolein concentrations in heated fats and oils and in the headspace above these materials increase with increasing cooking temperature (Casella and Contursi 2004; Ghilarducci and Tjeerdema 1995; WHO 2002). For example, peanut oil heated for 2 hours at 110, 145, and 200 °C resulted in the production of acrolein at concentrations of 0.2, 2.7, and 24 μM, respectively (Casella and Contursi 2004). In

comparison to other oils, peanut oil was found to have the lowest production of acrolein after 2 hours of heating at 145 °C, with higher concentrations found in sunflower (2.9 μM), corn oil (4.3 μM), and olive oil (9.3 μM) when heated under the same conditions. Sufficient data are not available to establish the level of acrolein typically encountered in these foods. Trace levels of acrolein have been found in wine, whiskey, and lager beer (IARC 1985). Further information regarding the occurrence of acrolein in food and related products is provided by EPA (1980).

Acrolein is a gaseous constituent of tobacco and marijuana smoke, occurring in both mainstream and sidestream smoke (Ayer and Yeager 1982; Hoffman et al. 1968; Holzer et al. 1976; Rylander 1974; Weber-Tschopp et al. 1977). The level of acrolein in sidestream smoke has been found to be notably higher (12 times higher) than in mainstream smoke (Triebig and Zober 1984). The amount of acrolein emitted in tobacco smoke varies depending upon the kind of cigarette, smoking conditions, puff volume, puff rate, nature, and type of tobacco, as well as a number of other extraneous factors (Holzer et al. 1976). Smoke from various cigarettes has been found to contain 3–220 μg acrolein per cigarette (Dodson 1994; Hoffman et al. 1968; Horton and Guerin 1974; Magin 1980; Manning et al. 1983). Smoke from a marijuana cigarette was also found to contain 92–145 μg/cigarette (Hoffman et al. 1968; Horton and Guerin 1974). Studies performed to determine the concentration of acrolein in smoke-filled rooms (Rylander 1974; Triebig and Zober 1984; Weber-Tschopp et al. 1977) indicate that the concentration of acrolein in indoor air is highly dependent upon such factors as the number of cigarettes smoked, rate at which the cigarettes are smoked, size of the room, number of people in the room, and type of ventilation. Acrolein levels measured in various settings where people were smoking are: cafe, 30–100 ppb; train, 10–120 ppb; car with three smokers (windows open), 30 ppb (average); car with three smokers (windows closed), 300 ppb (average); restaurant, 3–13 ppb; tavern, 5–18 ppb; and cafeteria, l–10 ppb (Triebig and Zober 1984).

6.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

The general population may be exposed to acrolein through inhalation of contaminated air, inhalation of cigarette smoke, and through ingestion of certain foods. Widespread exposure occurs due to the formation of acrolein during the overheating of fats. Acrolein has been detected in the vapor of rapeseed oil, which is used frequently in Chinese wok cooking (Pellizzari et al. 1995). Primary factors influencing the level of exposure to acrolein via inhalation are: location (urban versus rural), duration and frequency of exposure to tobacco smoke, concentration of tobacco smoke, duration and frequency of exposure to high concentrations of vehicle exhaust (e.g., in parking garages, in heavy traffic), occupational exposure,

and downwind distance of residence or work site relative to stationary point sources. Primary factors influencing the level of exposure to acrolein via ingestion are diet and volume of intake, which is typically related to age and sex.

Because of the lack of recent comprehensive monitoring data for acrolein in water and food, the average daily intake of acrolein and the relative importance of each source of exposure cannot be determined. However, probabilistic estimates of 24-hour time-weighted concentrations of acrolein in air have been used to assess human exposures to acrolein in the Canadian population (Environment Canada 2000; WHO 2002). Mean and median estimates of acrolein concentration of 1.3 and 0.6 μg/m^3 (0.56 and 0.26 ppb), respectively, were derived, with a 95% percentile value of 5.0 μg/m^3 (2.1 ppb). The estimate uses measured data on acrolein concentrations obtained between 1989 and 1996 for outdoor air in rural, suburban, and urban sites and indoor air measurements taken in 40 homes between 1991 and 1993. The exposure estimate assumes both a mean time of 3 hours spent outdoors and that the general population is exposed to concentrations of acrolein similar to those in indoor air of their homes. Based on the mean estimate for acrolein concentration and an inhalation volume of 20 m^3 of air per day, it is estimated that an average adult will inhale 26 μg acrolein/day.

ETS is a major source of acrolein exposure for many individuals in the general population. Nazaroff and Singer (2004) estimate that in 2000, between 31 and 53 million nonsmokers in the United States were exposed to acrolein concentrations in indoor air ranging from 1.6 to 3.6 μg/m^3 in households where ETS is generated by one or more individuals residing in the same household. Between 15 and 25 million of the affected number of nonsmokers are adults. Based on the lifetime average for the volume of inspired air of 14 m^3/day for males and 10 m^3/day for females, it is estimated that the inhalation intake of acrolein through inspiration of ETS over a lifetime is 22–50 μg/day for males and 16–36 μg/day for females. Assuming that the exposure data obtained from the Canadian study (Environment Canada 2000) discussed above are representative of exposures of residents in the United States to acrolein in households without ETS, then it is estimated that the inhalation intake of acrolein for nonsmokers exposed to ETS in the residence is 2.2–3.8 times greater for both males and females than in households without ETS. This comparison is based on inhalation intakes of acrolein for males and females in non-ETS households of 18 and 13 μg/day, respectively, that are based on an estimated mean acrolein concentration in air of 1.6 μg/L taken from the Canadian study (Environment Canada 2000) and on the average daily inhalation volumes of air for males and females given by Nazaroff and Singer (2004).

Everybody is exposed to small amounts of endogenous acrolein. This endogenous acrolein is formed as a consequence of the peroxidation of lipid membranes and metabolism of α-hydroxy amino acids and polyamines (Alarcon 1970; Uchida et al. 1998a; WHO 2002). Acrolein has also been shown to be formed by phagocytes in response to infection or inflammation and as a result of the progression of Alzheimer's disease and atherosclerosis (Anderson et al. 1997; Calingasan et al. 1999; Gómez-Ramos et al. 2003; Uchida et al. 1998b). Due to the reactivity of acrolein with biomolecules, especially thiol-containing proteins and glutathione, the formation of acrolein *in vivo* has been measured as the byproducts of the reaction of acrolein with these biomolecules. These biomarkers of *in vivo* acrolein formation include acrolein-protein adducts and the urine metabolites, S-(3-hydroxypropyl)mercapturic acid and S-(2-carboxyethyl)mercapturic acid (Calingasan et al. 1999; Li et al. 2004; WHO 2002). However, studies to correlate the concentrations of these biomarkers with *in vivo* acrolein production in humans have yet to be conducted.

According to a National Occupational Exposure Survey (NOES) by NIOSH between 1981 and 1983, an estimated 1,298 workers (including 5 females) in 37 facilities in the United States are occupationally exposed to acrolein (NIOSH 1988). This is a tentative estimate and is subject to change as further information regarding trade name compounds becomes available. There is potential for exposure to acrolein in many occupational settings as the result of its varied uses and its formation during the combustion and pyrolysis of materials such as wood, petrochemical fuels, and plastics. As a result, it would be difficult to list all the occupations in which work-related exposure to acrolein occurs. Some of these occupations include those involved in the production of acrylates, methionine, perfumes, plastics, refrigerants, rubber, or textile resins (Ghilarducci and Tjeerdema 1995).

Acrolein has been detected in workplace air at a number of locations (Ahrenholz and Egilman 1983; Apol 1982; IARC 1985; Tharr and Singal 1986; Treitman et al. 1980; Woskie et al. 1988). Acrolein concentrations of 0.057–0.085 ppm were detected during system testing conducted as part of a submarine overhaul in Portsmouth Naval Shipyard in Portsmouth, New Hampshire (Tharr and Singal 1986). Ahrenholz and Egilman (1983) reported >0.0044–0.18 ppm acrolein in the wire line department of Rubbermaid Inc. in Wooster, Ohio, and Apol (1982) reported >0.06 ppm in molding areas of Gerlinger Casting Corp. in Salem, Oregon.

The concentrations of acrolein were 0.01 mg/m^3 (0.004 ppm) in the air of a food factory, 0.59, 0.31, 0.15, 0.16, and 0.06 mg/m^3 (0.25, 0.13, 0.064, 0.069, and 0.026 ppm) in the air of five restaurant kitchens, and 0.02 mg/m^3 (0.009 ppm) in the air of two bakeries (Vainiotalo and Matveinen 1993). Henriks-Eckerman

et al. (1990) reported acrolein was emitted from coated steel plates heated to 350 °C. This indicates that workers involved in welding or heating painted metal may be exposed to acrolein

Firefighters are at risk to exposure to acrolein when battling house fires and wild fires (Ghilarducci and Tjeerdema 1995; Gochfeld 1995; Lees 1995; Materna et al. 1992). The concentrations of acrolein measured in a NIOSH house fire study ranged from not detected to 3.2 ppm, with half of the exposures exceeding the 0.3 ppm short-term exposure limit. During a study of over 224 structural fires, firefighters were exposed to acrolein at levels as high as 6.9 mg/m^3 (2.3 ppm) (Ghilarducci and Tjeerdema 1995). The concentration of acrolein in a single sample collected during a wildfire was reported to be 0.23 ppm (Lees 1995; Materna et al. 1992).

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. 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).

For children living in a residence where one or more individuals smokes some form of tobacco product, long-term exposure to environmental tobacco smoke (ETS) and the compounds therein are expected, which can cause a number of health effects (WHO 1999). Consequently, because of the acrolein content in ETS it is expected that the largest source of acrolein exposure for children living with a smoker is through inhalation of ETS. Information on exposures of acrolein through ETS that are specific for children living in the United States could not be identified. However, based on data obtained from Nazaroff and Singer (2004), it is estimated that individuals who do not smoke over their lifetimes but reside with one or more individuals who do smoke, will intake between 22 and 50 μg acrolein/day for males and between 16 and 36 μg acrolein/day for females through the inhalation of acrolein in ETS over

their lifetimes. This amounts to 2.2–3.7 times greater exposure to acrolein for these children than for children who are not exposed to ETS over their lifetimes (see Section 6.5). For children without exposures to ETS, their main exposures to acrolein are expected to be similar to those noted for the general population in Section 6.5 in air. Estimates of the concentration in the total diet of children in the United States were not located in the available literature. Therefore, no estimate of daily acrolein intake from food can be made. Because of the lack of recent comprehensive monitoring data for acrolein in water, the average daily intake of acrolein and the relative importance of this source of exposure cannot be determined.

6.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

Those segments of the general population with potentially high exposure to acrolein from exogenic sources include people who come in frequent or prolonged contact with tobacco or marijuana smoke, people who are occupationally exposed, and people who live or work near dense traffic areas, in smoggy areas (e.g., Los Angeles), or downwind from stationary point sources. Acrolein uptake from cigarette smoke for individuals working in bars and taverns can range from 15 to 1,830 μg/day, based on an 8-hour shift, a respiration volume of 20 m^3 air per day, and a concentration range of acrolein in air of 2.3–275 $\mu g/m^3$ (IARC 1995). Individuals who work or reside near irrigation canals and other bodies of water that are undergoing treatment with acrolein to eliminate unwanted plants or aquatic life are at risk for exposure to acrolein. Individuals living near some land-fills and other waste sites may be exposed to acrolein in ambient air or drinking water. For example, acrolein has been measured at concentrations of 1.3, 4.24, and 43 ppb in groundwater obtained from private wells offsite from two NPL landfills (HazDat 2006).

Patients receiving oxazaphosphorine drugs, such as cyclophosphamide and ifosfamide, for their cancer treatment are at risk for exposure to acrolein, a metabolite of these drugs (Furlanut and Franceschi 2003; Kaijser et al. 1993). For example, patients receiving cyclophosphamide at a dose of 60 mg/kg body weight/day by 1-hour infusion for 2 consecutive days had peak blood acrolein concentrations ranging between 6.2 and 10.2 μM (Ren et al. 1999). The urinary clearance of acrolein from blood during therapy results in concentrations of acrolein in urine ranging from 0.3 to 406.8 nM, depending on urine volume (Takamoto et al. 2004). This range of urinary acrolein concentrations is sufficient to result in acrolein-induced urotoxicities that must be reduced through increasing urine volume during treatment with diuretics or receiving uroprotective drugs during treatment (Kaijser et al. 1993).

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 acrolein 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 acrolein.

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. Physical and chemical property data are essential for estimating the partitioning of a chemical in the environment. Physical and chemical property data are available for acrolein and are sufficient for estimating the environmental fate of acrolein (Amoore and Hautala 1983; Daubert and Danner 1987; Gaffney et al. 1987; Hansch and Leo 1995; HSDB 2007; Lewis 1997; Lide 2000; O'Neil 2001; Seidell 1941; Tomlin 2003; Verschueren 2001).

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 2004, became available in May of 2006. This database is updated yearly and should provide a list of industrial production facilities and emissions.

Data regarding the production methods for acrolein, production facilities, use, and disposal are adequate (Etzkorn et al. 2002; SRI 2004). Data regarding current gross estimates of production volumes and capacities are available (Arntz et al. 2002; EPA 1989b; Etzkorn et al. 2002; Hess et al. 1978; HSDB 2007; IARC 1995; IUR 2002; Lewis 1997; OHM-TADS 1988; O'Neil 2001; TRI04 2006; Windholz et al. 1983). Production data may be difficult to obtain, since many companies desire to maintain their confidentiality. Information regarding import/export of acrolein could not be located. Data regarding

release of acrolein into air are available for mobile and stationary sources (CEPA 2002; EPA 1983, 1998a, 1998b, 2001b; WHO 2002). Harley and Case (1994) estimated the relative contributions of source emissions and photochemical production of acrolein to the amount of acrolein in the air over Los Angeles. However, estimates could not be located on the contribution of photochemical production of acrolein to acrolein concentrations in the ambient air in other regions of the United States, nor were data available on the expected seasonal variations in photochemical production of acrolein. Limited data are available on the release of acrolein to publicly owned treatment works and the release of acrolein as a pesticide to irrigation waters in California (EPA 1991, 2003), but no data could be located on release of acrolein to soil. Use, release, and disposal information is useful for determining where environmental exposure to acrolein may be high. Determining the percentage of acrolein used as a captive intermediate (i.e., consumed in closed processes in which the compound is not isolated) rather than as an isolated, refined product is important in estimating the amount of release to the environment from stationary, noncombustion-related sources. An estimate of the amount of acrolein released from stationary sources would be useful in establishing the relative importance of each source of acrolein. Even with the availability of information on the production, use, and disposal of acrolein, the amounts released would be difficult to estimate, since major factors contributing to its occurrence in the environment are its formation as a product of the photochemical degradation of other atmospheric pollutants and its release in emissions from a wide variety of combustion processes.

Environmental Fate. The environmental fate of acrolein in air is well studied (Atkinson 1985; Atkinson et al. 1987; Gardner et al. 1987; Grosjean 1990). Given that acrolein occurs in the atmosphere from both natural and anthropogenic sources (Eisler 1994; EPA 1998a, 1998b; Ghilarducci and Tjeerdema 1995; Graedel et al. 1978; Hodgkin et al. 1982; Jonsson et al. 1985; Lipari et al. 1984; Liu et al. 1999a, 1999b; Maldotti et al. 1980; WHO 1991, 2002), it would be helpful to have estimates of the relative contributions of these sources to acrolein concentrations in air, especially the contribution of the photochemical production of acrolein. Data on the dissipation and degradation of acrolein in water are available (Bowmer and Higgins 1976; Bowmer et al. 1974; EPA 1979; Ghilarducci and Tjeerdema 1995; Jacobson and Smith 1990; Kissel et al. 1978; Nordone et al. 1996a, 1996b; Rathbun 1998; Smith et al. 1995; Tabak et al. 1981). No data were located on the removal of acrolein from water through reactions with dissolved and suspended organic matter in water. Studies on this route of removal of acrolein from water would be useful for determining the lifetime of acrolein in waters with high organic content. Measured soil-water partition coefficient data are not available. This information would be helpful for describing the absorption and mobility of acrolein in soil. Experimental data pertaining to the persistence of acrolein in soil and groundwater are lacking. Studies on volatilization from soil surfaces, anaerobic

biodegradation in soil and simulated groundwater, and aerobic biodegradation in simulated groundwater would be useful in establishing the likelihood of exposure near hazardous waste disposal sites resulting from volatilization from soil surfaces or from groundwater contamination.

Bioavailability from Environmental Media. No studies were located regarding the bioavailability of acrolein from environmental media. Since acrolein has been detected in ambient air and in food and beverages (ppb levels), it is important to determine if acrolein can be absorbed by humans from environmental samples. However, the chemical structure of acrolein makes it a highly reactive molecule, which presumably is why its effects are, for the most part, restricted to the area of exposure (i.e., respiratory system for inhalation exposure or localized skin damage for dermal exposure). The limited information available regarding absorption parameters of acrolein in experimental animals indicates that acrolein is easily retained in the respiratory airways (Egle 1972; Morris 1996; Morris et al. 2003) and is, therefore, likely to act as an irritant of the eyes and respiratory tract with negligible absorption into the body. Virtually no information is available regarding absorption by the gastrointestinal tract or skin; additional studies would be useful in establishing whether acrolein is absorbed through these sites or is retained.

Food Chain Bioaccumulation. Measured and estimated BCF values for acrolein indicate that this compound would not bioaccumulate significantly in fish (Bysshe 1982; Hansch and Leo 1995; Veith et al. 1980). No information was available on the bioaccumulation of acrolein in organisms at other trophic levels in aquatic environments. Monitoring for the accumulation of acrolein in organisms from several trophic levels would be useful in estimating the levels of acrolein to which humans are exposed through dietary intake.

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

Data are available regarding the detection of acrolein in the environment, most notably in ambient air (Altshuller and McPherson 1963; CARB 1991; Destaillats et al. 2002; EPA 1998c, 1999, 2004a; Highsmith and Zweidinger 1988; IARC 1995; Mohamed et al. 2002; Morello-Frosch et al. 2000; Renzetti and Bryan 1961; Rosenbaum et al. 1999; WHO 1991, 2002), and also in water (Grosjean and Wright 1983; Krill and Sonzogni 1986; Otson 1987; WHO 2002), soil, and sediment (Hauser and Bromberg

1982). Some data are available on acrolein concentrations in air, water, landfill leachate, soil, and sediment samples taken either onsite or offsite from NPL or Superfund sites (HazDat 2006; Sabel and Clark 1984). Additional information on exposure to acrolein in air in urban areas, rural areas, near hazardous waste disposal sites, as well as in water (specifically, drinking water supplied from groundwater down gradient from hazardous waste disposal sites and contaminated surface waters) and soil at waste disposal sites would be useful. Monitoring air and water over a 1-year period would provide some indication of seasonal variations.

Exposure Levels in Humans. Data for residential exposure to acrolein are limited to a probabilistic study that provided a 24-hour time-weighted estimate of acrolein concentrations in air and inhalation intake for Canadian residents (Environment Canada 2000) and to a study on exposure of nonsmokers in the United States to acrolein in ETS (Nazaroff and Singer 2004). The development of a program for monitoring environmental media would provide information for better estimations of acrolein exposure levels in humans. Data are not available for intake of acrolein through the diet. Market basket surveys or total diet studies similar to those conducted by the FDA are needed to provide data on typical levels of exposure via dietary intake given the presence of acrolein in a number of foods (Cantoni et al. 1969; EPA 1980, 1985; Feron et al. 1991; IARC 1985; Umano and Shibamoto 1987; WHO 2002). Monitoring studies of acrolein concentrations in air are available for a few occupations such as shipyard workers, welders, plastic manufacturers, food service employees, and firefighters (Ahrenholz and Egilman 1983; Apol 1982; Ghilarducci and Tjeerdema 1995; Gochfeld 1995; Henriks-Eckerman et al. 1990; IARC 1985; Lees 1995; Materna et al. 1992; Tharr and Singal 1986; Treitman et al. 1980; Vainiotalo and Matveinen 1993; Woskie et al. 1988). Given the high likelihood of occupational exposures to acrolein as a consequence of its emission from combustion sources and the variability in the frequency and amount of exposures to the compound in various occupational settings, additional monitoring data are needed to provide reliable estimates of average daily intake of acrolein in workers.

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

Exposures of Children. Data on the exposure of children to acrolein are very limited (Nazaroff and Singer 2004; WHO 2002). For children living in a residence where one or more individuals smokes some form of tobacco product, long-term exposure to acrolein and other compounds in ETS are expected (Nazaroff and Singer 2004; WHO 1999). Lifetime exposures to acrolein in ETS have been estimated for individuals residing with one or more smokers (Nazaroff and Singer 2004); however, there are no data that specifically address the inhalation intake of acrolein from ETS in individuals below the age of 18.

Information on acrolein concentrations in indoor air is limited for residences in the United States (CARB 1992; Highsmith and Zweidinger 1988; WHO 2002). More data are needed to adequately assess the exposures of children to acrolein generated from indoor combustion sources, especially tobacco products. Determination of the average daily intake of acrolein would be complicated by the variability in the frequency and amount of exposure to cigarette smoke and other acrolein sources. Therefore, exposure studies should be structured to assess the temporal variations in acrolein concentrations over a typical day and should also account for seasonal changes in air exchange within a residence (i.e., winter versus summer). For children who are not exposed to ETS in the home environment, it is expected that the largest exposure to acrolein will be through inhalation of ambient air, especially in urban areas, and through the diet. Therefore, studies that are tailored to assessing exposure of children to acrolein in ambient air would be useful given the tendency for children to spend more time outdoors than many adults. Also, market basket surveys or total diet studies similar to those conducted by the FDA would be useful for providing data on typical levels of exposure via dietary intake for children.

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 acrolein 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 EPA is developing the methods, data, and models of exposure that will provide the scientific basis for EPA to move to a risk-based program that will enhance the National-Scale Air Toxics Assessment (NATA) program. No other pertinent ongoing studies were identified.