# 6. POTENTIAL FOR HUMAN EXPOSURE

## 6.1 OVERVIEW

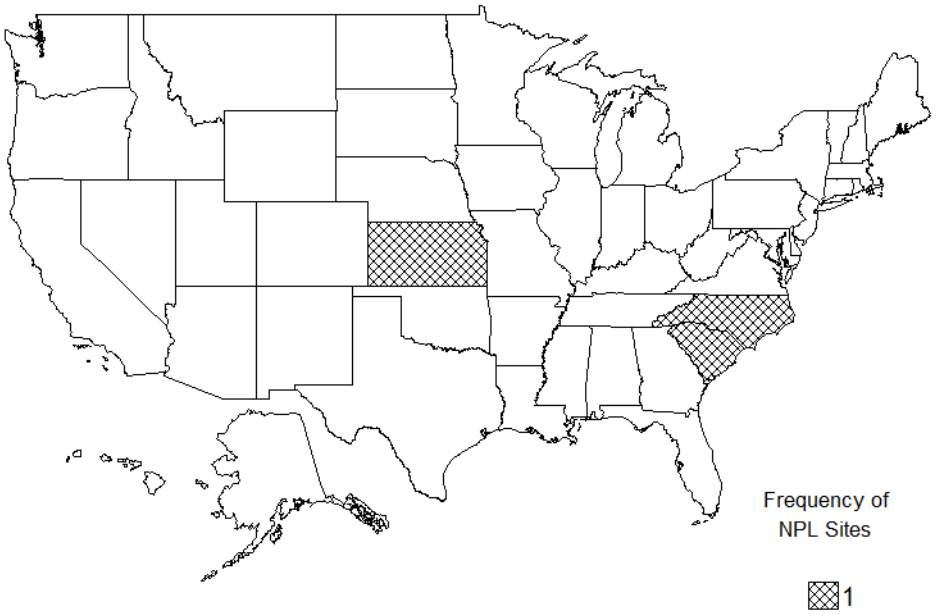
Acrylamide has been identified in at least 3 of the 1,699 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 2007). However, the number of sites evaluated for acrylamide is not known. The frequency of these sites can be seen in Figure 6-1.

Acrylamide is an industrial chemical used mainly in the production of polyacrylamides, which are primarily used as flocculants for clarifying drinking and treating municipal and industrial effluents (Abdelmagid 1982; EPA 2006c; EU 2002; Haberman 2002; O'Neil et al. 2006; WHO 2003). Acrylamide may be released to the environment during production and use of polyacrylamides, which are used as clarifiers in water treatment. Residual monomer released from the polyacrylamide coagulants is the main source of acrylamide contamination of drinking water (Abdelmagid 1982; Cavalli et al. 2004; EPA 2006c; WHO 2003), though it can also be released from plastics and dye industries and from acrylamide-containing grouting agents used in reservoirs and wells (Cavalli et al. 2004; EPA 2006c). Acrylamide can be released to the environment during formulation of cosmetics or other consumer products and in the laboratory while gel chromatography is being performed (Boettcher and Angerer 2005; EU 2002). Due to its low vapor pressure and high water solubility, acrylamide is rarely identified in atmospheric samples (WHO 2003).

Acrylamide is expected to be highly mobile in soil and water (EPA 2006c; HSDB 2009; WHO 2003). It is highly susceptible to biodegradation in both soils and surface water (Abdelmagid 1982; EPA 2006c; Haberman 2002; WHO 2003). It is not typically present in the atmosphere (HSDB 2009; Pratt 2000). Acrylamide is not expected to significantly bioconcentrate (EPA 2006c; Haberman 2002; WHO 2003).

Acrylamide is sometimes present in drinking water due to leaching of monomer during treatment processes (Abdelmagid 1982; Cavalli et al. 2004; EPA 2006c; van Dijk-Looijaard and van Genderen 2000; WHO 2003), as well as release from grouting agents in wells and dams and releases from plastics and dye industries (Cavalli et al. 2004). It is rarely found in soil samples (HSDB 2009). Acrylamide is not a common air pollutant, due to its low vapor pressure and high water solubility. It is rarely identified in atmospheric samples (HSDB 2009; Pratt 2000; WHO 2003).

## Figure 6-1. Frequency of NPL Sites with Acrylamide Contamination




Derived from HazDat 2007

In 2002, acrylamide was first identified in samples of food cooked at high temperatures (Tareke et al. 2002). Concentrations of acrylamide in food vary with the type of food and method of cooking, and typically increase with temperature and length of heating (Sorgel et al. 2002; WHO 2002, 2003). Carbohydrate-rich foods typically contain the highest levels of acrylamide (Muttucumaru et al. 2008; Tareke et al. 2002; WHO 2003), whereas protein-based foods contain smaller amounts (Tareke et al. 2002). As acrylamide content in food appears to be affected by temperature, water content, food thickness, and length of heating, various steps can be taken to minimize the exposure of acrylamide from food sources (Sorgel et al. 2002; WHO 2002, 2003).

Acrylamide is a carcinogen with the potential to cause nervous system damage (Arisetto et al. 2007; EPA 2006c; Lewis 2000). Exposure occurs mainly via ingestion, dermal contact, and inhalation routes (Lewis 2000; Sorgel et al. 2002). Ingestion of foodstuffs containing acrylamide appears to be one of the most common methods of exposure for the general public. Average estimated intake of acrylamide from food sources ranged from 0.8 to 6.0 μg/kg bw/day for short-term exposure and 0.3 to 0.8 μg/kg bw/day for long-term exposure (WHO 2002, 2003). Children may be susceptible to food-borne exposure 2–3 times that of adults on a body weight basis (WHO 2002, 2003).

Ingestion of polyacrylamide-treated drinking water containing residual monomer as well as water in contact with acrylamide-containing products, such as grouting agents, can result in exposure to acrylamide (EPA 2006c; EU 2002; WHO 2003). The presence of acrylamide in tobacco smoke can result in inhalation exposure for both adults and children (EU 2002; Moreno Navarro et al. 2007). Dermal exposure can result from contact with cosmetics and toiletries containing polyacrylamides (EU 2002). Once in the body, acrylamide is widely dispersed by body fluids, and can also cross the placental barrier (WHO 2003), resulting in exposure to unborn children. Breast milk of mothers with diets high in acrylamide-containing foods can contain high amounts of acrylamide (Sorgel et al. 2002). Occupational exposure to acrylamide is primarily due to dermal contact when handling bags and drums of the chemical or preparing polyacrylamide gels, followed by inhalation of dust or aerosols (EU 2002; Lewis 2000).

## 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 2005a). 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 2005a).

### 6.2.1 Air

Estimated releases of 8,509 pounds (~3.9 metric tons) of acrylamide to the atmosphere from 76 domestic manufacturing and processing facilities in 2009, accounted for about 0.2% of the estimated total environmental releases from facilities required to report to the TRI (TRI09 2011). These releases are summarized in Table 6-1.

Acrylamide may be released to the atmosphere during production of polymers, formulation of cosmetics or other consumer products, and in the laboratory while gel chromatography is being performed (Boettcher and Angerer 2005). Release of tobacco smoke, which was shown to contain acrylamide (Schumacher et al. 1977), may also contribute to airborne acrylamide in confined spaces. However, acrylamide is not anticipated to be a common air contaminant due to its low vapor pressure and high water solubility. Acrylamide is not expected to be removed from soils or water by volatilization (WHO 2003). Limited data indicate that atmospheric acrylamide concentrations are very low, when it is identified at all (Pratt 2000; HSDB 2009).

The total national baseline National Toxics Inventory (NTI) emissions for acrylamide during 1900–1998 were 35.4 tons/year. Total urban emissions were 33.5 tons/year while total rural emissions were 1.9 tons/year (EPA 2000). The National Emissions Inventory database, maintained by the EPA, reports total U.S. acrylamide emissions of 12.12 tons per year in 2005 from various sources, including degreasing, waste disposal, pulp and paper, solvents, oil and gas production, industrial surface coatings, and oil and gasoline production among others (EPA 2009b).

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

| | | Reported amounts released in pounds per year[b] | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Total release | | |
| State[c] | RF[d] | Air[e] | Water[f] | UI[g] | Land[h] | Other[i] | On-site[j] | Off-site[k] | On- and off-site |
| AL | 1 | 83 | 0 | 0 | 467 | 0 | 83 | 467 | 550 |
| AR | 1 | 0 | 0 | 0 | 1 | 1 | 0 | 2 | 2 |
| CA | 2 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| CO | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| CT | 2 | 2 | 3 | 0 | 152 | 0 | 5 | 152 | 157 |
| GA | 5 | 5,117 | 0 | 0 | 1,089 | 0 | 5,117 | 1,089 | 6,206 |
| IL | 7 | 91 | 0 | 0 | 0 | 0 | 91 | 0 | 91 |
| KY | 1 | 458 | 0 | 0 | 0 | 0 | 458 | 0 | 458 |
| LA | 5 | 295 | 86 | 778,620 | 2,735 | 0 | 779,636 | 2,100 | 781,736 |
| MD | 2 | 5 | 0 | 0 | 0 | 0 | 5 | 0 | 5 |
| MI | 4 | 183 | 0 | 0 | 0 | 0 | 183 | 0 | 183 |
| NC | 6 | 1,769 | 0 | 0 | 0 | 0 | 1,769 | 0 | 1,769 |
| NE | 1 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| NJ | 2 | 8 | 0 | 0 | 0 | 0 | 8 | 0 | 8 |
| OH | 5 | 1 | 0 | 890,000 | 5 | 12 | 890,001 | 17 | 890,018 |
| PA | 5 | 89 | 5 | 0 | 0 | 987 | 94 | 987 | 1,081 |
| SC | 8 | 281 | 250 | 0 | 0 | 0 | 531 | 0 | 532 |
| TN | 4 | 2 | 0 | 0 | 0 | 200 | 2 | 200 | 202 |
| TX | 8 | 24 | 0 | 2,890,223 | 253 | 250 | 2,890,250 | 500 | 2,890,750 |
| VA | 1 | 65 | 0 | 0 | 180 | 0 | 65 | 180 | 245 |
| WA | 1 | 23 | 0 | 0 | 0 | 0 | 23 | 0 | 23 |
| WI | 3 | 11 | 0 | 0 | 28 | 0 | 11 | 28 | 39 |
| Total | 76 | 8,509 | 344 | 4,558,843 | 4,910 | 1,450 | 4,568,334 | 5,722 | 4,574,057 |

[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 onsite 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: TRI09 2011 (Data are from 2009)

### 6.2.2 Water

Estimated releases of 344 pounds (~0.16 metric tons) of acrylamide to surface water from 76 domestic manufacturing and processing facilities in 2009, accounted for about 0.008% of the estimated total environmental releases from facilities required to report to the TRI (TRI09 2011). These releases are summarized in Table 6-1.

Acrylamide in drinking water is typically a result of the release of residual monomer from polyacrylamide coagulants that are used as clarifiers in the treatment of raw water (Abdelmagid 1982; Cavalli et al. 2004; EPA 2006c; WHO 2003). The clarifiers served to coagulate and trap suspended solids such that they may be removed more easily from the water. Acrylamide that does not coagulate is released into the environment as a drinking water contaminant (Abdelmagid 1982; EPA 2006c). Use of polyacrylamides as grouting agents in reservoirs and wells can result in release to drinking water supplies. They can also be released in water from plastics and dye industries (Cavalli et al. 2004; EPA 2006c). When released to land, acrylamide does not bind to soil and will thus move rapidly through the soil column (EPA 2006c), which can result in increased risk of surface or groundwater contamination.

Total release of acrylamide to water between 1987 and 1993 was 36,287 pounds. Major industries found to release acrylamide to water include plastics and resins (19,002 pounds), pulp mills (8,000 pounds), industrial organics (3,107 pounds), and industrial inorganics (2,510 pounds) (EPA 2006c). According to the EPA's Toxic Chemical Release Inventory, release of acrylamide to land and water from 1987 to 1993 was over 40,000 pounds. Acrylamide releases were typically from plastics industries, with the largest releases occurring in Michigan (EPA 2006c).

As required by the 1974 Safe Drinking Water Act, EPA developed a maximum contaminant level (MCL) for acrylamide, which specifies the concentration at which it is not expected to cause health problems. The MCL for acrylamide is zero (EPA 2006c). EPA requires water suppliers to control the amount of acrylamide added to water during the treatment process. This can be accomplished by limiting the amount of acrylamide in the polyacrylamide flocculants or by limiting the dose of flocculants (WHO 2003). Uncoagulated acrylamide in drinking water must be <0.5 μg/L (ppb) (Cavalli et al. 2004; EPA 2006c). This concentration corresponds to a maximum authorized dose of polymer of 1 mg/L (for a monomer content of 0.05%), as this corresponds to 0.5 μg/L of monomer in the water (Cavalli et al. 2004; WHO 2003). In European Union countries, the maximum allowable concentration in drinking water is 0.1 μg/L (Cavalli et al. 2004).

If acrylamide is found to be present in raw water, the concentration can be decreased through ozonation or by treating the water with potassium permanganate. Acrylamide is not removed by conventional water treatment processes (WHO 2003).

### 6.2.3 Soil

Estimated releases of 4,910 pounds (~2.2 metric tons) of acrylamide to soils from 76 domestic manufacturing and processing facilities in 2009, accounted for about 0.1% of the estimated total environmental releases from facilities required to report to the TRI (TRI09 2011). An additional 4,558,843 pounds (~2,783 metric tons), constituting about 99.6% of the total environmental emissions, were released via underground injection (TRI09 2011). These releases are summarized in Table 6-1.

Polyacrylamides releases to land can occur from plastics and dye industries (Cavalli et al. 2004; EPA 2006c). When released to land, acrylamide does not bind to soil and will thus move rapidly through the soil column (EPA 2006c; WHO 2003). Total release of acrylamide to land between 1987 and 1993 was 5,818 pounds. Major industries found to release acrylamide to land include plastics and resins (2,177 pounds), industrial organics (2,200 pounds), and industrial inorganics (500 pounds) (EPA 2006c). According to the EPA's Toxic Chemical Release Inventory, release of acrylamide to land and water from 1987 to 1993 was over 40,000 pounds. Acrylamide releases were typically from plastics industries, with the largest releases occurring in Michigan (EPA 2006c).

## 6.3 ENVIRONMENTAL FATE

### 6.3.1 Transport and Partitioning

Acrylamide is expected to be highly mobile in soil and water. When released to land, acrylamide does not bind to soil and will thus move rapidly through the soil column and into groundwater, where it is also expected to have high mobility (EPA 2006c; HSDB 2009; WHO 2003). Acrylamide has a higher mobility in sandy soils than in clay soils, which can result in increased risk of surface or groundwater contamination (Abdelmagid 1982; EPA 2006c; WHO 2003). Acrylamide is not expected to be removed from soils or water by volatilization (WHO 2003).

Acrylamide is not expected to significantly bioconcentrate in aquatic organisms due to its high water solubility and its ability to be degraded by microorganisms (EPA 2006c; Haberman 2002; WHO 2003).

Acrylamide was determined to have a bioconcentration factor (BCF) of around 1 in both the carcass and viscera (0.86–1.44 and 1.12–1.65, respectively) of rainbow trout exposed to acrylamide concentrations of 0.338 and 0.710 mg/L. This indicates that acrylamide did not bioaccumulate significantly in the trout (Petersen et al. 1985).

### 6.3.2 Transformation and Degradation

Acrylamide is susceptible to biodegradation in both soils and surface water (Abdelmagid 1982; EPA 2006c; Haberman 2002; HSDB 2009; WHO 2003). As it does not bind to soil, acrylamide moves rapidly through the soil column where it is quickly degraded (EPA 2006c; WHO 2003).

In sandy soils, acrylamide has a higher mobility and lower rate of degradation than in clay soils (Abdelmagid 1982; EPA 2006c; WHO 2003). Acrylamide has been shown to biodegrade in effluent from a sludge dewatering process (WHO 2003).

Enzyme-catalyzed hydrolysis is a dominant mechanism for removal of acrylamide from soils (WHO 2003). Abdelmagid (1982) showed that acrylamide is readily hydrolyzed in soils under both aerobic and anaerobic conditions, resulting in the release of $NH_4^+$. The rate of decomposition in soil is influenced by temperature and incubation time. Soil type did not appear to be a factor, as acrylamide decomposed in both sandy and heavy-textured soils (Abdelmagid 1982).

Acrylamide was shown to degrade by an average of 41.5% by BOD in 14 days at 25 °C and 100 mg/L test substance, with 30 mg/L activated sludge (CERI 1999).

#### 6.3.2.1 Air

Limited data indicate that acrylamide concentrations in the atmosphere are very low (HSDB 2009; Pratt 2000). In the atmosphere, acrylamide is susceptible to degradation via photochemically generated hydroxyl radicals and ozone. The rate constant for acrylamide's reaction with hydroxyl radicals has been estimated as $1.1 \times 10^{-11}$ $cm^3$/molecule-second, and its reaction with ozone is estimated as $1.7 \times 10^{-18}$ $cm^3$/molecule-second using a structure estimation method (Meylan and Howard 1993). Using an average atmospheric hydroxyl radical concentration of $1.5 \times 10^6$ molecules/$cm^3$ and ozone concentration of $7 \times 10^{11}$ molecules/$cm^3$, estimated half-lives of approximately 12 hours (hydroxyl radical reaction) and 6.5 days (ozone reaction) can be calculated.

#### 6.3.2.2 Water

Acrylamide is expected to be quickly degraded in water by biological processes (Abdelmagid 1982; EPA 2006c; Haberman 2002; WHO 2003). At an initial concentration of 10 ppm, acrylamide was completely degraded in about 12 days using water obtained from the Hackensack River (Cherry et al. 1956). Acrylamide, at an initial concentration of 8 µg/L, was rapidly degraded following a lag period of approximately 9 days in well-aerated, sunlit river water obtained from the Thames River, England (Croll et al. 1974). Subsequent re-seeding of the water with acrylamide resulted in rapid degradation with little or no lag period.

#### 6.3.2.3 Sediment and Soil

When released to land, acrylamide does not bind to soil and will thus move rapidly through the soil column, where it is expected to be quickly degraded (EPA 2006c; Haberman 2002; WHO 2003). Acrylamide has a higher mobility and lower rate of degradation in sandy soils than in clay soils (Abdelmagid 1982; EPA 2006c; WHO 2003).

Enzyme-catalyzed hydrolysis is a dominant mechanism for removal of acrylamide from soils (WHO 2003). Abdelmagid (1982) showed that acrylamide is readily hydrolyzed in soils, producing $NH_4^+$. The rate of decomposition in soil is influenced by temperature and incubation time. Soil type did not appear to be a factor, as acrylamide decomposed in both sandy and heavy-textured soils. Acrylamide, therefore, does not appear to accumulate in soils (Abdelmagid 1982).

### 6.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

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

### 6.4.1 Air

Acrylamide is not anticipated to be a common air contaminant due to its low vapor pressure and high water solubility. Limited data indicate that acrylamide concentrations in the atmosphere are very low (HSDB 2009; Pratt 2000). Air samples were collected at 25 sites throughout Minnesota over various periods of time (up to 8 years) from 1991 to 1998. Acrylamide was not identified in any of the samples (Pratt 2000). As of 1978, acrylamide concentrations in the air near six U.S. acrylamide/polyacrylamide producers or users averaged <0.2 μg/m$^3$ in vapor or particulate form. Concentrations ranged from <0.1 to 1.1 μg/m$^3$ (EPA 1978).

The total national baseline NTI emissions for acrylamide during 1900–1998 were 35.4 tons/year. Total urban emissions were 33.5 tons/year, while total rural emissions were 1.9 tons per year (EPA 2000).

### 6.4.2 Water

Acrylamide in drinking water is typically a result of leaching during treatment processes (van Dijk-Looijaard and van Genderen 2000). Acrylamide results from the release of residual monomer from polyacrylamide coagulants that are used as clarifiers in the treatment of raw water (Abdelmagid 1982; Cavalli et al. 2004; EPA 2006c; WHO 2003). Polyacrylamides can enter water supplies due to their use as grouting agents in reservoirs and wells in addition to being released in water from plastics and dye industries (Cavalli et al. 2004; EPA 2006c).

Acrylamide concentrations of <5 μg/L were found in river and tap water. In the sampling area, polyacrylamides were known to be used in treating potable water (WHO 2003). Public drinking water supply wells in West Virginia contained acrylamide concentrations of 0.024–0.041 μg/L (WHO 2003).

One water sample, taken from downstream of the effluent of a producer of polyacrylamide, contained acrylamide at 1,500 μg/L, while samples from other industrial sites (acrylamide and polyacrylamide production and use locations) contained <0.8 μg/L acrylamide (EPA 1978).

Various effluents in the United Kingdom have been found to contain acrylamide, including effluent from a clay pit (16.0 μg/L), a tailings lagoon (39–42 μg/L), coal washing lagoon (1.8 μg/L), colliery/cooking plant, (0.74 μg/L), treated paper mill (0.47–14.4 μg/L), and paper mill process water (45.4 μg/L) (Croll et al. 1974; IPCS 1985). In Devon, England, 17.4 ppb of acrylamide was detected in sewage effluent (Brown and Rhead 1979).

### 6.4.3 Sediment and Soil

Soil samples obtained near six U.S. acrylamide and/or polyacrylamide producers contained <0.02 μg/g of acrylamide. Concentrations ranged from <0.02 to <0.08 μg/g (EPA 1978).

### 6.4.4 Other Environmental Media

Acrylamide was detected in waste materials and containers at 3 of the 1,699 hazardous waste sites that have been proposed for inclusion on the EPA NPL (HazDat 2007). Acrylamide was also identified in tobacco smoke samples, obtained from nonfiltered cigarettes smoked under typically conditions. The presence of acrylamide was identified in the smoke condensate by infrared (IR), mass spectrometry (MS), and nuclear magnetic resonance (NMR) (Schumacher et al. 1977).

In 2002, acrylamide was first shown to be produced when foods are cooked at high temperatures (Tareke et al. 2002). Concentrations of acrylamide in food vary with the type of food and method of processing and cooking. Acrylamide concentrations in food typically increase with temperature and length of heating (Sorgel et al. 2002; WHO 2002, 2003). Starchy foods, such as potato-based products, typically contain the highest levels of acrylamide (Muttucumaru et al. 2008; Tareke et al. 2002; WHO 2003), whereas protein-based foods contain smaller amounts (Tareke et al. 2002).

Tareke et al. (2002) analyzed acrylamide content of various heated foods. Acrylamide concentrations were determined by gas chromatography (GC)-MS and liquid chromatography (LC)-MS/MS. In heated protein-rich foods, acrylamide concentrations of 5–50 μg/kg were found. Carbohydrate-rich foods, such as potato, beetroot, potato products, and crispbread, contained much higher concentrations, ranging from 150 to 4,000 μg/kg. The median acrylamide concentration in fried foods, including beef, chicken, soymeal, grated potatoes, boiled mashed potatoes, and grated beetroot were 17, 28, 16, 447, 172, and 850 μg/kg, respectively. In microwave-heated grated potatoes, the median acrylamide concentration was found to be 551 μg/kg, while microwave-heated cod was less than the detection limit (<5 μg/kg). In restaurant-prepared or purchased foods, the median acrylamide concentrations for hamburger, French fries, potato crisps, and three types of crispbread were 18, 424, 1,739, and 208, respectively. Unheated controls and boiled foods contained very little acrylamide, with all results being less than the detection limits for the methods (<5 μg/kg by GC-MS and <10 μg/kg by LC-MS/MS) (Tareke et al. 2002).

Acrylamide concentrations were analyzed in food samples in Norway, Sweden, Switzerland, the United Kingdom, and the United States in various studies (Table 6-2). Almost all items analyzed contained some level of acrylamide. Of the food tested, chips and crisps contained the highest average concentrations, ranging from not detectable to 3.5 mg/kg (WHO 2002).

Acrylamide concentrations were determined for various foods prepared using home cooking methods, including the use of a household oven and microwave oven. The data are presented in Tables 6-3 and 6-4. The authors concluded that longer cooking times and higher temperatures appear to cause increased acrylamide concentrations in food (Sorgel et al. 2002).

The acrylamide content of various carbohydrate-rich Brazilian foods was determined by LC-MS/MS. Samples were obtained from grocery stores, restaurants, and fast food restaurants in Campinas, Sao Paulo, Brazil between September 2004 and April 2006. The foods sampled included French fries, potato chips, bread, crispbread, crackers, breakfast cereals, coffee, beer, and other high carbohydrate foods typically processed at high temperatures. Of 111 samples from 19 product categories, acrylamide concentrations ranged from <20 to 2,528 mg/kg. Considerable differences in concentration existed between individual foods within the same product class. Potato chips, processed at high temperatures, and instant coffee had the highest concentrations of acrylamide, while cassava- and maize-based foods, bread, and beer were found to have the lowest levels. The detection limit and limit of quantification were 10 and 20 mg/kg, respectively (Arisseto et al. 2007).

An ongoing U.S. Food and Drug Administration (FDA) survey collects acrylamide data on U.S. food products under the Total Diet Study (TDS). Data on approximately 280 core foods (or TDS foods) were collected to determine the nutrient and contaminant levels in the foods from each of the geographic regions in the United States (West, North Central, South, and Northeast). Food samples are collected from grocery stores and fast food restaurants from three cities in each region, prepared for consumption, and then analyzed (FDA 2009). The summary results for the 2003–2006 TDS data on acrylamide are presented in Table 6-5. Foods within certain food groups (i.e., grains/starches/baked goods) have been found to have very high levels of acrylamide. It should be noted that the high levels within the vegetable group were primarily due to potato chips and French fries.

## Table 6-2. Acrylamide Levels in Different Food and Food Product Groups from Norway, Sweden, Switzerland, the United Kingdom, and the United States

| Food/product group | Acrylamide levels (µg/kg)[a] | | | |
|---|---|---|---|---|
| | Mean[b] | Median[2] | Minimum-Maximum | Number of Samples |
| Crisps, potato/sweet potato[c] | 1,312 | 1,343 | 170–2,287 | 38 |
| Chips, potato[d] | 537 | 330 | <50–3,500 | 39 |
| Batter based products | 36 | 36 | <30–42 | 2 |
| Bakery products | 112 | <50 | <50–450 | 19 |
| Biscuits, crackers, toast, bread crisps | 423 | 142 | <30–3,200 | 58 |
| Breakfast cereals | 298 | 150 | <30–1346 | 29 |
| Crisps, corn | 218 | 167 | 34–416 | 7 |
| Bread, soft | 50 | 30 | <30–162 | 41 |
| Fish and seafood products, crumbled, battered | 35 | 35 | 30–39 | 4 |
| Poultry or game, crumbed, battered | 52 | 52 | 39–64 | 2 |
| Instant malt drinks | 50 | 50 | <50–70 | 3 |
| Chocolate powder | 75 | 75 | <50–100 | 2 |
| Coffee powder | 200 | 200 | 170–230 | 3 |
| Beer | <30 | <30 | <30 | 1 |

[a]The limits of detection and quantification varied among laboratories; values reported as less than a value are below the limit reported by the laboratory.
[b]Mean and median values were calculated where individual data were available; sample sizes were extremely small, particularly for some food categories; where the mean and median are different, it reflects the skewed distribution of the underlying data that were collected in different countries and may represent different food items within the larger category.
[c]Products that are thinly sliced and fried (such as potato chips).
[d]Products that are more thickly sliced (such as French fries).

Source: WHO 2002

## Table 6-3. Acrylamide in Potato Products

| Sample | Size (mm) | Cooking temperature (°C) | Cooking time (minutes) | Acrylamide concentration (µg/kg) |
|---|---|---|---|---|
| Potato chips | 3 | 180 | 2 | 2,557.9 |
| | 3 | 140 | 2 | 36.3 |
| | 3 | 180 | 4 | 7,678.3 |
| | 3 | 140 | 4 | 53.3 |
| | 1 | 180 | 1 | 121.1 |
| | 1 | 140 | 2 | 20.0 |
| | 1 | 180 | 3 | 9,670.2 |
| | 1 | 140 | 4 | 35.3 |
| French fries | 5 | 180 | 2 | 1,716.9 |
| | 5 | 160 | 2 | 53.8 |
| | 5 | 140 | 2 | 11 |
| | 5 | 180 | 4 | 2,687.0 |
| | 5 | 160 | 4 | 1,049.5 |
| | 5 | 140 | 4 | 77.0 |
| | 10 | 180 | 2 | 674.2 |
| | 10 | 160 | 2 | <8 |
| | 10 | 140 | 2 | <8 |
| | 10 | 180 | 4 | 1,084.3 |
| | 10 | 160 | 4 | 87.3 |
| | 10 | 140 | 4 | 10.0 |
| | 13 | 180 | 4 | 476.2 |
| | 13 | 140 | 2 | <8 |
| | 13 | 180 | 6 | 880.0 |
| | 13 | 140 | 4 | <8 |

Source: Sorgel et al. 2002

## Table 6-4. Acrylamide Content of Food

| Sample | Description | Cooking method | Cooking time (minutes) | Acrylamide concentration (µg/kg) |
|---|---|---|---|---|
| Walnuts | | Roasted | | <8 |
| Pine seeds | | Roasted | | 16.4 |
| Candied chestnuts | | Baked | | 8.3 |
| Crème carmel | | Boiled | | <8 |
| Whiskey | | | | <8 |
| Liver of beef | | Baked | | <8 |
| Breaded fish | | Baked | | <8 |
| Goose skin | | Oven | | <8 |
| Goose meat | | Oven | | <8 |
| Gingerbread | Lightly baked | 200 °C | | 12.1 |
| Gingerbread | Dark baked | 200 °C | | 113.0 |
| Gingerbread | | | | 180.7 |
| Gingerbread balls | | | | 639.5 |
| Spiced cookies | | | | 143.6 |
| Popcorn | | Microwave, 800 W | 1 | 29.7 |
| Popcorn | | Microwave, 600 W | 2 | 50.0 |
| Popcorn | | Microwave, 800 W | 2 | 132.8 |
| Popcorn | | Microwave, 600 W | 3 | 250.3 |
| Popcorn | | Microwave, 600 W | 4 | 307.1 |
| Croquettes | | 180 °C | 2 | 354.7 |
| Croquettes | | 180 °C | 4 | 453.4 |
| Hash browns | | 180 °C | 2 | 446.1 |
| Hash browns | | 180 °C | 4 | 751.7 |
| Fried potatoes | | | | 543.1 |
| Croutons | | Baked | | 27.9 |
| Bread crust | | | | 370.6 |
| Toast | | Toasted | | 54.1 |

Source: Sorgel et al. 2002

## Table 6-5. Acrylamide Levels in Food Products Sampled for the 2003–2006 Total Diet Studies (TDS) Summary

| TDS food category | Acrylamide range (ppb) for 2003 | Acrylamide range (ppb) for 2004 | Acrylamide range (ppb) for 2005 | Acrylamide range (ppb) for 2006 |
|---|---|---|---|---|
| Dairy | ND–16 | ND | ND–16 | ND–18 |
| Eggs | ND | No data | ND | No data |
| Baby food | ND–267 | ND–407 | ND–442 | ND–381 |
| Meat/poultry/fish | ND–26 | ND–27 | ND–30 | ND–19 |
| Legumes | ND–93 | ND–93 | ND–84 | ND–54 |
| Grains/starches/ baked goods | ND–647 | ND–946 | ND–616 | ND–470 |
| Fruits | ND–202 | 109–355[a] | ND–250 | 326[a] |
| Vegetables | ND–536[b] | ND-829[b] | ND–529 | ND–393 |
| Mixtures (e.g., casseroles, sandwiches, soups, pizza) | ND–187 | ND–210 | ND–93 | ND–55 |
| Candy/sweets/ Sugars/syrups | ND–29 | ND–42 | 13–50 | 17–32 |
| Fats/oils | ND | No data | No data | No data |
| Beverages | ND–12 | ND–21 | ND–28 | ND |

[a]Only bottle prune juice was included in this category for 2004 and 2006.
[b]The highest levels in the vegetable category are for potato chips and French fries; the majority of the vegetables had low or non-detectable levels.

ND = Not detected

Source: U.S. Food and Drug Administration 2006

Temperature, water content, food thickness, and length of heating appear to be important factors affecting the amount of acrylamide that is formed while cooking foods (Sorgel et al. 2002; WHO 2002, 2003). Reversing these factors could help minimize acrylamide formation (e.g., heating to <180 °C, making products very thick, etc.) (Sorgel et al. 2002). In many countries, food manufacturers have been requested to take steps to decrease the formation of acrylamide in their products, which has become an important area of research (Amrein et al. 2007).

With potato products, controlling reducing sugars, processing temperature, and moisture content can help limit formation of acrylamide (Amrein et al. 2007). It is thought that the amount of asparagine is also an important factor in acrylamide formation in potatoes (Muttucumaru et al. 2008). With bakery products, the amount of free asparagines as well as the type of baking agent can affect acrylamide concentration. Replacing the baking agent $NH_4HCO_3$, for example, with $NaHCO_3$ in sweet bakery products can help reduce acrylamide content. High concentrations of acrylamide can form during heating of olives and dried fruit (Amrein et al. 2007). In almonds, roasting time and temperature, water content, and asparagine levels (which can range from 500 to 2,760 mg/kg in almonds) are thought to affect the amount of acrylamide formed during roasting (Amrein et al. 2007; Zhang et al. 2011). Additional research is needed to determine the acrylamide reduction strategies for each type of food.

## 6.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

Acrylamide is a probable or confirmed carcinogen with the potential to cause nervous system damage, weakness, and incoordination of the legs following short-term exposure to high levels. Long-term effects of acrylamide include nervous system damage, paralysis, and cancer (Arisetto et al. 2007; EPA 2006c; Lewis 2000). Exposure can occur via ingestion, dermal contact, inhalation, and intraperitoneal routes (Lewis 2000; Sorgel et al. 2002). Once in the body, acrylamide is widely dispersed by body fluids. It can also cross the placental barrier (WHO 2003).

Exposure to high levels of acrylamide can result from ingestion of various foodstuffs. Acrylamide is produced when certain foods are cooked at high temperatures (Tareke et al. 2002). Concentrations of acrylamide in food vary with the type of food and method of processing and cooking, and typically increase with temperature and length of heating (Sorgel et al. 2002; WHO 2002, 2003). Foodstuffs, particularly those rich in carbohydrates, may develop high levels of acrylamide when cooked at high temperatures (Moreno Navarro et al. 2007). As shown in Table 6-3, acrylamide concentrations in potato products such as potato chips and French fries are related to size and cooking temperature and duration.

Potato chips made from 1 mm thick potato slices cooked at 180 °C for 3 minutes contain an estimated 9,670 µg acrylamide/kg potato or the equivalent of 541 µg acrylamide in a 2 ounce bag of potato chips. For an adult (70 kg) or child (15 kg), acrylamide doses from consumption of such a 2-ounce bag of potato chips are estimated at 7.7 and 36 µg/kg, respectively, and are in the range of the acute-duration oral MRL of 10 mg/kg/day for acrylamide. For potato chips made from 1 mm thick potato slices, but cooked at a lower temperature (140 °C) for 4 minutes, estimated adult and child doses are 0.028 and 0.13 µg/kg, respectively, and are somewhat lower than the ATSDR intermediate- and chronic-duration oral MRL of 1 µg/kg for acrylamide. Potato products, including chips and other potatoes cooked at high temperatures, may comprise a large percentage of the total acrylamide intake from food. However, foods with lower acrylamide content but eaten at a higher frequency, such as bread, may contribute significantly to the total exposure from foods (WHO 2002).

Food was found to contribute significantly to the overall exposure of the general population to acrylamide. Short-term dietary intake of acrylamide was estimated using Monte Carlo techniques based on data from The Netherlands, United States, and Sweden. The short-term exposure estimates ranged from 0.8 to 6.0 µg/kg-bw/day for the average consumer to the 98$^{th}$ percentile consumer (WHO 2002). Average estimated long-term intake of acrylamide from food sources was determined based on data from Australia, Norway, The Netherlands, Sweden, and the United States, as well as data from the International Agency for Research on Cancer (IARC). Long-term intake estimates ranged from 0.3 to 0.8 µg/kg-bw/day (WHO 2002, 2003). Tareke et al. (2002) concluded that the acrylamide levels found in heated foods could result in a daily acrylamide intake of a few tens of micrograms.

Due to the presence of acrylamide in tobacco smoke (EU 2002; Schumacher et al. 1977), the general population can be exposed to acrylamide via inhalation (Moreno Navarro et al. 2007). This may include second-hand smoke, and is thought to be a significant source of exposure for the general population (WHO 2002). Due to its low vapor pressure and high water solubility, acrylamide is not anticipated to be a common air contaminant (WHO 2003), and thus, inhalation exposure is likely limited to tobacco smoke exposure.

Nonfood exposure to acrylamide is thought to be low for nonsmokers. Exposure can result from ingestion of polyacrylamide-treated drinking water containing residual monomer (EPA 2006c; EU 2002; WHO 2003), and from water in contact with acrylamide-containing products (e.g., grouting agents in dams) (Sorgel et al. 2002, van Dijk-Looijaard and van Genderen 2000). The MCL for acrylamide in drinking water, which specifies the concentration at which it is not expected to cause health problems, is

zero (EPA 2006c). Dermal exposure can result from contact with cosmetics and toiletries containing polyacrylamides (EU 2002).

Acrylamide and its metabolite, glycidamide, can react with biomolecules such as hemoglobin. Concentrations of adducts in the blood relevant to dietary exposure can be determined, and thus, the adducts can be used as biomarkers to determine a time-averaged estimate of acrylamide exposure. Urinary biomarker use may also be possible (WHO 2002). The Fourth National Report on Human Exposures to Environmental Chemicals (CDC 2009) reported the levels of acrylamide and glycidamide hemoglobin adducts from the National Health and Nutrition Examination Survey (NHANES) 2003–2004. The acrylamide and glycidamide hemoglobin adduct levels for a variety of age groups and ethnicities are presented in Tables 6-6 and 6-7, respectively. The geometric mean acrylamide and glycidamide hemoglobin adduct concentrations were 61.2 and 59.3 pmol/g hemoglobin, respectively.

Occupational exposure occurs mainly through dermal contact of acrylamide from solution, followed by inhalation of dry monomer or aerosols of solution during manufacture of acrylamide and polyacrylamide as well as during acrylamide grouting or preparation of polyacrylamide gels in the laboratory. Ingestion is a minimal concern in the workplace (EU 2002; Lewis 2000). A unique danger of acrylamide is its ability to be absorbed through unbroken skin (Lewis 2000). In the workplace, handling of dry acrylamide, such as emptying bags and drums, results in an inhalation and dermal hazard from dust (Haberman 2002).

Smoking and alcohol consumption, as well as race/ethnicity, may impact acrylamide toxicity. Therefore, results from studies of acrylamide toxicity performed in different geographical areas may indicate variation in levels of concern for acrylamide toxicity.

## 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

## Table 6-6. Geometric Mean and Selected Percentile of Acrylamide Hemoglobin Adduct Concentrations (pmol/g hemoglobin) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES)[a]

| | Survey years | Geometric mean (95% CI) | | Selected percentiles (95% CI) | | | | | | | | Sample size |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | 50th | | 75th | | 90th | | 95th | | |
| **Total** | 03–04 | **61.2** | (58.1–64.4) | **54.8** | (52.8–57.7) | **79.1** | (73.5–85.6) | **141** | (124–155) | **192** | (168–217) | 7,101 |
| **Age group** | | | | | | | | | | | | |
| 3–5 years | 03–04 | **59.4** | (53.6–65.7) | **58.6** | (51.7–64.9) | **75.7** | (63.4–83.6) | **90.6** | (81.9–105) | **108** | (86.2–118) | 350 |
| 6–11 years | 03–04 | **58.6** | (56.1–61.2) | **57.3** | (55.2–59.7) | **71.0** | (67.4–76.3) | **86.8** | (81.2–91.4) | **98.8** | (91.0–104) | 769 |
| 12–19 years | 03–04 | **57.4** | (54.4–60.5) | **54.5** | (52.1–57.4) | **70.7** | (65.6–75.7) | **100** | (89.2–114) | **132** | (115–151) | 1,889 |
| 20–59 years | 03–04 | **66.2** | (62.2–70.6) | **57.9** | (54.6–61.1) | **96.1** | (83.6–108) | **163** | (147–191) | **223** | (194–243) | 2,570 |
| ≥60 years | 03–04 | **50.1** | (47.9–52.3) | **46.5** | (44.0–49.2) | **61.0** | (57.6–66.0) | **96.1** | (88.0–108) | **141** | (120–152) | 1,523 |
| **Gender** | | | | | | | | | | | | |
| Males | 03–04 | **63.9** | (60.2–67.9) | **57.0** | (53.7–60.1) | **85.5** | (79.2–93.7) | **152** | (139–175) | **220** | (189–237) | 3,509 |
| Females | 03–04 | **58.7** | (55.9–61.5) | **53.4** | (51.8–55.9) | **73.9** | (69.5–80.6) | **126** | (111–142) | **164** | (147–191) | 3,592 |
| **Race/ethnicity** | | | | | | | | | | | | |
| Mexican Americans | 03–04 | **61.7** | (58.7–64.9) | **57.4** | (54.4–60.4) | **73.0** | (69.2–77.3) | **101** | (95.0–115) | **149** | (125–179) | 1,792 |
| Non-Hispanic blacks | 03–04 | **63.8** | (57.3–71.1) | **57.1** | (52.1–64.1) | **86.5** | (74.6–104) | **156** | (120–203) | **218** | (172–271) | 1,874 |
| Non-Hispanic whites | 03–04 | **62.4** | (59.0–66.0) | **55.3** | (53.0–58.6) | **82.2** | (75.4–89.1) | **146** | (129–163) | **197** | (172–223) | 2,994 |

[a]Limit of detection is 3.0 pmol/g hemoglobin.

CI = confidence interval

Source: CDC 2009

## Table 6-7. Geometric Mean and Selected Percentile of Glycidamide Hemoglobin Adduct Concentrations (pmol/g hemoglobin) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES)[a]

| | Survey years | Geometric mean (95% CI) | Selected percentiles (95% CI) | | | | Sample size |
|---|---|---|---|---|---|---|---|
| | | | 50th | 75th | 90th | 95th | |
| **Total** | 03–04 | **59.3** (56.7–62.1) | **59.9** (57.6–62.5) | **85.9** (81.6–90.5) | **130** (120–141) | **167** (153–181) | 7,278 |
| **Age group** | | | | | | | |
| 3–5 years | 03–04 | **71.6** (66.9–76.7) | **71.1** (66.9–78.9) | **94.7** (87.3–101) | **118** (103–126) | **126** (119–135) | 411 |
| 6–11 years | 03–04 | **74.1** (70.3–78.2) | **75.0** (70.9–77.9) | **95.6** (90.4–103) | **121** (112–134) | **141** (126–157) | 784 |
| 12–19 years | 03–04 | **55.4** (51.1–60.1) | **59.2** (56.1–62.1) | **79.2** (72.7–86.7) | **113** (94.9–138) | **146** (123–169) | 1,931 |
| 20–59 years | 03–04 | **62.5** (59.4–65.8) | **60.9** (58.7–64.4) | **90.7** (84.4–98.2) | **143** (130–159) | **187** (169–204) | 2,623 |
| ≥60 years | 03–04 | **45.5** (42.8–48.3) | **46.8** (44.8–49.3) | **65.2** (63.5–66.9) | **96.4** (90.0–103) | **129** (111–141) | 1,529 |
| **Gender** | | | | | | | |
| Males | 03–04 | **59.5** (56.9–62.3) | **59.4** (56.8–61.8) | **87.1** (82.5–92.3) | **136** (123–148) | **174** (157–197) | 3,604 |
| Females | 03–04 | **59.1** (56.0–62.5) | **60.4** (57.5–64.0) | **85.0** (80.2–90.0) | **125** (116–135) | **159** (143–175) | 3,674 |
| **Race/ethnicity** | | | | | | | |
| Mexican Americans | 03–04 | **64.7** (61.2–68.4) | **65.4** (61.1–70.1) | **87.4** (81.5–94.4) | **118** (110–129) | **152** (135–170) | 1,841 |
| Non-Hispanic blacks | 03–04 | **53.8** (51.1–56.7) | **56.0** (52.4–59.7) | **83.0** (75.2–91.5) | **121** (108–140) | **159** (129–204) | 1,954 |
| Non-Hispanic whites | 03–04 | **61.1** (57.6–64.9) | **60.7** (57.9–64.2) | **87.5** (83.0–93.5) | **136** (124–149) | **172** (157–194) | 3,044 |

[a]Limit of detection is 4.0 pmol/g hemoglobin.

CI = confidence interval

Source: CDC 2009

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).

Acrylamide exposure to children can occur via ingestion and inhalation. Food was found to contribute significantly to the overall exposure of the general population to acrylamide. The short-term exposure estimates ranged from 0.8 to 6.0 µg/kg bw/day, while the estimated long-term intake of acrylamide from food sources ranged from 0.3 to 0.8 µg/kg bw/day for adults. Exposure in children is expected to be 2–3 times that of adults on a body weight basis (WHO 2002, 2003). In a study of 110 children between the ages of 5 and 6, the median urinary levels were found to be 36.0 µg AAMA/L and 13.4 µg GAMA/L (where AAMA are mercapturic acids of acrylamide and GAMA are mercapturic acids of glycidamide). The median acrylamide exposure was determined to be 0.54 µg/kg bw/day, mainly resulting from dietary sources (Heudorf et al. 2009). Children can also be exposed to acrylamide via inhalation of second-hand smoke (Moreno Navarro et al. 2007; WHO 2002).

In the body, acrylamide is widely dispersed by body fluids, and can also cross the placental barrier, exposing the fetus to high levels of acrylamide (Sorgel et al. 2002, WHO 2003). Fetuses and newborns do not have a fully developed blood-brain barrier, and thus, women who consume large amounts of acrylamide-containing foods risk significant acrylamide transfer to the fetus. Postnatally, acrylamide can be transferred via breast milk. According to Sorgel et al. (2002), when mothers consume foods with high acrylamide concentrations, daily consumption of 500 mL of breast milk can result in up to 10 µg of acrylamide transferred to the baby. Women who eat lesser amounts of acrylamide-containing foods, such as potato chips, may still transfer 2 µg of acrylamide to the baby. For these two scenarios, the doses for a 3 kg infant would be 3.3 and 0.66 µg/kg, respectively (Sorgel et al. 2002).

## 6.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

In addition to the individuals who are occupationally exposed to acrylamide (see Section 6.5), there are several groups within the general population that may receive potentially high exposures (higher than background levels) to acrylamide. These populations include individuals living in proximity to sites where acrylamide is produced or used in manufacturing or sites where acrylamide is disposed, and includes individuals living near the three NPL hazardous waste sites where acrylamide has been detected in some environmental media (HazDat 2007). Smokers and those breathing second-hand smoke are

subject to high acrylamide exposures (Moreno Navarro et al. 2007). Children of mothers whose diets are high in acrylamide-containing foods can be exposed to high amounts of acrylamide through breast milk (Sorgel et al. 2002).

## 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 acrylamide 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 acrylamide.

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 physical and chemical properties of acrylamide are well understood and have been discussed in Chapter 4 (Table 4-2). There are no data needs.

**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 2009, became available in February of 2011. This database is updated yearly and should provide a list of industrial production facilities and emissions.

Methods of manufacturing, production volumes, and uses of acrylamide are available and have been discussed in Chapter 5. Demand and import/export volumes are also available. No data needs are identified.

**Environmental Fate**. The environmental fate of acrylamide is reasonably well understood. If released to the environment, acrylamide has a low potential to volatilize to air from water or soil surfaces; however, its low soil adsorption coefficient suggests that it has potential to leach into groundwater. Acrylamide appears to degrade fairly rapidly by biological processes in both soil (Abdelmagid 1982) and water (Cherry et al. 1956; Croll et al. 1974). If released to air, acrylamide is expected to degrade through reaction with photochemically generated hydroxyl radicals and ozone molecules. No data needs are identified.

**Bioavailability from Environmental Media.** No data exist regarding acrylamide's bioavailability from environmental media such as soil or drinking water. Acrylamide is produced when certain foods are cooked at high temperatures (Tareke et al. 2002). Foodstuffs, particularly those rich in carbohydrates, may develop high levels of acrylamide when cooked at high temperatures (Moreno Navarro et al. 2007).

**Food Chain Bioaccumulation.** Acrylamide is not expected to significantly bioconcentrate due to its high water solubility and its ability to be degraded by microorganisms (EPA 2006c; Haberman 2002; WHO 2003). Therefore, bioaccumulation through the food chain is expected to be low. No data needs are identified.

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

**Exposure Levels in Humans.** Acrylamide was found to be present in various foods at high concentrations (Tareke et al. 2002). In order to better characterize the degree of exposure from food sources, further information is necessary to determine the background levels of acrylamide in both the environment and in humans. Additional information concerning methods to decrease acrylamide content in foods, and thus decrease exposure, is needed.

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

**Exposures of Children.** Children are exposed to acrylamide by the same routes as adults. Food was found to contribute to high levels of exposure in children, possibly 2–3 times that of an adult (WHO

2002, 2003). Inhalation of second-hand smoke can also result in exposure (Moreno Navarro et al. 2007; WHO 2002). Acrylamide has been detected in breast milk, which can result in significant exposure to infants (Sorgel et al. 2002). Additional data would be useful to determine the exposures of children to acrylamide via these methods.

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 acrylamide 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 2009) database provides additional information obtainable from a few ongoing studies that may fill data needs identified in Section 6.8.1. Titles for these studies are listed in Table 6-8.

## Table 6-8. Ongoing Research Regarding the Environmental Fate and Exposure to Acrylamide

| Investigator | Affiliation | Description | Sponsor |
|---|---|---|---|
| Lehotay SJ, Handel A | Drexel University | Development of new methods to detect and control acrylamide formation in deep-fat fried foods | USDA |
| Chen H | University of Delaware | Improvement of thermal and alternative processes for foods | USDA |
| Calder BL, Perkins LB, Bushway AA, et al. | University of Maine | Improving the post-harvest quality of fresh-cut and processed Maine potatoes | USDA |
| Daley LS | Oregon State University | Regulation of photosynthetic processes | USDA |

USDA = U.S. Department of Agriculture

Source: FEDRIP 2009