6. POTENTIAL FOR HUMAN EXPOSURE

6.1 OVERVIEW

The stable isotope of iodine, ^{127}I, and two of its radioactive isotopes, ^{129}I and ^{131}I, have been identified in at least 8, 3, and 6, respectively, of the 1,636 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 2004). However, the number of sites evaluated for iodine is not known. The frequency of these sites can be seen in Figures 6-1, 6-2, and 6-3. All of these sites are located within the United States.

Iodine is a naturally occurring constituent of the earth's crust and is the least abundant of the halogen elements (Straub et al. 1966). The stable isotope iodine, ^{127}I, is ubiquitous throughout the earth's surface. The concentration of ^{127}I in the earth's crust is approximately 0.5 ppm; in the oceans, the concentration is 45–60 µg/L, and in the atmosphere, the concentration ranges from 10 to 20 ng/m^3. Concentrations of iodine in the environment throughout the United States vary depending on the proximity to the seacoast and the soil type. The concentration of iodine in bedrock varies between 0.5 and 380 ppm, depending on whether the rock is igneous or sedimentary.

Iodine exists in many chemical forms (e.g., molecular iodine, iodide, iodate, periodate) and can undergo oxidation-reduction as well as microbial alkylation (mostly methyl iodide). Iodine has nine radioisotopes, of which ^{123}I, ^{125}I, ^{129}I, and ^{131}I are commonly encountered in acute or chronic exposures to human populations, due either to the life-times of the radioisotope in the environment, their production, and/or their use in industry, medicine, and research.

^{129}I is the only naturally occurring iodine radioisotope. It is produced as a fission product of uranium and thorium in soils and oceans, and is also formed in reactions of xenon with high energy particles in the upper atmosphere and reactions of neutrons with ^{128}Te and ^{130}Te (Soldat 1976). ^{129}I has a half-life of 1.6×10^7 years and decays through β emission; ^{129}I has a mass/decay rate equivalency of 1 g ^{129}I=6.55 MBq (177 µCi) (Robkin and Shleien 1995). ^{129}I/^{127}I ratios from natural production of ^{129}I should be 3×10^{-14} in the environment, but with the introduction of ^{129}I from nuclear weapons testing and nuclear energy activities, the ratio is now 10^{-8} (Ballad et al. 1978). The estimated global inventory of ^{129}I is approximately 9,600 Ci (0.36 PBq or 5.4×10^7 g ^{129}I), of which 9,200 Ci (0.34 PBq or 5.2×10^7 g ^{129}I) is associated with igneous activity (DOE 1994).

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

Frequency of
NPL Sites

	1
	3

Derived from HazDat 2004

6. POTENTIAL FOR HUMAN EXPOSURE

Figure 6-2. Frequency of NPL Sites with ^{129}I Contamination

Derived from HazDat 2004

6. POTENTIAL FOR HUMAN EXPOSURE

Figure 6-3. Frequency of NPL Sites with ^{131}I Contamination

Frequency of
NPL Sites

1
3

Derived from HazDat 2004

^{125}I and ^{131}I are produced in the fission of uranium and plutonium by neutron bombardment in reactors and/or heavy nuclei particles in accelerators. ^{125}I has a half-life of 60 days and decays through electron capture (EC) emitting a 35.5 keV gamma-ray and K-shell x-rays (27.4 keV, 112%; 31.4 keV, 24%). The specific activity of ^{125}I is 67.7 TBq/g or 18,277 Ci/g. ^{131}I has a half-life of 8.04 days and decays through β emission; ^{131}I has a specific activity of 457 TBq/g or 123,429 Ci/g. Unlike ^{129}I, ^{125}I and ^{131}I do not have long residency times in the environment due to their short half-lives and, thus, do not pose risks associated with an accumulation in the environment. However, in acute exposures to ^{125}I and ^{131}I, there is the potential for significant radiation exposure to the thyroid.

Releases of iodine into the environment occur from both natural sources and human activity. The natural sources include volatilization of iodine from the oceans, weathering of rock, and volcanic activity (Cohen 1985; Whitehead 1984). Sources of iodine from human activities include release of radioiodine from nuclear weapons testing and nuclear fuel reprocessing, waste stream effluent from municipal plants, and combustion of waste and fossil fuels (Likhtarev et al. 1993; Moran et al. 1999; NAS 1974; NCRP 1983; Stetar et al. 1993).

Iodine enters the atmosphere mainly through volatilization of methyl iodide and, to a lesser extent, molecular iodine from the ocean surface. ^{129}I is introduced naturally through the conversion of ^{129}Xe (xenon-129) to ^{129}I through the interaction with high energy particles in the upper atmosphere. ^{131}I was released through weapons production/utilization, nuclear fuel reprocessing, and energy production (AEC 1974; Likhtarev et al. 1993; Marter 1993; Moran et al. 1999; NCRP 1983; Robkin and Sheien 1995). In the atmosphere, iodine undergoes extensive photochemical changes and can exist as gaseous inorganic, gaseous organic, or particulate forms. These forms have an average residency time in the atmosphere of 10, 18, and 14 days, respectively (Whitehead 1984).

The gaseous inorganic and particulate forms of iodine are precipitated from the atmosphere through wet (rain, sleet, and snow) and dry (gravitational settling and wind turbulence) deposition processes (Whitehead 1984). Alkyl iodides, such as methyl iodide, have a low susceptibility to both wet and dry deposition. The deposition of iodine will depend on particle size and concentration, wind turbulence, and the chemical form of iodine. If precipitation occurs over land, iodine will be deposited onto plant surfaces or soil surfaces, or into surface waters. The average retention time of iodine on plant surfaces is 7.5–14 days due to weathering (AEC 1974; Heinemann and Vogt 1980; Kirchner 1994). Retention of iodine in the soil is influenced by a number of factors, including soil pH, soil moistness, porosity of soil,

and composition of organic and inorganic (e.g., aluminum and iron oxides) components (Sheppard et al. 1995; Whitehead 1984). Approximately 1% of iodine received through atmosphere-to-soil deposition is returned through volatilization of molecular iodine and methyl iodide; the remaining iodine is eventually returned to the oceans through surface water and groundwater (USNRC 1979; Whitehead 1984). The average residency time of iodine in the soil at 0.3- and 1-meter depths has been suggested to be 80 and 800 years, with only 1–3% of deposited iodine migrating to the 1-meter depth (DOE 1986).

Transport of iodine through surface water and groundwater is not greatly retarded by the soil, rock, and sediments over or through which these waters flow (USNRC 1981). The concentration of iodine in river water ranges between 0.1 and 18 μg/L, which parallels the concentration of iodine in rainwater of 0.1– 15 μg/L (USNRC 1979). In groundwater, the mean concentration is 1 μg/L (Yuita 1994a). The concentration of iodine in river water often increases downstream of urban areas due to the discharge of waste streams from municipal treatment facilities. This is especially true for ^{131}I that enters sewage streams from patients undergoing radioiodine therapies (Tubiana 1982; UNSCEAR 2000). Slightly elevated concentrations of [129]I have been observed in surface water and groundwater near nuclear fuel reprocessing facilities (Beals and Hayes 1995; DOE 1994).

Iodine has been shown to bioaccumulate in many seawater and freshwater aquatic plants (Poston 1986). Freshwater plants (e.g., algae) contain 10^{-5}% by weight of iodine, whereas marine plants (algae) contain 10^{-3}% by weight (NCRP 1983). In freshwater fish, iodine concentrations in tissues range from 0.003 to 0.81 ppm, which gives concentration ratios (fish/water) of 0.9–810. In marine fish, the iodine concentrations range between 0.023 and 0.11 ppm, yielding concentration ratios of between 10 and 20 (Poston 1986). In terrestrial plants, iodine can be taken up through the roots, mainly as iodide and to a lesser extent, as iodate or iodine (Burte et al. 1991; Whitehead 1984). The average iodine concentration in terrestrial plants is 0.42 μg/g. The uptake is dependent on soil conditions and the use of fertilizers (Moiseyev et al. 1984). Distribution of iodine and iodide varies throughout the plant (Voigt et al. 1988). The uptake of iodine into terrestrial plants in combination with deposition of iodine onto the surfaces of plants plays an important role in the transfer of iodine through the soil-plant-cow-milk pathway. The efficiency through which iodine is transferred through this pathway is important in ascertaining the risk of radioiodine exposures in the general human population from continuous or accidental releases of ^{131}I and [129]I, especially in children (AEC 1974; Soldat 1976; Tubiana 1982; Voigt et al. 1989).

The iodine content of food has been studied extensively, with intakes of iodine typically ranging from 0.064 to 0.379 mg/day (FDA 1974; Pennington et al. 1984, 1986). The major sources of iodine intake

from food in a typical U.S. diet are added salt and food additives, followed by meat and meat products, milk and milk products, and green/yellow vegetables (FDA 1974). Other foods that can provide a high amount of iodine in the diet include seaweed, marine shellfish, and marine fish.

It is estimated that the intake of iodine through inhalation is 4×10^{-5} g/year (USNRC 1979). The average intake of iodine from drinking water, assuming an average iodine concentration of 3 μg/L, is estimated to be 1.5×10^{-3} g/year (USNRC 1979). If the average intake of iodine from food is assumed to be the recommended dietary allowance (RDA) for iodine of 150 mg/day, then the yearly intake of iodine would be approximately 55 g. Thus, the largest source of iodine in the average U.S. diet comes from food intake. The intake of iodine through food consumption can be increased greatly in diets high in marine fish (- 800 μg/kg wet weight), shellfish (- 800 μg/kg), and seaweed-based products (0.8–4.5 g/kg dry weight) (FDA 1974). Other sources of iodine intake are alternative medicines and nutritional supplements which, depending on the specific iodine content and dosage, can approach toxic levels (e.g., >6 g/day) (Cassileth 1999).

Currently, the intake of ^{129}I and ^{131}I by the general population through inhalation, drinking water, and food intake does not pose any significant risk, due to the extremely low levels of ^{129}I and ^{131}I in the general environment. However, there are certain populations of individuals who are at risk to potential exposures to high levels of iodine or acute/chronic levels of radioiodine. Individuals undergoing specific diagnostic or therapeutic procedures or receiving certain types of medications can significantly increase whole-body and thyroid burdens of iodine and ^{131}I (FDA 1989b; Tubiana 1982). Family members, especially children, of patients undergoing ^{131}I therapies can experience exposure to both the radioisotope and the radiation emitted from ^{131}I (Barrington et al. 1999; Jacobson et al. 1978). Likewise, medical personnel working with, or in proximity to, ^{131}I can also have elevated whole-body and thyroid burdens of this radioisotope and are at risk to exposure to the photon radiation emitted from ^{131}I (Blum and Liuzzi 1967; Mountford and O'Doherty 1999; Tubiana 1982). Workers in nuclear power plants or nuclear fuel reprocessing facilities are at risk for potentially high acute exposures of ^{129}I and ^{131}I (Bhat et al. 1973; Raghavendran et al. 1978). Laboratory workers who are involved in the iodination of chemicals or biologics with ^{125}I/^{131}I or the use of these radioiodinated materials also show increased thyroid burdens of these radioisotopes (Bogdanove and Strash 1975; de Groot 1979; Dunn and Dunscombe 1981; Jönsson and Mattsson 1998; Kivinitty et al. 1984; Krzesniak et al. 1979; Kwok and Hilditch 1982; Pomroy 1979).

6.2 RELEASES TO THE ENVIRONMENT

The stable isotope of iodine, ^{127}I, and two of its radioactive isotopes, ^{129}I and ^{131}I, have been identified in 8, 3, and 6, respectively, of 1,636 current or former NPL hazardous waste sites within a variety of environmental media (air, leachate, and groundwater) collected at these sites (HazDat 2004).

Releases of iodine and its radioisotopes into the environment occur from natural sources and from human activity (Figures 6-4 and 6-5). The emphasis of the discussion of iodine release into the atmosphere will focus on atmospheric (air), water (marine and surface waters), and soils, which are major compartments in the geochemical cycling of iodine (Figures 6-4 and 6-5). Throughout this chapter, the units used to express concentration or intake of iodine and its radioisotopes are the same units reported by the authors. In some cases, values are expressed in mass units, while in other cases, the values are expressed as activities (either in Bq or Ci). For ^{129}I, the mass/decay rate equivalencies is 1 g ^{129}I = 6.55 MBq (177 µCi) (Robkin and Shleien 1995). For ^{131}I, the specific activity of this radioisotope is 457 TBq/g or 123,429 Ci/g. For ^{125}I, the specific activity is 67.7 TBq/g or 18,277 Ci/g.

6.2.1   Air

Iodine (^{127}I), ^{129}I, and ^{131}I have been identified in 2, 1, and 5 air samples, respectively, collected from the 1,636 NPL hazardous waste sites where they were detected in some environmental media (HazDat 2004).

The introduction of iodine into the atmosphere is derived from both natural and human activities (Figures 6-4 and 6-5), amounting to approximately 4×10^7 kg within the total global atmosphere at an average concentration of 10–20 ng/m^3 (Whitehead 1984). The predominant source of iodine in the atmosphere is obtained from the transfer of iodine from the ocean to the surrounding atmosphere (FDA 1974). Evaporation of sea spray is one pathway through which iodine can enter the atmosphere. However, a high ratio (ca. 1,000) of iodine to chlorine measured in the atmosphere and rainwater in comparison to that found for seawater strongly suggests that other, more important pathways are responsible for the transfer of iodine from oceans and the surrounding atmosphere. These could include the photochemical or ozone-induced oxidation of iodide to elemental iodine (NCRP 1983; Whitehead 1984). Indeed, the concentration of ozone near the ocean surface could account for upwards of 6–12×10^7 kg of iodine released yearly into the atmosphere from the world's oceans. Yet, the concentration of iodine at the ocean's surface is too low to support this iodide oxidation mechanism as a major

Figure 6-4. Geochemical Cycle of Triad Elements (I, Br, Cl)
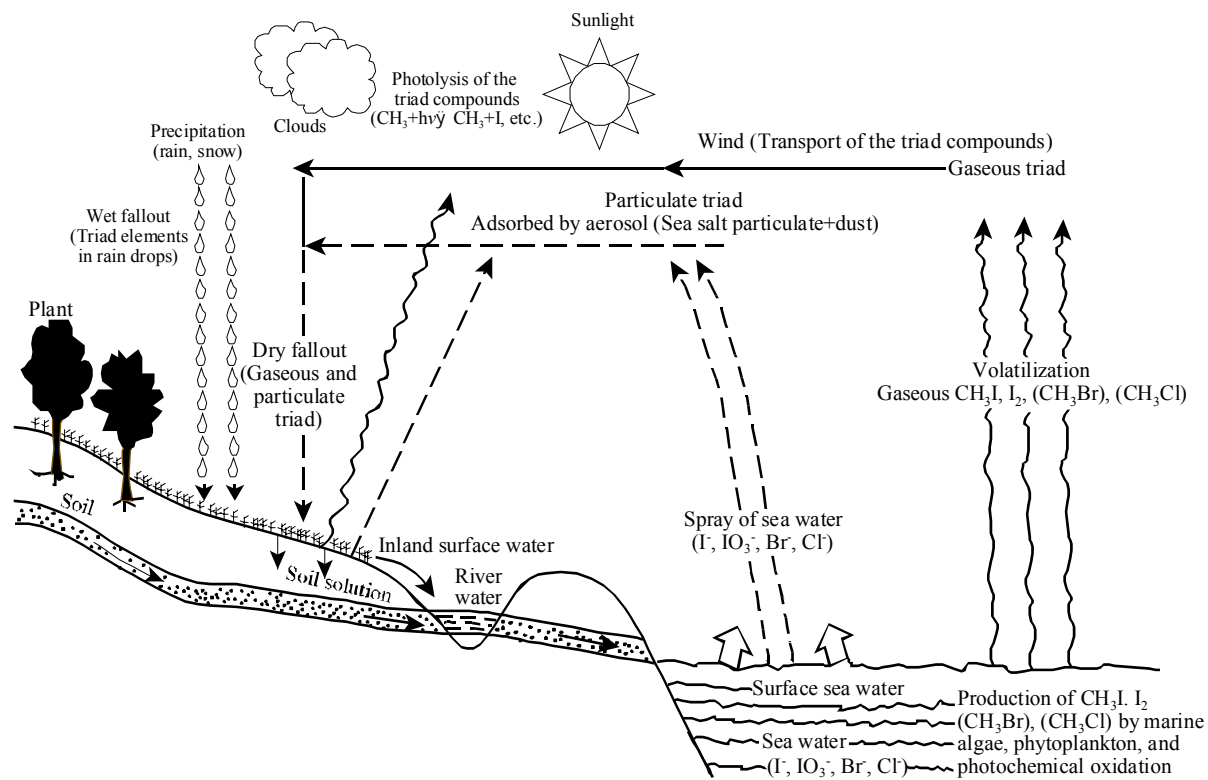
Source: Yuita 1994a

Figure 6-5. An Outline of the Movement of Iodine in the Environment*
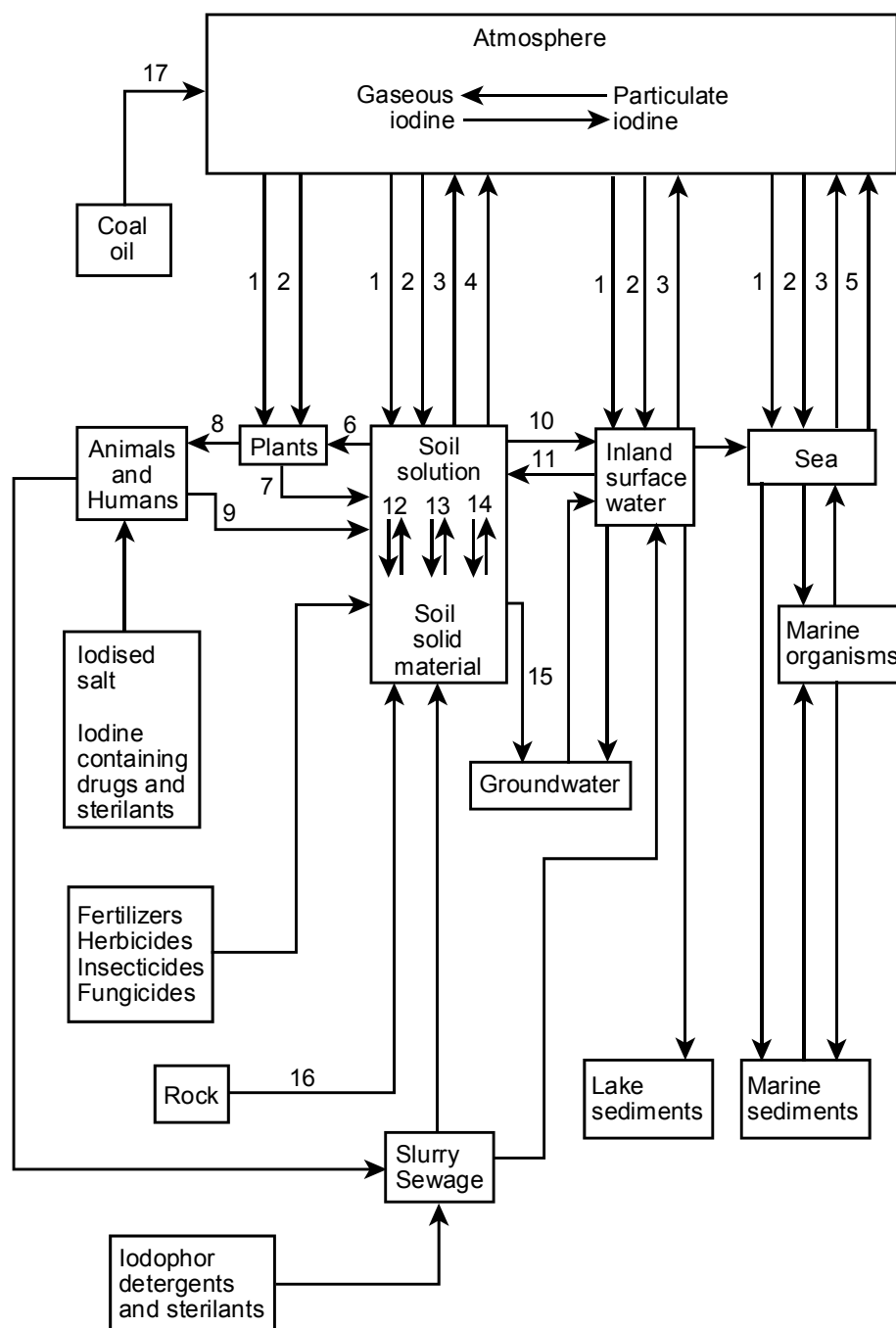
*1 = deposition in rainfall; 2 = dry deposition (including absorption by plant leaves); 3 = volatilization; 4 = suspension
of dust; 5 = suspension of marine aerosols; 6 = uptake by plant roots; 7 = decomposition of plant residues;
8 = consumption of food; 9 = decomposition of animal excreta and residues; 10 = run-off; 11 = irrigation;
12 = non-specific adsorption/desorption; 14 = immobilization/mineralisation involving soil organic matter;
15 = leaching; 16 = weathering; 17 = combustion of fossil fuels

Source: Whitehead 1984

contributor to the transfer of iodine into the atmosphere. Instead, it has been suggested that the formation of methyl iodide and other alkyl iodides from the biological metabolism of iodine/iodide may, in fact, play a major role in the annual transfer of approximately $1.3–2.0x10^9$ kg of iodine from the ocean into the atmosphere (Rasmussen et al. 1982; USNRC 1981; Whitehead 1984). Under this mechanism, the methyl iodide that is transferred into the atmosphere can undergo photolytic dissociation into methyl and I radicals, with the resultant formation of elemental iodine and other forms of inorganic iodine (HI, HOI, INO_2, $IONO_2$, OIO, etc.) (Chameides and Davis 1980; Cox et al. 1999; Vogt et al. 1999). Other sources of iodine introduction into the atmosphere from the ocean include the release of particulate forms of iodine (IO_3^-), iodine-bearing particulates, and/or organically-bound iodine into the marine atmosphere with an airborne concentration ranging from 2 to 4 ng/m^3 (NCRP 1983; Vogt et al. 1999; Whitehead 1984).

The transfer of iodine from land surface(s) into the atmosphere also occurs, but to a much lesser extent than what is observed for the transfer of iodine between the ocean and atmospheric compartments (Figures 6-4 and 6-5). It is estimated that $1.6x10^6$ kg/year of iodine is transferred into the atmosphere from surface soils and from the terrestrial biosphere, with an average airborne concentration of iodine ranging from 3 to 49 ng/m^3 as both the gaseous and particulate forms of organic and inorganic iodine (USNRC 1981; Whitehead 1984). Iodine is transferred into the atmosphere from land sources through processes such as volatilization of iodine from soil and suspension of soil. For example, it is estimated that approximately $2x10^{10}$ g of iodine (as methyl iodide) volatilizes from rice fields worldwide (Muramatsu and Yoshida 1995). Like the atmosphere over the ocean compartment, iodine is found in the gaseous form ranging from 3 to 45 ng/m^3 versus 0.5–6.9 ng/m^3 bound to particulates (Whitehead 1984). The ratios of gaseous to particulate forms of iodine (2–5) and inorganic to organic forms of iodine (0.1–2) vary, depending on location (Whitehead 1984).

A natural radioisotope of iodine, ^{129}I, is also introduced into the atmosphere. Sources of ^{129}I include interaction of ^{129}Xe with high energy particles in the upper atmosphere and, to a lesser extent, spontaneous fission and the reaction of neutrons with ^{128}Te (tellurium-128) and ^{130}Te (tellurium-130) (NCRP 1983). This leads to a natural abundance of ^{129}I that varies between 10^{-12} and 10^{-15} atoms per atom of the stable isotope of iodine, ^{127}I (Soldat 1976). This amounts to approximately 80 kg of ^{129}I in the surface environment (e.g., oceans, atmosphere, land) with $5x10^{-4}$ kg in the atmosphere (Moran et al. 1999). Because ^{129}I has a half-life of $1.6x10^7$ years, the introduction of this radioisotope into the atmosphere, and the environment as a whole, is cumulative from the standpoint of assessing human exposures.

Introduction of iodine and its radioisotopes can occur as a consequence of energy production, nuclear weapons production/use, and agricultural and medicinal/research uses. For the stable form of iodine (^{127}I), the introduction of iodine from these manufactured sources is much smaller than that observed for the introduction of iodine into the atmosphere from natural sources. However, the introduction of the radioisotopes of iodine (i.e., ^{123}I, ^{125}I, and ^{131}I) into the atmosphere, and the environment as a whole, is derived mainly from human activities.

Combustion of fossil fuels also leads to the introduction of iodine from land-based sources (Figure 6-5). The average iodine content of coal is reported to be approximately 4 mg/kg, whereas petroleum contains iodine at an average concentration of 1 mg/kg (Chameides and Davis 1980). Based on fossil fuel (e.g., coal and petroleum) consumption estimates for 1971 of approximately 2,500 million tons of oil equivalent per year (Mtoe) per year, this would amount to approximately $4x10^5$ kg of iodine introduced into the atmosphere for that year (0.1% of the total iodine transferred to and from the atmosphere) (Bertine and Goldberg 1971; Whitehead 1984). At the current rate of global coal and petroleum consumption of approximately 3,000 Mtoe/year, the introduction of iodine into the atmosphere from this source for the year 2001 would be $5x10^5$ kg per year (IEA 2000; Whitehead 1984).

The production and use of nuclear materials for the generation of electrical energy has also contributed to the release of iodine and its radioisotopes. ^{129}I is formed in nuclear fission reactions of ^{235}U and ^{239}Pu with an atomic yield of 0.12% from uranium and 0.5% from plutonium. The yields of ^{129}I from the fission of ^{235}U and ^{238}Pu are 0.9 and 1.7%, respectively (AEC 1974). This gives an approximate ratio of 4 for ^{129}I/^{127}I in the fission of ^{235}U and ^{238}Pu. Since the nuclear fuel elements are contained in a metal cladding, the release of ^{127}I and ^{129}I that is produced in the fission reactions does not occur until the fuel is reprocessed. For example, it is estimated that the DOE Savannah River fuel reprocessing plant released approximately 2.8 kg/year of ^{129}I during 1964–1965, but has now fallen below 0.7 kg/year since the 1970s. This amounts to a total of 5.7 Ci (210 GBq or 32 kg) of ^{129}I released during the 1954–1989 operating history of the plant (DOE 1998; Marter 1993). In 1999, the release into air of 7.27 mCi (0.269 GBq or 41 g) of ^{129}I and 10.1 µCi (0.374 MBq or 0.0818 ng) of ^{131}I was reported at the Savannah River site (DOE 1999). Releases of ^{129}I from the Hanford Reservation between 1944 and 1995 are estimated to be 1,900 GBq (51 Ci or 290 kg ^{129}I) (Robkin and Sheien 1995). The releases of another iodine radioisotope, ^{131}I, during the years 1966–1972 was estimated to be $3x10^5$ Bq ($8x10^{-6}$ Ci)/MW(e)y from fuel reprocessing. From the Savannah site, an estimated 2,520 Ci (93.2 TBq or 20.4 mg) of ^{131}I was released from 1954 to 1989 (DOE 1998; Marter 1993). The average releases of ^{131}I from boiling water

reactors (BWR) ranges between $2x10^{-3}$ Ci ($7x10^7$ Bq) and $5x10^{-3}$ Ci ($2x10^8$ Bq) per MW(e)y, and for

pressurized water reactors (PWR), the values range from $5x10^{-5}$ Ci ($2x10^6$ Bq) to $50x10^{-5}$ Ci ($2x10^7$ Bq)

per MW(e)y (NRCC 1980).

Surface testing of nuclear weapons release of ^{129}I into the environment from nuclear explosions of ^{235}U

and ^{239}Pu amounted to approximately 30 and 50 µCi (1.1 and 1.9 MBq) per kiloton, respectively. Thus, it

is estimated that atmospheric testing of nuclear weapons has released approximately 50 kg of ^{129}I into the

atmosphere. The transport and diffusion of this radioisotope depended on initial height of the nuclear

cloud and meteorological conditions; residency times were <0.5 years in the lower stratosphere and

approximately 2 years at medium altitudes. Diffusion of radioisotopes from higher to lower altitudes then

deposited through either precipitation or dry deposition (NCRP 1983); dry deposition could be as

important as precipitation in surface deposition of iodine (Machta 1963; Straub et al. 1966). Release of

^{131}I also occurred during these surface tests of nuclear weapons; however, the ^{131}I that was released into

the environment from these tests has decayed (half-life of 8.04 days) to levels that are no longer of

concern in the environment.

Accidental releases of iodine and its radioisotopes are also sources of iodine introduction into the

atmosphere. The 1986 Chernobyl reactor accident has released an estimated 1.3 kg of ^{129}I and 1,200–

1,700 PBq (2.6–3.7 kg) of ^{131}I into the atmosphere (Balonov et al. 1999; Likhtarev et al. 1993; Moran et

al. 1999; Mould 2000). Other notable accidental releases of ^{131}I include the 1957 Windscale, United

Kingdom, radiochemical plant fire and the Three Mile Island accident that released approximately

700 TBq (2 kCi or 2 g) and 0.6 TBq (2 Ci or 1 mg) of ^{131}I, respectively (Likhtarev et al. 1993)

6.2.2   Water

Iodine (^{127}I), ^{129}I, and ^{131}I have been identified in 1, 2, and 1 groundwater samples, respectively, and

no surface water samples collected from the 1,636 NPL hazardous waste sites, where they were detected

in some environmental media (HazDat 2004).

Introduction of iodine into surface waters and groundwater occurs predominately through rainwater for

noncoastal land regions and the combination of rainwater and ocean spray in coastal regions (Figures 6-4

and 6-5). It is estimated that $1.0x10^{11}$ g/year of iodine is deposited onto land surfaces, of which

$8.1x10^{10}$ g/year enters surface waters and $1.5x10^{10}$ enters groundwater (USNRC 1981). The iodine in

rainwater is derived from the transfer of iodine from the oceans to the atmosphere (FDA 1974).

Other natural releases of iodine into surface waters and groundwater include the leaching of iodine from the weathering of rock and volcanic activity (Figure 6-5). It is estimated that rocks contribute between $1x10^9$ and $1.6x10^{10}$ g/year depending on the iodine content of the rock (0.5–8.8 ppm) (Cohen 1985). Volcanic activity can add an estimated $1.2x10^9$ g of iodine per year to the surface environment, where the greatest contribution to the oceans is due to undersea volcanic activity (Miyake and Tsunogai 1963; USNRC 1979).

Municipal waste water treatment plants introduce iodine and ^{131}I into surface waters, predominantly derived from human waste and the use of ^{131}I in medical treatments. Iodine is poorly captured within sludge (2–25%), with the remainder released into surface waters (1.0–16 μg/L) in the waste water stream (NAS 1974; Stetar et al. 1993).

Release of radioiodine has occurred as a result of the reprocessing of nuclear fuel. Release of ^{129}I from waste water generated by the Idaho National Engineering Laboratory into Snake River Plain aquifer through deep disposal wells (before February 1984) and unlined disposal ponds (1984–1990) amounted to approximately 0.56–1.18 Ci (21–44 GBq or 3.2–6.7 kg of ^{129}I) (DOE 1994). This release in both shallow and deep groundwater horizons has been minimized through the recycling of the waste stream and storage of this stream with high-level radioactive waste. Release of ^{131}I into streams on the DOE Savannah River site between 1957 and 1978 totaled 300 Ci (11.1 TBq or 2.43 mg ^{131}I) (DOE 1998). In 1999, 0.0782 Ci (2.89 GBq or 441 g) of ^{129}I was released into surface waters at the DOE Savannah River site (DOE 1999). The Sellafield (United Kingdom) and Cape de la Hague (France) reprocessing facilities have cumulatively released 1,440 kg of ^{129}I directly into ocean waters since operations began in the 1960s; direct releases into the ocean amount to 200 kg/year since 1994 and have been increasing (Moran et al. 1999).

6.2.3   Soil

Iodine (^{127}I), ^{129}I, and ^{131}I have not been identified in soil or sediment samples collected from the 1,636 NPL hazardous waste sites (HazDat 2004).

The contribution of iodine to soils is derived from natural sources, such as the weathering of rock, decay of vegetation, iodine received from rainfall, and from human activities (Figures 6-4 and 6-5). Most soils contain, on average, approximately 5 mg/kg of iodine worldwide (Whitehead 1984). It is thought that

only a small proportion of this iodine is derived from the weathering of rock (Fuge 1987; Goldschmidt 1958; Whitehead 1984), although there is some argument to the contrary (Cohen 1985). The natural content of iodine in natural geologic materials is (in ppm): ultramafic igneous (0.06–0.3), basaltic igneous (0.5), granitic igneous (0.5), shales/clays (212–380), deep sea clays (11–50), limestones (0.4–29), sandstones (1.7), and coals/ash (1–11) (NAS 1974). It is expected that the contribution of iodine to soils in regions where the bedrock is composed primarily of igneous rock will be much less than that for regions where the underlying bedrock is composed of sedimentary rock, which has a higher iodine content (Cohen 1985; NAS 1974; Whitehead 1984).

Wet deposition of iodine from the atmosphere in rain or snowfall contains an average of 2.0 µg iodine/L. Assuming an average precipitation rate of 800 mm (32 in) per year, the wet deposition of iodine would amount to the addition of 16 g iodine/ha'year (Whitehead 1984). Dry deposition of iodine in a particulate form or bound to a particulate carrier can add an estimated 9.6 g iodine/ha'year, assuming a deposition rate of 0.2 cm/second and an average concentration of 15 ng/m^3 of particulate iodine (Whitehead 1984). However, the iodine that is derived from both the wet and dry deposition will only increase the content of the soil (to a 15 cm depth) approximately 0.7 ng/g if all of the iodine is effectively trapped in the soil (Whitehead 1984).

Agricultural activities increase iodine in soils through animal excrement and the use of fertilizers/pesticides (Figure 6-5). Animal feces can contain up to 10 mg/kg iodine, and urine can contain up to 4 mg/L iodine. The iodine in sewage sludges and fertilizers used in agriculture can vary between 0.5 and 30 mg/kg. Most inorganic fertilizers contain <0.5 µg/g iodine, except fertilizers containing Chilean nitrate, which can provide upwards of 80 µg/g iodine. Superphosphate and compound fertilizers derived from rock phosphate can contain up to 26 µg/g iodine (Whitehead 1979). The use of fertilizers, however, will not result in an appreciable increase in the iodine content of soil (Whitehead 1984). Yet, the use of iodine-containing herbicides, such as ioxynil/ioxynil octanoate (recommended application of 0.5 kg/ha) and the fungicide benodanil (recommended application of 1.1 kg/ha), can increase iodine content of soil about 0.17 and 0.21 µg/g to a depth of 15 cm, respectively (Whitehead 1979, 1984).

Combustion of coal and petroleum is another source of iodine in soils. In the combustion of coal and petroleum, the iodine is introduced to the atmosphere through the flue gases. A large proportion (- 80%) of the iodine released through the flue gases is deposited onto the surrounding soils by both wet and dry deposition, adding approximately 4×10^5 kg iodine/year to soils globally.

6.3 ENVIRONMENTAL FATE

6.3.1   Transport and Partitioning

It is estimated that the earth's surface contains 6.3×10^{18} g of iodine. The majority of this iodine is in the
earth's crust (3.4×10^{18} g) and in sedimentary rock (2.9×10^{18} g). However, this iodine is inaccessible,
except for the small portion that is liberated through weathering processes ($\approx 10^{9}$ g/year) and eventually
enters into the oceans. The ocean, on the other hand, is the largest compartment of accessible iodine that
can be transferred to other environmental compartments. The earth's oceans contain 8.1×10^{16} g of iodine
(Figure 6-6) at an average concentration of between 45 and 60 μg/L. Other environmental compartments
with steady-state levels of iodine include atmosphere (8.8×10^{10} g), surface soil (4.1×10^{14} g), subsurface
region (2.5×10^{13} g), and terrestrial biosphere (3.0×10^{11} g). The iodine in the ocean is in equilibrium with
the iodine in ocean sediments (8.0×10^{17} g), with a net flux of 1.8×10^{11} g/year. A net transfer of iodine
also occurs between the ocean surface and the global atmosphere at an average rate of 2.0×10^{12} g/year, of
which 1.9×10^{12} g/year returns to the ocean through wet/dry deposition processes, and 1.2×10^{11} g/year is
deposited onto land surfaces. Of the iodine that is deposited on land, 7.7×10^{10} g/year is returned to the
ocean through groundwater and river effluents, and 1.6×10^{10} g/year enters into the terrestrial biosphere, of
which 1.4×10^{10} g/year returns to the soil surface through weathering and decay of vegetation (USNRC
1979).

The transfer of iodine between the ocean compartment, atmosphere, and land surfaces is due to the
volatility of iodine in its molecular (I_2) and organic (most methyl iodide) forms. The extent to which
iodine partitions into these compartments, and its residency time, will depend on the chemical form of the
iodine as it enters into a specific compartment, any chemical alterations that the iodine undergoes in that
particular compartment, and the solubility/uptake/retention of the various chemical forms of iodine in the
compartment. The formation of alkyl iodides (predominantly methyl iodide) and, to a lesser extent,
molecular iodine from biological activity and photochemical reactions on the ocean's surface, provide for
the transfer of these iodine species into the ocean atmosphere, where they undergo further photochemical
conversions into other gaseous and particulate forms of iodine (Cox et al. 1999; Filistovic and
Nedveckait 1998; Vogt et al. 1999; Whitehead 1984). The relative proportions of iodine as inorganic
particulates and organic gaseous forms are on average 25% for particulates and 40–80% for organic forms
as methyl iodide (Moran et al. 1999). The residence times for iodine in the atmosphere are 14 days for
particulates, 10 days for inorganic gases (i.e., I_2), and 18 days for organic gases (compared to a 9-day

Figure 6-6. Diagram of the Global Iodine Cycle at Steady State Showing Environmental Compartments, Compartment Inventories in Grams (g), Transport Pathways, and Fluxes in Grams per Year (g/year)
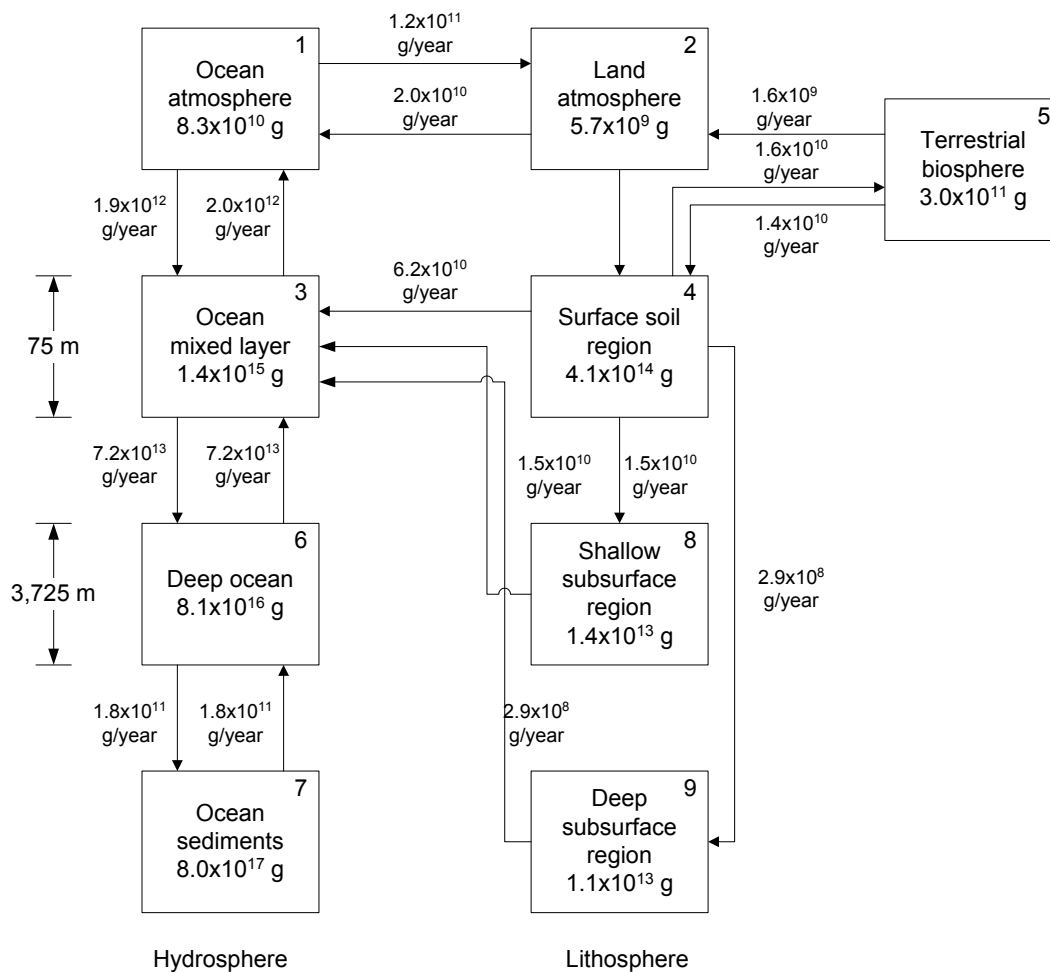
Source: Kocher 1981

residency time for water vapor), providing for extended global transport and substantial mixing (Moran et al. 1999).

The gaseous and particulate forms of iodine in the atmosphere are deposited onto ocean or land surfaces through wet and dry deposition. Gaseous elemental iodine and particulate forms of iodine are susceptible to wet deposition, whereas methyl iodide has a low susceptibility. Accordingly, gaseous molecular iodine and particulate forms of iodine are susceptible to dry deposition; methyl iodide has a low susceptibility (Whitehead 1984).

The dry deposition rate is dependent on particle size, wind speed, and turbulence. Iodine will settle onto soil and plant surfaces. Direct deposition of iodine onto plant surfaces is limited to 7.5–14 days, where particulate iodine is removed from plant surfaces through weathering processes (AEC 1974; Heinemann and Vogt 1980; Kirchner 1994). Dry deposition onto plant surfaces is affected by the moistness of the surface; deposition is approximately 2-fold greater on moist plant surfaces versus dry surfaces (Heinemann and Vogt 1980). Also, dry deposition onto plants is affected by the surface area of the plant, as is evident from the 2-fold increase in iodine deposited on clover versus grasses (Heinemann and Vogt 1980).

The wet deposition of iodine will be predominantly deposited into soil. The relative amounts of iodine initially depositing onto the soil will greatly depend on the density and type of plant cover over the soil. Upwards of 90% of elemental iodine vapor can be intercepted by a dense cover of grassland herbage, but 40–70% can typically be expected to be intercepted by a more average density of plant cover (Whitehead 1984).

Evaporation of iodine from the land surface to the atmosphere is only about 1% of the flux of iodine from the atmosphere to the land surface (USNRC 1979) and iodine is cycled back to the ocean through groundwater and river effluent (USNRC 1979; Whitehead 1984). However, the overall content of iodine in a soil is determined by the inputs of iodine into the soil and the ability of the soil to retain iodine (versus leaching and volatilization), where the main input is from atmospheric deposition, both wet and dry, followed by degradation of plant material (mostly from adsorbed iodine) (Whitehead 1984).

The low flux of iodine from land surfaces to the atmosphere is due to the retention of iodine within surface soils, especially in soils rich in organic matter and iron/aluminum oxides (Sheppard et al. 1995). When the various chemical forms of iodine enter into the soil, these species are initially retained by their

absorbance onto soil components in equilibrium with their solubility in soil solution. Isotopic exchange studies indicate that between 5 and 30% of iodine is exchangeable (Whitehead 1984). Retention of inorganic and organic iodine will depend on both nonspecific and specific sorption onto soil components. Nonspecific ion-exchange interactions of iodide and iodate anions occur on free hydrous oxides of iron and aluminum; such exchanges involve electrostatic attractions and are dependent on pH and the concentration of other anions (Sheppard et al. 1995). Retention of molecular iodine in soil is thought to be mediated through the interaction of iodine with thiols and polyphenols present in the organic components of soils (Fawcett and Kirkwood 1953; Jirousek and Pritchard 1971; Whitehead 1984) and may also involve the oxidation/reduction of iodide and free radical reactions (Huang and Lu 1991). Methyl iodide is sorbed by soils to a lesser extent than inorganic iodide, but the factors that determine this sorption of methyl iodide to soils are unclear (Whitehead 1984).

Transport of iodine to lower soil depths is dependent on the porosity and saturation of the soil. Macropores formed from roots and earthworm channels allow for rapid transport of iodine into the soil. Mobility of iodine into the soil is greatest when the soil is saturated with water. The drier the soil, the thinner the water films within the soil, thus limiting the flow rate of water through the soil (Whitehead 1984.

In addition to the aforementioned direct deposition of particulate deposition of iodine onto plant surfaces, there is also evidence of the uptake of inorganic iodine into the plant through the roots and gaseous iodine through the leaves (Whitehead 1984). Iodide is more readily taken up into plant roots than is iodate or iodine (Burte et al. 1991; Whitehead 1984); the uptake is dependent on the concentration of iodine in the soil, the properties of the soil, and the use of fertilizers (Moiseyev et al. 1984; Shinonaga et al. 2001). Soil-to-plant transfer factors (TF), which are defined as the grams iodine per kilogram wet or dry weight of plant material divided by the grams of iodine per kilograms dry soil, typically range between 0.001 and 1.5 for plants of agricultural importance (Shinonaga et al. 2001). Molecular iodine can be absorbed through the stomata in the leaves, whereas methyl iodide and hypoiodous acid are not as readily absorbed through this route (Whitehead 1984).

Both the deposition of particulate iodine onto plant surfaces and the direct uptake of iodine into the plant factor into the transfer of iodine through the soil-plant-cow-milk pathway. The level of iodine in feed has a direct relationship with the level of iodine measured in milk (Tracy et al. 1989; Voigt et al. 1989) and is dependent on the season, nutritive quality of pastureland, and ambient temperature (Ekman et al. 1967; Lengemann and Wentworth 1979; Pennington 1990a). The transfer coefficient for iodine through the

total pathway is approximately 0.003–0.01 for cow and 0.2 for goat, expressed as the fraction of daily intake per liter milk (d/L) (AEC 1974; Kirchner 1994; Voigt et al. 1988, 1989).

Transfer of iodine in the feed-meat pathway has also been determined in various animals and tissues. Transfer factors in whole animals (expressed as the fraction of daily intake per kg [d/kg]) are 0.02 (beef), 0.09 (pork), and 0.004 (chicken). The thyroid is the organ with the greatest uptake of iodine (AEC 1974). Individual tissue measurements have transfer factors range from 10^{-4} (kidney) to 10^{-3} (liver) to 10^{-2} (muscle) (Handl and Pfau 1989; Handl et al. 1990). Transfer factors have also been determined for feed-chicken eggs (0.03) (AEC 1974).

Iodine can also bioaccumulate to varying extents in aquatic organisms. Aquatic bioaccumulation factors for iodine in fresh water are 40 (algae), 5 (invertebrates), and 15 (fish); in salt water, these factors are 4,000–10,000 (algae), 50–100 (invertebrates), and 10–20 (fish) (AEC 1974). Certain seaweeds and algae can concentrate iodine to levels as high as 0.8–4.5 g/kg of dried material; these high levels are usually associated with the relatively high levels of iodine in seawater (50 µg/kg) (FDA 1974).
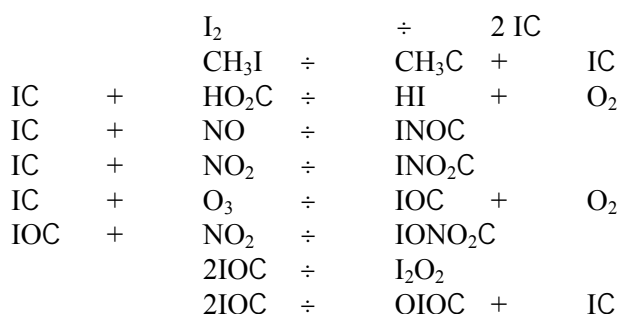
6.3.2   Transformation and Degradation

Iodine consists of one stable isotope (^{127}I) and a number of radioisotopes, of which ^{123}I, ^{125}I, ^{129}I and ^{131}I are the most common in environmental and occupational exposures. The radioisotopes ^{129}I and ^{131}I decay by β-emission to form ^{129}Xe and ^{131}Xe, respectively, whereas ^{125}I decays by electron capture, emitting gamma and Te x-rays. ^{125}I and ^{131}I disappear rapidly from the environment due to their short half-lives of 60 and 8.04 days, respectively, and do not undergo long-term accumulation in the environment. However, the long physical half-life of ^{129}I (1.57×10^7 years) means that any release of this radioisotope into the environment is essentially a permanent addition to the total inventory of iodine in the environment from the standpoint of assessing human exposures (NCRP 1983).

The chemical reactions of iodine and its radioisotopes within the environment are the same, but the ratios of the isotopes in these reactions may differ, depending on the relative concentrations of the isotopes in a particular environment. ^{127}I and its radioisotopes can exist in many forms and oxidation states (iodides [-1], molecular iodine [0], iodohalides [+1], iodates [+5], and periodates [+7]) (Holland 1963); organic forms of iodine include methyl iodide, ethyl iodide, isopropyl iodide, and methylene iodide (Vogt et al. 1999). Thus, "iodine" in the discussion of the chemical reactions of iodine in the environment will be used to refer to both iodine and its radioisotopes, unless indicated otherwise.

6.3.2.1 Air

The major source of iodine in air is from the evaporation of alkyl iodides (mostly methyl iodide) and, to a lesser extent, molecular iodine from ocean surfaces (Figures 6-4 and 6-7). At ordinary pressures and temperature, methyl iodide and iodine have high vapor pressures and will exist predominately in a free gaseous form in air. Both iodine and methyl iodide undergo photochemical reactions to form iodine radicals (IO), which can then go on to form a number of other iodine species through a complex series of reaction pathways (Cox et al. 1999; Filistovic and Nedveckait 1998; Vogt et al. 1999); some of these are shown below and in Figure 6-4.

$$
\begin{array}{lllllll}
I_2 & & & \div & 2\,IC & & \\
CH_3I & \div & & CH_3C & + & IC & \\
IC & + & HO_2C & \div & HI & + & O_2 \\
IC & + & NO & \div & INOC & & \\
IC & + & NO_2 & \div & INO_2C & & \\
IC & + & O_3 & \div & IOC & + & O_2 \\
IOC & + & NO_2 & \div & IONO_2C & & \\
& & 2IOC & \div & I_2O_2 & & \\
& & 2IOC & \div & OIOC & + & IC \\
\end{array}
$$

The overall photochemical dissociation of CH_3I and I_2 in the atmosphere results in the steady-state formation of inorganic iodine species consisting mostly of $IONO_2C$(75%), IOC(15%), and HI+HOI (10%) during the daytime. Only minor changes in these percentages are observed during the night-time, $IONO_2C$(75%), IOC(7%), and HI+HOI (18%) (Filistovic and Nedveckait 1998).

Some of these iodine species (e.g., IOC, I_2O_2) can then go on to react in aerosols or water droplets to form IO_2^- and IO_3^- (Figure 6-7). Gaseous iodine can also dissolve in water droplets to form iodide or hypoiodate ions, especially in the presence of alkaline impurities. Iodine is readily reduced to iodide by hydrogen sulfide and can be oxidized to free iodine by ozone, hydrogen peroxide, or possibly air and sunlight. If the iodine is acidified sufficiently, gaseous hydrogen iodide is liberated (Straub et al. 1966). Conversely, HI formed in air can dissolve in water droplets, forming iodide anions that can be oxidized to molecular iodine.

Figure 6-7. A Simplified Scheme of Iodine Cycling in the Marine Boundary Layer*
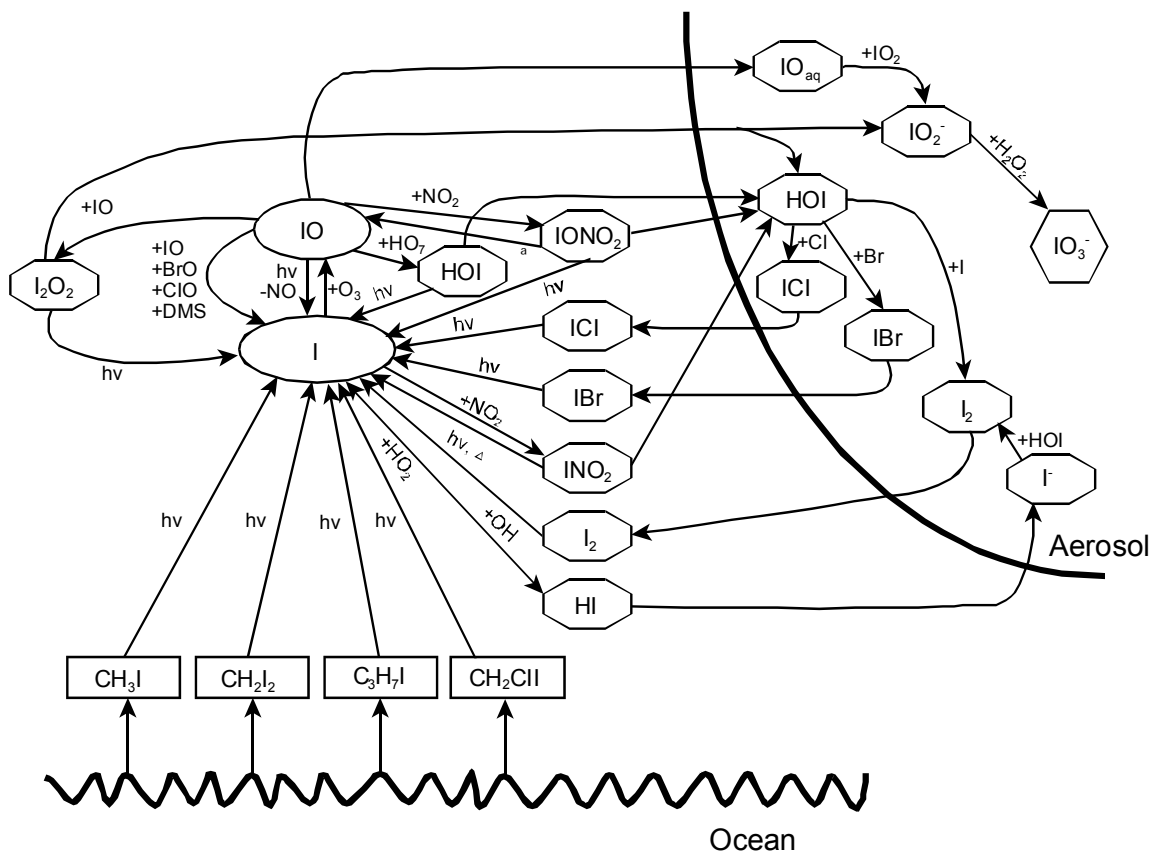
*Organic iodine compounds are shown in rectangular boxes; temporary iodine reservoir species in the gas and in the aerosol phase are shown in octagons

Source: Vogt et al. 1999

6.3.2.2 Water

Iodine in water exists as iodide and iodate. In rainwater, the relative proportion of iodide to iodate is 55:45. In surface waters, the proportion of iodide to iodate will vary depending on microbial activity and the release of iodine species from terrestrial sources (De Luca Rebello et al. 1990). Iodide is also converted in the surface layer of seawater to hypoiodous acid (HOI) by photochemically generated ozone (Bichsel and von Gunten 1999). Microbial action converts iodide to organic forms of iodine, primarily methyl iodide (iodomethane). The low vapor pressure and limited solubility in water promote the volatilization of methyl iodide from surface waters to the surrounding atmosphere. Likewise, microbial activity and photochemical reactions, including photolysis of biogenic iodine, can lead to the formation of iodine from iodide or iodate; iodine can evaporate into the atmosphere due to its low vapor pressure (Xie et al. 1999). In marine waters, up to 40% of iodine can be found as dissolved organic iodine (DOI), with the remainder as iodide (Wong and Cheng 1998).

Disinfection of natural waters results in the oxidation of iodide to hypoiodous acid (HOI) (Bichsel and von Gunten 1999). Ozone, chlorine, and monochloramine easily oxidize iodide to HOI. Ozone, at concentrations used in the disinfection of water, rapidly oxidizes HOI and hypoiodate (OI$^-$) to iodinate (IO$_3^-$), whereas chlorine oxidizes HOI in a slower, more complex reaction mechanism. Monochloramine was unable to oxidize HOI. As a consequence, the formation of iodoorganics (e.g., iodoform or CH$_3$I), which results from the reaction of HOI with organics in natural waters and often causes a problems with the taste and odor of drinking water, is much more prevalent when chlorine and chloramines are used as oxidants in the disinfection process.

6.3.2.3 Sediment and Soil

Iodine can enter sediments through accumulation of plant matter or fixation of iodide in water to humic substances in the sediments through microbial action (R@llinger and Heumann 2000). Weaker and reversible binding of iodide to inorganic components in sediments has also been shown to occur, with affinities measured as partition coefficients [K$_d$s] ranging from -0.22 mL/g for chlorite minerals to 15.14 mL/g for illite minerals (Kaplan et al. 2000). Iodine can enter soil as I$_2$, iodide, iodate, or methyl iodide through wet or dry deposition. Molecular iodine dissolves in soil water to form iodide or is oxidized to iodate. Conversely, chemical, and to a lesser extent, microbial, reduction of iodide or iodate

forms molecular iodine that can evaporate from the soil water into the atmosphere. Iodine can react with organic components of soil, such as humic substances, undergoing iodination reactions with polyphenols and tyrosine residues (Fawcett and Kirkwood 1953; Jirousek and Pritchard 1971; Whitehead 1984), which may involve the oxidation/reduction of iodide and free radical reactions (Huang and Lu 1991).

Decreases in pH can alter the proportion of iodide to iodate in soil and water due to the protonation of iodide to form HI, which can volatilize into the atmosphere. The relative proportions of iodine species can also differ in soils under flooded and nonflooded conditions. The proportions of I_2:I^-:IO_3^-:organic iodine change from 0.045:0.065:0.89:0.016 under the oxidizing nonflooded conditions to 0.007:0.90:0.097:0 under the reducing flooded conditions (Yuita 1994a).

6.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

6.4.1   Air

The average global air iodine concentration ranges between 10 and 20 ng/m^3 with gaseous iodine usually exceeding particulate iodine by a factor of 2–6 (Whitehead 1984). Atmospheric concentrations over land range from 2 to 14 ng/m^3, while atmospheric levels over oceans average between 17 and 52 ng/m^3 (USNRC 1979). In an urban air environment, iodine content in air over San Francisco in 1970 ranged between 4.7 and 10 ng/m^3 over nine monitoring stations (John et al. 1973). In remote air environments, for example the arctic, the iodine concentrations fall dramatically. Iodine in the arctic stratosphere is 0.27 ng/m^3, and is 0.33 ng/m^3 at the marine boundary layer (Sheridan and Zoller 1989). The annual average concentration of iodine in Canadian arctic air ranges between 0.43 and 0.96 ng/m^3 (Barrie and Hoff 1985)

Iodine introduction in the form of methyl iodide emission from a municipal waste incineration plant has been measured at an average of 0.50 μg/m^3 in Karlsruhe, Germany (Stieglitz 1995).

Radioiodine releases from nuclear fuel reprocessing facilities have been well documented. Discharges of ^{129}I from the Hanford Reservation between 1983 and 1992 ranged from 0.02 to 0.6 Ci/year, with a maximum output of 0.5–0.6 between 1986 and 1988 (DOE 1993). The average airborne concentrations of ^{129}I onsite ranged from 69–2,000 to 2.2–60 attoCi/m^3 (atto=10^{-18}) along the perimeter to 0.13–2.4 attoCi/m^3 at distant monitoring sites (DOE 1993). At the Savannah River site, ^{129}I and ^{131}I releases between 1954 and 1989 totaled 5.67 and 2.52 Ci, respectively (DOE 1990). Discharges of ^{129}I into the air from the Sellafield (United Kingdom) and La Hague (France) nuclear processing facilities is thought to

contribute to the $4–40 \times 10^8$ atoms ^{129}I/L (15–150 attoCi/L or 0.086–0.86 ng/L) measured in rain and snow over Sweden between 1998 and 1999, although it is not clear how much of these releases contributed to the total concentration of ^{129}I in precipitation (Buraglio et al. 2001). Strong seasonal variations in the concentration of ^{129}I in precipitation are observed, due to such factors as seasonal changes in the volatilization of $CH_3{}^{129}$I from soils and decaying plant matter, and seasonal changes in weather patterns that determine the source and availability of moisture in the Baltic region.

The release of radioiodine into the atmosphere from nuclear weapons testing and its deposition onto distant targets has been measured. ^{131}I that was produced in the above-ground nuclear Test Harry and deposited (wet) on select New England monitoring sites ranged from 0.2×10^9 Bq/km in Buffalo to 3.4×10^9 Bq/km in Pittsburgh to 6.1×10^9 Bq/km in Binghamton (Hoecker and Machta 1990). The emissions of radioiodine are often associated with particulates. The amount of ^{131}I associated with particulates increases with the distance from the source of release. Upwards of 60% of ^{131}I released into atmosphere is associated with particulates, based on ground-level measurements. It is assumed that 80–85% of the fallout of ^{131}I is in the reduced state, 15–20% is present as $IO_3{}^-$, and a few percent or more is present as $IO_4{}^-$ (Perkins 1963; Straub et al. 1966). The cumulative release of ^{129}I (expressed as Ci) in nuclear weapons testing beginning in 1945 and ending in 1975 has been estimated and is shown in Table 6-1 (NCRP 1983).

6.4.2   Water

The average iodine content in seawater is 40–65 µg/L (USNRC 1979). The iodine content in rainwater averages between 0.1 and 15 µg/L, and in rainwater over oceans, the iodine content is 1–15 µg/L (USNRC 1979). The iodine content in river water averages between 0.1 and 18 µg/L (USNRC 1979). The concentration of iodine in river water will be locally influenced by municipal waste water streams. The average iodine content in municipal waste water effluent is 4.0 µg/L (range 1.0–16 µg/L) (NAS 1974). In groundwater, the average iodine concentration is 1 µg/L (Yuita 1994a).

The concentration of ^{129}I in ocean surface waters averages between 10^7 and 10^8 atoms ^{129}I per kg (Cooper et al. 2001). Some examples reported by Cooper et al. (2001) are $0.18–0.82 \times 10^8$ atoms ^{129}I/kg in the Bering, East, and Chukchi Seas, with several concentrations as high as 9.60×10^8 and 16.7×10^8 atoms ^{129}I/kg in the Chirikov and Chukchi benthic zones, respectively. Cooper et al. (2001) also showed that the concentration of ^{129}I in ocean waters varies as a function of depth of the water column. In the East Sea

Table 6-1. Approximate Releases of ^{129}I from Atmospheric and High Altitude Nuclear Weapons Tests

Year	Cumulative ^{129}I Released (Ci)
1945–1951	0.04
1952–1954	2
1955–1956	3
1957–1958	5
1959–1961	6
1962–1963	10
1963–1975	10

Source: NCRP 1983

(Sea of Japan), the concentration of ^{129}I decreased from approximately 0.35×10^8 atoms ^{129}I/kg in surfaces water to 0.05×10^8 atoms/kg at a depth of 2,000 meters, but increased again to 0.15×10^8 atoms/kg at a depth of 2,800 meters near the sea floor (3,000 meters). It is suspected that the increase in ^{129}I concentration near the sea floor is due to a flux of iodine from sediments (^{129}I concentrations in sediments were $168–902 \times 10^8$ atoms/kg), although this could not be confirmed in their study.

The iodine content in drinking water typically varies between 0 and 8 µg/kg, with a more nominal range averaging between 2 and 4 µg/kg. Concentrations of iodine in drinking water approaching or exceeding 8 µg/kg are usually associated with water that is directly contaminated with sewage or effluent from sewage discharge sites or from urban run-off (FDA 1974). For example, the concentration of iodine in the Potomac River was 4.0 µg/L upstream of Alexandria, but increased to 8.0 µg/L downstream. Sewage effluent from Alexandria was believed to be the cause. Some effluent streams can have iodine concentrations as high as 1,910 µg/L (FDA 1974).

Seasonal variations in iodine have been measured in Rhode Island precipitation. The volume-weighted mean concentration in annual precipitation is 1.71 µg/L, with a concentration range of 0.04–11.3 µg/L. The seasonal variations in volume-weighted iodine concentration were 2.14 µg/L during the warm season and 1.35 µg/L during the cold season (Heaton et al. 1990).

Radioiodine is released into surface waters through the effluent of municipal sewage treatment plants. On an average working day, approximately 24 mCi (0.89 GBq) of ^{131}I is released into the Ohio River, resulting in a concentration of 0.3 pCi (11 mBq) ^{131}I/L downstream from the treatment plant (Sodd et al. 1975). The concentration of ^{131}I in water downstream from other municipal treatment plants throughout the United States has also been determined, ranging from 0 to 83 pCi (0 –3.1 Bq) ^{131}I/L (Prichard et al. 1981). The ^{131}I that has been measured in these waters is due to the introduction of ^{131}I into the sewer systems of cities from the excrement of patients undergoing treatments with this radioisotope.

The release of ^{129}I into surface waters has also been measured. It has been estimated that the above-ground total fission yield of a 207 megaton equivalent of plutonium fission devices contributed approximately 10 Ci (370 GBq) of ^{129}I to the environment (NCRP 1983). Reprocessing of spent fuel could release additional amounts of ^{129}I into the environment (at most 2 Ci or 70 GBq) depending on the amount of fuel that is reprocessed and the efficiency of gaseous effluent decontamination equipment. This has resulted in an increase in the ratio of ^{129}I/^{127}I, which has been changing since 1945 due to the release of ^{129}I into environment from nuclear weapon explosions and nuclear facilities. These releases

range from 10^{-8}–10^{-7} in the ambient environment to 10^{-4}–10^{-3} near some nuclear facilities, as measured in thyroid tissues (NCRP 1983). The release of ^{129}I has also resulted in a steady-state inventory of 8.7x10^{26} atoms (31 Ci or 1.8x10^{5} g) of ^{129}I in the oceans (AEC 1966). Slow releases of ^{129}I from radioactive waste dump sites in the North Atlantic and Pacific Oceans, Arctic Ocean, Sea of Japan, and Sea of Okhotsk are also contributing to increased inventories of ^{129}I in ocean waters (Povinec et al. 2000). The concentrations of ^{129}I can vary significantly.

The average content of ^{127}I and ^{129}I in air over the United States varies between 0.6 and 12.1 ppb (Table 6–2). These ^{129}I concentrations did not appear to differ greatly between coastal (e.g., Galveston, Texas) and interior (e.g., Lafayette, Indiana) measurement sites (Moran et al. 1999). The measured ratios of ^{129}I to ^{127}I in air and in precipitation was found to vary, ranging between 2.03 and 27.90 (x10^{-12}) in air and between 755 and 12,390 (x10^{-12}) in precipitation (Moran et al. 1999). These variations in the ratios of ^{129}I to ^{127}I may reflect the distances of the various collection sites from the sources of these isotopes (e.g., distance from coastal regions for ^{127}I and distance from nuclear fuel reprocessing facilities for ^{129}I) (Moran et al. 1999).

The effect of ^{129}I release from nuclear fuel reprocessing facilities on surface water and groundwater has been measured. In 1990–1991, ^{129}I concentrations in the Snake River aquifer at and near the Idaho National Engineering Laboratory range between 0.6 aCi/L (22 nBq/L) and 3.82 pCi/L (0.141 Bq/L), with a mean of 0.81 pCi/L (30 mBq/L), which is a change from 1.30 pCi/L (48.1 mBq/L) measured in 1986. This change reflects a decrease in the amount of ^{129}I disposal and changes in disposal techniques (DOE 1994). Between January 1993 and June 1994, ^{129}I concentrations measured at 29 sites in and around the Savannah River site in surface waters ranged between 0.027 and 3.2 pCi/L (1.0 and 120 mBq/L) and are primarily derived from continued discharges of ^{129}I from the facility (Beals and Hayes 1995).

6.4.3   Sediment and Soil

The natural occurrence of iodine in igneous and sedimentary rock is approximately 0.2–5.8 ppm and it is 5–10 times higher in shales rich in organic matter, in soils, and in coals (NAS 1974). Some references indicate that these estimates may be low, suggesting instead an average soil content for iodine as high as 5.85 ppm (range: 1.5–13.5 ppm) (NAS 1974). Indeed, in one survey of iodine content in soils (Table 6-3), the iodine concentration in common soil types is consistent with the mean of 5.85 ppm (Whitehead 1979). This survey also shows the large variation in iodine concentrations as a function of soil type (Whitehead 1979, Table 6-3). In a study of the iodine concentration of soils in the contiguous

Table 6-2. Concentration of Iodine, Chloride, and $^{129}I/^{127}I$ Ratio in Air and Precipitation as a Function of Location and Collection Time

Location[a]	Air		Rain/Snow			
	I (ppb)	$^{129}I/^{127}I$ (10^{-12})	$^{129}I/^{127}I$ (10^{-12})	^{129}I atoms/L (10^7)	Cl⁻ (ppm)	I/Cl
WL 12/95-2	1.4	12.03	5,756	3.7		
WL 12/95-3	0.9	12.45	8,327	3.7	0.2	0.0045
WL 12/95-4	0.6	13.85	12,390	3.7		
B 11/95	1.2	7.38	2,027	1.1	1.6	0.0008
CS 8/22/96	6.6	10.39	755	2.3		
CS 9/1/96–9/15/96	1.9	4.17	913	2.4	0.3	0.0061
CS 9/15/96–10/15/96	2.5					
CS 10/20/96–10/26/96	2.0				1.2	0.0017
CS 10/26/96–11/24/96	1.7	2.03	893	0.7	1.1	0.0015
CS 11/24/96–11/30/96	3.3				1.6	0.0021
CS 12/21/96–1/16/97	2.9	26.80	3,408	4.5		
CS 1/20/97–1/30/97	2.5	7.60	2,121	2.5		
CS 2/20/97–2/26/97	1.8	5.30	975	0.9		
CS 3/18/97-1	1.4					
CS 3/18/97-2	1.3					
CS 3/18/97-3	0.6					
CS 3/18/97-4	0.8					
CS 3/18/97-5	0.7					
G 1/96–10/96	1.9	9.44	1,735	1.6		
G 11/96	12.1	27.90	1,064	6.0	46.5	0.0003
G 12/6/96–1/6/97	1.7				4.9	0.0003
G 1/7/97–1/16/97	1.2	14.87	3,946	2.3		
Ohio snow 1/14/97	0.7	11.10	9,150	3.0		

[a]WL = West Lafayette, IN; B = Bryan, TX; CS = College Station, TX; G = Galveston, TX

Source: Moran et al. 1999

Table 6-3. Iodine Content in Specific Soil Types

Soil	Concentration of iodine (µg/g dry soil)	
Acid igneous rocks and associated till	10.4	(4.4–15.7)[a]
Till associated with basic igneous rocks	10.9	(3.4–16.3)
Slate, shale, and associated till	9.8	(4.4–27.6)
Sand and sandstone	3.7	(1.7–5.4)
Chalk and limestone	12.3	(7.9–21.8)
Clay	5.2	(2.1–8.9)
River and river terrace, alluvium	3.8	(0.5–7.1)
Marine and estuarine alluvium	19.6	(8.8–36.9)
Peat	46.8	(18.7–46.8)

[a]Average iodine concentration with the range of measurements given in the parentheses

Source: Whitehead 1979

United States, the average iodine concentration in soils was 1.2 µg/g. This average does not differ

between soils measured in the western United States (west of 96th meridian) (1.2 µg/g) or the eastern

United States (1.2 µg/g) (USGS 1984). Iodine concentration in soils has also been measured as a function

of soil depth (Table 6-4), showing minimal variation in iodine concentration to depths of 12–24 inches

(Fuge 1987). The data in Table 6-4 also show that soil concentrations were typically higher than the

concentration of iodine in the underlying bedrock.

The iodine content in sewage sludges ranges between 1.0 and 17.1 µg/g dry weight; these values are

similar to those found in soils, with the mean for iodine in sludges generally being lower. Iodine content

of sludges was not related to size or degree of industrialization of a particular city or town.

Measurements of iodine in sludge indicate that iodine does not partition strongly into sludges (Whitehead

1979). [131]I content in sludge generated from the Oak Ridge municipal waste water plant averages

0.16 nCi/L (5.9 Bq/L or 1.3 fg/L). The background concentration of ^{131}I content in sludge generated at a

municipal sewage treatment in Ann Arbor, Michigan, was reported to be 1.4 pCi/L (52 mBq/L or

0.011 fg/L), but could rise as high as 15 pCi/L (0.55 Bq/L or 0.12 fg/L) (Fenner and Martin 1997). These

concentrations of ^{131}I in sewage sludge are due to the introduction of ^{131}I into city sewer systems from the

excrement from patients who are undergoing treatment therapies that utilize [131]I (Stetar et al. 1993)

The soil content of [129]I has been compared to [127]I and has been found to be generally higher near nuclear

fuel reprocessing facilities than elsewhere. The [129]I/[127]I ratios near facilities range between 10^{-4} and 10^{-3},

whereas more remote locations yield ratios between 10^{-9} and 10^{-8} (Robens and Aumann 1988).

6.4.4   Other Environmental Media

Iodine content of aquatic plants varies, depending on whether they are fresh or salt water. Freshwater

algae contain 10^{-5}% by weight of iodine, whereas marine algae contain 10^{-3}% by weight (NCRP 1983).

For example, concentrations of iodine have been measured in edible marine algae obtained from the St.

Lawrence River, which vary as a function of species. *Enteromorpha* and *Porphyra* had the lowest

average concentrations of iodine of 22.7 and 31.7 µg/g (dry weight) in contrast to *Ascophyllum nodosum*

and *Laminaria longicruris,* which contained the highest average iodine concentrations of 482 and

763 µg/g (dry weight) (Phaneuf et al. 1999).

Epiphytes (plants that have no root systems and acquire their nutrients from the air), such as Spanish

moss in the southern United States, are used to measure long-term exposures of airborne trace elements.

Table 6-4. Iodine Content in Missouri Soils as a Function of Depth and Parent Material

Bedrock	Soil depth (inches)	Iodine concentration (µg/g)
Sandstone	0–5	0.80
	5–8	0.78
	8–12	1.05
	Bedrock	<0.04
Dolomite	0–4	0.89
	4–8	0.85
	8–12	1.13
	12–18	1.08
	18–24	1.28
	Bedrock	<0.04
Alluvium-river valley	0–3	0.91
	3–6	0.65
	6–12	0.72
	12–18	0.91
Limestone (thin soil)	0–3	5.98
Shale	0–6	1.02
	6–12	1.03
	12–18	1.13
	Bedrock	0.37
Granite	0–5	2.90
	5–10	5.00
	10–15	7.21
	Bedrock	<0.04
Glacial material	0–5	0.12
	5–10	0.31
	10–15	0.68
	15–20	0.28
	20–24	0.53

Source: Fuge 1987

6. POTENTIAL FOR HUMAN EXPOSURE

The concentration of ^{129}I in Spanish moss varies between 0.4 and 4.9 ppm and roughly correlates with estimated airborne concentrations of ^{129}I (Moran et al. 1999). In Norway, the iodine in moss averages 3.3 ppm (Schaug et al. 1990).

The average content of iodine in terrestrial plants has been reported to be 0.42 mg/kg worldwide (Yuita 1994a). In some specific examples, iodine content (mg/kg) has been measured at 0.60–2.6 in beet root flesh; 0.1–2.4 in cabbage head; and 0.30–1.1 in corn kernel (Sheppard et al. 1993). The iodine content in hay has been measured at 0.08 μg/g fresh weight feed (Voigt et al. 1988). The distributions of I_2 in plants, such as wheat, can vary over the various parts of the plant. In wheat, iodine concentrations (μg/mg) change between shoots (0.136), roots (0.206), and total plant (0.153). For iodide, the distribution throughout the plant differs as well. In wheat, the iodide concentrations (μg/mg) are 0.645 in shoots, 0.100 in roots, and 0.261 in plant (Voigt et al. 1988).

The ^{129}I content in deer thyroids has been assessed as a function of proximity to a nuclear fuel processing facility. The ^{129}I thyroid concentrations were highest in deer captured near the nuclear fuel reprocessing plant at the Savannah River Site, South Carolina (1–102 Bq/g or 0.03–2.8 nCi/g thyroid in 6.8% of deer) and Oak Ridge, Tennessee (0.01–1.0 Bq/g or 0.3–27 pCi/g thyroid in 38% of deer). However, no thyroids from deer in Florida or in west Tennessee, which are distant from nuclear fuel processing facilities, contained ^{129}I at concentrations above 4 mBq/g or 0.1 pCi/g thyroid (Van Middlesworth 1993).

Iodine measurements in milk and milk products have yielded the following results (expressed as μg/100 g): low fat milk (24), skim milk (21), buttermilk (24), chocolate milk (25), plain lowfat yogurt (33), strawberry lowfat yogurt (17), evaporated milk (37), half-and-half (17), cottage cheese (27), American cheese (49), cheddar cheese (47), chocolate fast-food milkshake (55), chocolate ice cream (47), vanilla ice milk (30), ice cream sandwich (51), and chocolate instant pudding (36) (Pennington 1990a).

Measurements of ^{131}I have been monitored in milk through the Environmental Radiation Ambient Monitoring System (ERAMS) using gamma spectral analysis of milk samples taken from 65 monitoring sites with at least one located in each U.S. state, Puerto Rico, and the Panama Canal Zone. The most recent measurements of ^{131}I in milk samples taken from July–September 1993 through July–September 1997 are below the detection limit at all monitoring sites (see ERD 1993 and ERD 1997 for examples).

6.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

Exposure and uptake of iodine and its radioisotopes can be obtained through several routes including inhalation, transepidermal absorption, dietary intake, use of medications, and medical procedures. The uptake, distribution, and effect of these exposures vary depending on the iodine isotope and the population of interest. These points are discussed below.

Inhalation Exposures. Inhalation exposure of the general population to iodine through inhalation represents an intake pathway. Normal human respiratory exposure to iodine has been estimated to be 5 μg/day from an atmospheric exposure of 0.7 μg/m^3 (FDA 1974). Uptake of ^{129}I through inhalation is a minor pathway for human intake of this radioisotope. It is has been calculated that approximately $4x10^{-5}$ g/year of ^{129}I is taken up by the average individual (USNRC 1979).

Epidermal Exposures. Iodine vapor can penetrate through the skin. Iodine penetration through the epidermis was measured under controlled conditions in which volunteers were exposed to various concentrations of ^{131}I in the air (3.1–350x10^{-10} Ci/L or 11–1,300 Bq/L) (Gorodinskii et al. 1979). Entry of ^{131}I through inhalation was prevented through the use of a specially designed head mask connected to a clean air supply line. Penetration of ^{131}I through the epidermis was monitored by ^{131}I uptake in the thyroid (3.1–303x10^{-10} Ci or 11–1,100 Bq), as measured by a scintillation sensor. K values were calculated to compare the uptake of ^{131}I in the thyroid (A_m) versus the ^{131}I concentration in the air (C), where $K=A_m$ (Ci)/C (Ci/L). The K values varied between 0.7 and 2.9, indicating individual variations in iodine penetration through the skin and uptake of iodine into the thyroid. The results of this study also suggest that the value A_m after a 4-hour exposure to iodine vapor can be approximated using the relationship $A_m=3C$. A comparison of the penetration of iodine through the skin to the penetration of iodine through the lungs in previous work shows that entrance of iodine through the skin is 1–2% of its entrance through the lungs.

Dietary Intake of Iodine. The average daily dietary intake of iodine varies considerably, depending on the diet. Vought and London found individual daily intakes of iodine to vary from 15 to as high as 1,540 μg iodine/day, with mean intakes varying from 64 to 379 μg/day (FDA 1974). The recommended dietary allowance for iodine is 0.150 mg/day for adults and adolescents (FDA 1989b).

Several studies have attempted to describe the daily intake of iodine as a function of diet and age grouping. Hospital diets were measured to have mean iodine intakes of 0.533 mg/day (range 0.274–

0.842 mg/day), 0.677 mg/day (range 0.595–0.713 mg/day), and 0.377 mg/day (range 0.246–
0.506 mg/day) (Caplan et al. 1976; Pittman et al. 1969). One nursing home diet had an intake of
1.531 mg/day (Caplan et al. 1976). The daily iodine intake in children has also been measured: 8–
15 years old (0.450 mg/day) (Trowbridge et al. 1975), 6-month-old infants (0.359 mg/day, range 0.182–
0.576), 2-year-old children (0.435 mg/day, range 0.231–0.728), and 15–20-year-old males (0.527 mg/day,
range 0.319–0.827) (Pennington et al. 1984).

Dietary iodine intakes have been examined among age groups and were measured to be (in mg/day): 6–
11 months old (0.200), 2-year-old children (0.460), 14–16-year-old girls (0.420), 14–16-year-old boys
(0.710), 25–30-year-old women (0.270), 25–30-year-old men (0.520), 60–65-year-old women (0.250),
and 60–65-year-old men (0.340) (Pennington et al. 1986). The average daily iodine intake partitioned by
food category (expressed as μg/day) for men and women in the age group 20–34 years old has also been
reported and is shown in Table 6-5 (FDA 1974)

Drinking Water/Beverages. Human exposures to iodine through drinking water are typically too low to
provide for significant uptake of iodine. Surface waters rarely exceed 5.0 μg iodine/L, except where
waters are polluted with municipal waste stream effluent or urban run-off. In these cases, iodine
concentrations can be as high as 8.7 μg/L (FDA 1974). Some beverages, such as beer and wine, have
iodine contents in the ranges of 43–46 and 8–32 μg/kg, respectively, which could provide a significant
amount of iodine to the diet, depending on the level of daily consumption of these beverages (FDA 1974).

In emergency, camping, or military uses of iodine to disinfect water supplies, iodine concentrations
approach 8–16 mg/L (Zemlyn et al. 1981). Use of elemental iodine in the disinfecting of water, when
improperly used, can lead to acute iodine toxicity. Tetraglycine hydroperoxide is more commonly used at
a concentration of 8 mg/L to provide more accurate and reliable delivery of iodine to disinfect drinking
water. Prolonged (>7 days) use of iodine as a water disinfectant can lead to mild impairment of thyroid
function (Georgitis et al. 1993).

Food Exposures. The human diet is the major source of exposure to iodine for the general human
population. Although marine seafoods typically contain the highest amount of iodine (160–3,200 μg/kg
fresh basis, mean 660±180 μg/kg), they constitute only a small part of the American diet. The largest
sources of iodine in the human diet come from vegetables (320±100 μg/kg), meat products
(260±70 μg/kg), eggs (260±80 μg/kg), and diary products (130±10 μg/kg) (FDA 1974). The level of
iodine in vegetables depends upon the type of plant (e.g., spinach has by far the highest content among

Table 6-5. Estimated Average Iodine Intake for Adults in the United States

Food category	Average daily consumption		Average daily iodine intake	
	Male (g/day)	Female (g/day)	Male (µg/day)	Female (µg/day)
Milk and milk products	397	269	51.6	35.0
Eggs	55	31	14.3	8.1
Meat and meat products	325	192	84.5	49.9
Seafood	14	9	9.2	5.9
Legumes	40	24	No data	No data
Grain and cereal products	12	81	12.2	8.1
Yellow and green vegetables	104	88	33.3	28.2
Other vegetables and fruits	96	56	3.8	2.2
Sugar and sweets	44	35	No data	No data
Beverages (excluding milk)	749	739	3.0	3.0
Estimated salt intake	3.42	3.42	142.0	142.0
Iodine in food as additives	No data	No data	100.1	100.1
Total			454.0	382.5

Source: FDA 1974

the common vegetables) and whether iodine was present in fertilizers. The content of iodine of eggs and milk (milk products) depends on the dietary intake of chickens and lactating cows. Eggs accumulate systemic iodine; however, systemic levels are controlled by the limit that is placed on the content of iodine in feed or water (12.5 mg/kg or liter). Similarly, the content of iodine in milk is dependent upon iodine intake, in addition to the seasonal climatological variables, level of milk production, and fullness of the mammary gland. One source of iodine in the cow's diet is the addition of potassium iodide to feed. However, the iodine content of milk varied between regions and even between individual farms. These variations were found to be caused, in part, by differences in the use of iodized salt blocks, exposures of cows to iodine in disinfectants, and the presence of iodine in sanitizers and veterinary medications (FDA 1974).

Food sources of iodine that have caused adverse effects in humans include water, seaweed, ground beef containing thyroid tissue, foods to which iodine was added as a supplement (iodized water, bread, salt), and milk that contained iodine resulting from feed supplements and iodophor disinfectants (FDA 1989b). For example, iodine in seaweed-based supplements was found to be 0.045–5.0 mg/dose (Norman et al. 1988). The value of 5.0 mg/dose is approximately 30 times higher than the RDA for iodine and 5 times higher than the value of 1.0 mg/day, where acute and chronic toxicities for iodine intake begin to be seen (Pennington 1990a). Outside of these sources of high dietary iodine, the average iodine content in various food groups (expressed as μg/kg wet weight) are: seafoods (660±180), vegetables (320±100), meat products (260±70), eggs (260±80), dairy products (130±10), bread and cereals (100±20), and fruits (40±20) (FDA 1974).

The iodine contained in milk can be readily transferred to milk products, such as cheeses and ice cream. Typically, milk has been shown to contain iodine at concentrations ranging from 0.100 to 0.770 mg/L (Pennington 1988). Cheeses containing levels as high as 425 μg/kg have been reported (FDA 1974). The high concentrations of iodine in milk and in some milk products are thought to be derived from the use of iodophor disinfectants and sanitizers in the practice of dairy farming and the processing of milk. As a topical disinfectant, the concentration of iodine in iodophors is 0.5–1.0% by weight. Iodophors with an iodine concentration of 0.001–0.0025% have been used to disinfect equipment and used in teat dips and udder washings. Teat dipping can increase the iodine content in milk by an average 174 μg/L (range, 55–353 μg/L). However, there is evidence that the major contributor to iodine content in milk is feed supplementation rather than the use of iodophors (FDA 1974).

Processed foods, such as breads, have also been shown to be a source of iodine in the human diet.
Potassium iodate is used as a conditioner of bread dough by some, but not all, major bakeries. When used
as a dough conditioner, a concentration of 1–2 μg iodide/g bread is typically obtained. Iodine content of
fast food, ranging from McDonald's french fries to a filet-of-fish sandwich, varied between 20 and
84 μg/100 g total product, respectively (FDA 1974).

Food additives can also contribute to human iodine intake; table salt contains cuprous iodide and
potassium iodide, alginic acid, and alginate salts that are used as emulsifiers, and stabilizers and
thickeners that contain upwards of 9 mg/kg iodine, but may only constitute an average intake of
1 μg/person/day (FDA 1974). Iodized salt in the United States provides 0.076 mg iodine/g (0.418 mg per
teaspoon) (FDA 1989b).

Distribution of Iodine in Human Tissues. Iodine concentrations in nonthyroid tissues of recently
deceased individuals obtained from a healthy Chinese population have been assessed using neutron
activation analysis techniques (Hou et al. 1997b). Typical intake of iodine in the Chinese diet averages
between 94 and 169 μg/person/day (Hou et al. 1997a). The concentrations of iodine in five tissues, plus
hair, averaged over 9–11 individuals (and expressed as ng/g wet weight tissue±1 SD) were: heart
(46.6±14.9), liver (170±34), spleen (26±8.6), lung (33.3±10.6), muscle (23.5±14.3), and hair (927±528)
(Hou et al. 1997b).

Exposures Through Medications/Medical Uses of Iodine. Human exposures to iodine may come from
medications and vitamin supplements containing iodine in varying amounts. A survey of various
pharmaceuticals found that of those tested, eight contained between 0.251 and 0.375 mg iodine per dose,
with one containing 1.447 mg I/dose (Vought et al. 1972). The variation of iodine content could be
attributed to the use of erythrosine (2,4,5,7-tetraiodofluorescein) as a red coloration (FDA 1974).
Erythrosine can be metabolized in the digestive tract to liberate iodide, although the bioavailability of
iodine from erythrosine may be only 2–5% (FDA 1989b). Some medications directly contain added
potassium iodide or organic iodine compounds. For example, Lugol's solution that is used to treat
thyrotoxicosis is a 5% solution of iodine solubilized in 10% potassium iodide. Other iodine-containing
drugs (most commonly potassium iodide solutions) have been prescribed for their purported expectorant
for action in asthma, bronchitis, cystic fibrosis, and chronic pulmonary obstructive disease; also,
amiodarone is prescribed for heart arrhythmias (FDA 1989b). Topical application of iodine-containing
medications and dietary supplements can increase iodine in breast milk in lactating women; use of

povidone iodine vaginal gel (50 mg/day for 6 days) increased iodine concentration in breast milk by 3–
4 times (FDA 1989b).

Large exposures to iodine can be experienced during certain medical procedures (e.g., through the use of
iodinated compounds as contrast agents in radiological imaging procedures). Iodine in oil is used in
bronchograms, lymphangiograms, and for myelograms, and is excreted slowly, thus predisposing an
individual to imbalances in iodine homoeostasis.

Radioiodine -^{131}I. The greatest periods of exposure to ^{131}I (and other radioiodine isotopes derived from
nuclear fission) were during active nuclear testing in the years 1951–1958 and 1961–1962, the large
quantities of fission products released from nuclear accidents such as Three Mile Island and Chernobyl,
and the nuclear processing and waste facilities (e.g., Hanford, Washington; Aiken, South Carolina; Idaho
Falls, Idaho). Human uptake of ^{131}I from environmental sources is largely through ingestion of
contaminated food, with a smaller proportion obtained through inhalation (Straub et al. 1966; Wehmann
1963). The distribution of ^{131}I in food depends upon the time the isotope is produced, its presence in the
environment, and the degree of contamination. Some potential dietary sources of ^{131}I include marine
animals and plants, milk, and leafy vegetables (Straub et al. 1966).

The largest source of ^{131}I in the human diet is cow's milk. Approximately 70% of the ^{131}I that is
consumed by a cow is absorbed into the thyroid, with about 1% found in milk (milk-to-plasma ratio of ^{131}I
is 2:3) (Bustad et al. 1964; Lengemann and Comar 1964; Straub et al. 1966). The transfer of iodine is bi-
directional, and the iodine appears to freely diffuse between mammary gland and plasma (Miller and
Swanson 1963; Straub et al. 1966). ^{131}I exists in both the free inorganic form, as iodide, or bound to
protein in milk. It has been determined that in cow's milk, 82–91% of the ^{131}I is in the free inorganic
form, with 4.7–13% bound to protein and <0.1% associated with fat (Glassock 1954; Straub et al. 1966).

The occurrence and concentration of ^{131}I in milk is highly variable, depending on the locale and daily
variations within a specific locale (Pennington 1990a). Due to meteorological conditions, a large
proportion of ^{131}I from fallout is deposited on the ground through dry deposition processes, with a lesser
amount deposited through wet deposition processes (i.e., precipitation) (Straub et al. 1966). The highest
concentrations of ^{131}I in milk were observed shortly after atmospheric tests of nuclear weapons and
accidental releases from nuclear reactors or fuel reprocessing facilities (Anderson et al. 1996a; Black et
al. 1976; Cohn and Gusmano 1963; Kirchner 1994; Martin and Turner 1964; Tracy et al. 1989; Tubiana
1982; Voigt et al. 1989). The source of ^{131}I in cow's milk is derived mainly from the dry and wet

deposition (to a lesser extent uptake into plants from the soil) of ^{131}I onto grasses and other plants that are then consumed by dairy cows. A concentration of 1 µCi ^{131}I/kg (37 kBq ^{131}I/kg) of ^{131}I in pasture grass yields a ^{131}I concentration of 0.07 µCi ^{131}I/L (3 kBq ^{131}I/L) of milk (Voigt et al. 1989).

In cases of exposure to ^{131}I, the absorbed dose has been estimated in various tissues and whole body for the ingestion of 1 mCi (40 MBq) of ^{131}I. The values for the absorbed doses (expressed in cGy and based on the assumption of a 25% uptake of ingested ^{131}I into the thyroid) are: 0.48 (liver), 0.14 (ovary), 0.26 (bone marrow), 1.4 (gastric mucosa), 0.088 (testis), and 1,300 (thyroid), with a mean whole body dose of 0.71 cGy (Tubiana 1982).

The levels of ^{131}I in domestic and imported foods measured between 1987 and 1992 were found to be below the detection limit of the gamma-ray spectrometry method (<2 Bq/kg or <5 pCi/kg) (Cunningham et al. 1994). Detection and quantitation of ^{129}I is difficult due to the low energy of the beta (maximum energy=0.15 MeV) and gamma rays (0.04 MeV) that are emitted from this isotope. Concentration of ^{129}I in thyroid tissues increases the ability to detect this isotope, but a further limitation results from the low specific activity of the radioisotope (0.17 mCi/g or 6.3 MBq/g) (NCRP 1983). However, due to the steady-state levels of ^{129}I in the environment, there is a continual exposure of the general human population to this radioisotope through inhalation and intake through the diet. It has been estimated (AEC 1974) that a dose of approximately 0.2–0.5 mrem/year is delivered to the thyroid of an adult from ^{129}I, depending on diet. These estimates include the dose received through the inhalation of ^{129}I. For an infant, the doses to the thyroid from ^{129}I intake can vary between 0.15 and 0.4 mrem/year, depending on diet.

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 3.8 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).

Children appear to be more susceptible to the development of thyroid cancers from the irradiation of thyroid by [131]I. Irradiation of the thyroid from a 1–2 Gy dose in Japanese A-bomb survivors and a 3.3 Gy dose in Marshall Islanders exposed to fallout results in a high incidence (factor of 2) of thyroid cancers in children under the age of 10 compared the exposed adult population (Tubiana 1982).

Of the iodine radioisotopes, [129]I poses the least risk to children with respect to thyroid irradiation. Even at a high thyroid content of 1 mCi (40 MBq) [129]I in 1 g of stable iodine, the dose to the thyroid in a 6-month-old infant would be 0.9 nGy/year as compared to a dose of 7.2 nGy/year in an adult thyroid. In comparison, ^{131}I can deliver much higher doses to the thyroid at lower environmental concentrations than those observed for [129]I, due to the higher specific activity of [131]I (1.24×10^5 Ci/g or 457 TBq/g; beta particle energies of 0.334 MeV [7.3%] and 0.606 MeV [89.9%]) versus [129]I (177 µCi/g or 6.55 MBq/g; beta particle energy of 0.154 MeV) (Chu et al. 1999; Robkin and Shleien 1995). For example, the annual consumption of milk containing ^{131}I at a concentration of 1–20 nCi/L (37–740 Bq/L) would result in a dose to the thyroid in children of between 0.005 and 0.1 cGy/year (Tubiana 1982). In comparison, similar concentrations of [129]I in milk would yield a thyroid dose of between 0.014 and 0.28 pGy/year, based on the specific activities and beta energies listed above. Breast milk is also a source of ^{131}I uptake in children. For example, concentrations of ^{131}I in breast milk measured in women exposed to nuclear weapons fallout ranged between 0.050 and 0.100 nCi/L (1.9 and 3.7 Bq/L), when the mean body burden of ^{131}I in these women was 0.060 nCi (2.2 Bq) (Cohn and Gusmano 1963).

Children (both 1 and 10 year olds) appear to have a similar fractional uptake of iodine in the thyroid (i.e., approximately 31%) of that found in adults. For newborns, however, the fractional uptake is approximately 70% at 2 days after birth, but quickly declines to values accepted for an adult by day 5. After the first few weeks, uptake changes very little with age. The estimated dietary intake of iodine in 1 year olds is 151 µg/day, and for 10 year olds, it is 184 µg/day. The percent turnover rates of iodine in the thyroid does change with age (expressed as d^{-1}) are: 0–4 years old (3.4 ± 0.5), 4–8 years old (2.1 ± 0.5), and 8–12 years old (0.84 ± 0.36); this corresponds to 'apparent' half-lives of 20, 33, and 83 days, respectively. Iodine concentration in thyroid increases with age (expressed as µg/g): 1 year old (95), 2 years old (130), 4 years old (180), 10 years old (260), 16 years old (320), and adult (400) (Stather and Greenhalgh 1983).

In utero exposures of a human fetus to iodine radioisotopes with high specific activity (e.g., ^{131}I) have been assessed based on both the maternal intake of iodine radioisotopes and exposure to external radiation generated by these isotopes in the immediate environment. Iodine and its radioisotopes freely diffuse across the placenta and, as such, their levels within a fetus and the amniotic fluid will depend greatly on the concentration of iodine within the mother (Bašič et al. 1988; Dyer and Brill 1972; Etling et al. 1979; von Zallinger and Tempel 1998). Before 11 weeks of gestation, the thyroid is still undeveloped and does not actively take up iodine (Dyer and Brill 1972; von Zallinger and Tempel 1998). For example, the percent of iodine that is taken up by the thyroid in comparison to that contained in the total fetal tissues is quite low at a gestation age of 9–11 weeks (0.0002%), but increases after 11 weeks where the percentage becomes 0.001% for gestation ages between 13 and 15 weeks, and increases further to 0.002% between gestation ages of 16 and 22 weeks (Dyer and Brill 1972). A difference in placental transfer of iodine between the mother and fetus is also noted as a function of gestation time. The percentage of ^{131}I activity found in the fetus compared to the total activity within the mother at 11 weeks was 0.23%, but increased to 2.96% at 22 weeks due to the fact that the concentration of iodine in the fetal thyroid typically exceeds that of the maternal thyroid by 3–10 times (von Zallinger and Tempel 1998). Increases in the concentration of iodine in the fetus and amniotic fluid (6- to 90-fold increases) were observed in women exposed to topical iodine-containing medications (e.g., vaginal therapy with iodinated polyvinyl-pyrrolidine) or increased iodine intake in their diets (Etling et al. 1979). However, the uptake of iodine (measured by ^{131}I uptake) into the fetal thyroid (1.08 pCi/g or 0.0400 Bq/g) at a gestation time of 22 weeks is not significantly different from what is observed for the maternal thyroid (0.82 pCi/g or 0.030 Bq/g) (Beierwaltes et al. 1963).

Emission of ionizing radiation from iodine radioisotopes can also pose an exposure risk to the human fetus and the mother in occupations where the mother comes in contact with these isotopes. For example, fetal doses in imaging staff performing a whole body scan and/or therapy procedures for thyroid cancer patients using ^{131}I ranged between 6.7 and 9.0 μSv (Mountford and Steele 1995). Thus, restrictions on the exposure of pregnant women are 1.3 mSv to the maternal abdominal surface (corresponding to a 1.0 mSv dose to the fetus) (Mountford and Steele 1995).

6.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

Occupational Exposures (Medical Personnel, Laboratory Personnel, Personnel at Nuclear Power/Fabrication/Storage Facilities). Occupational exposures to airborne iodine can occur when iodine is used in the regular functions of the workplace. OSHA has set a limit for airborne iodine

concentrations in the workplace of 0.1 ppm. Exposures of workers to concentrations at or in excess of the OSHA limit can often occur. For example, at a plant that processes photo-polymer plates, most workers were exposed to an average concentration of iodine of 0.005 ppm, but were exposed to concentrations of 0.122–0.146 ppm when they were in the immediate proximity of the iodine holding tank or iodine applicator (Kim et al. 1981).

The USNRC has set Annual Limits of Intake (ALIs) for inhalation exposures to radioiodine in the workplace as specified in USNRC Regulations (10 CFR) (USNRC 2002). The ALIs are based on the annual intake of a particular radioisotope that would result in an effective dose equivalent of 5 mrems to an organ or tissue; in the case of radioiodine, the thyroid. For inhalation exposures, the ALI is derived from Derived Air Concentrations (DACs) that represent the concentration of radioiodine at which a "reference man" working 2,000 hours per year under light working conditions (inhalation rate of 1.2 m^3/hour) results in the intake of one ALI. The current ALIs for inhalation exposure based on the thyroid as the target organ are: ^{123}I, 6,000 µCi (DAC=3x10^{-6} µCi/mL); ^{125}I, 60 µCi (DAC=3x10^{-8} µCi/mL); and ^{131}I, 50 µCi (DAC=3x10^{-8} µCi/mL).

Workers in the nuclear industry, especially in nuclear fuel reprocessing facilities, have the potential for chronic thyroid burdens of ^{131}I in addition to acute exposures from accidental releases. For example, in Indian workers, the doses from chronic ^{131}I exposures were found to be as high as 47.4–68.1 rem for thyroid and 0.024–0.047 rem for whole body (Raghavendran et al. 1978). In an acute exposure to ^{131}I in a steam leak at a nuclear power plant, the mean thyroid burden of ^{131}I in the exposed workers was 1.32 µCi (48.8 kBq) on the third day after the exposure; the ^{131}I burden decreased exponentially, falling below 0.027 µCi (1.0 kBq) on the 38th day (Bhat et al. 1973).

Internal contamination of medical personnel by ^{131}I can be a problem, especially under conditions where the release of iodine as a vapor can occur (Eadie et al. 1980; Luckett and Stotler 1980). Thyroid burdens of ^{131}I in medical personnel can typically average around 2,400 pCi (ranges of 35–18,131 pCi or 1.3–671.52 Bq) (Blum and Liuzzi 1967). Personnel working with ^{131}I could potentially receive up to 5 nCi (200 Bq) per mCi (40 Bq) handled. This means that persons handling therapeutic doses of ^{131}I could have activities of 0.1–1.0 µCi (- 4,000–40,000 Bq) in their thyroids (Tubiana 1982). For the application of ^{131}I in nuclear medicine, it has been shown that the radiochemists and their support staff have yearly thyroid ^{131}I burdens of between 0.5–200 nCi (20–7,000 Bq) and 0.03–1.5 nCi (1–56 Bq), respectively (Jönsson and Mattsson 1998).

External exposures of medical personnel to radiation emitted from radioiodine have been assessed for both diagnostic and therapeutic applications of ^{131}I and [125]I. The dose rate per unit activity (μSv/h @MBq) of ^{131}I has been determined for thyrotoxicosis, thyroid ablation, and thyroid cancer therapies as a function of distance from the patient. The dose rate as a function of distance is approximately the same for all three therapy regimens; 1.43 μSv/h @MBq at 0.1 meter, 0.18 μSv/h @MBq at 0.5 meter, and 0.07 μSv/h @MBq at 1.0 meter (Mountford and O'Doherty 1999). Surgical implants of ^{125}I seeds in prostate brachytherapy have the potential for radiation exposures to the radiotherapist. However, in most instances when the implant procedure is performed properly, the dose rate is <1 μGy/mCi-hour (Liu and Edwards 1979).

Laboratory workers using ^{125}I are at risk for exposures to gamma- and x-rays to the hand. In a typical situation where 20 MBq (5 mCi) of ^{125}I from an unshielded source is used weekly (2 hours/week) throughout a year, a worker would receive a dose of approximately 225 mSv (22.5 rem), 3/10 of the recommended dose equivalent limit to the hands (de Groot 1979). Uptake of ^{125}I into the thyroid has also been shown to occur due to airborne radioiodine released from solutions or spills (Bogdanove and Strash 1975; Dunn and Dunscombe 1981; Krzesniak et al. 1979; Kwok and Hilditch 1982). Activity levels of 0.013–0.024 μCi (480–890 Bq) and 0.056–0.56 μCi (2,100–21,000 Bq) have been measured in the thyroid of a laboratory worker working with 1 and 5 mCi (40 and 200 MBq) of ^{125}I in the day's activities, respectively (Kivinitty et al. 1984). In a more general survey, it was found that 8% of laboratory technicians working with ^{125}I labeled materials had thyroid burdens of ^{125}I within 9–90 nCi (300–3,000 Bq). However, 33% of those individuals involved in the direct iodination of biomaterials and compounds with ^{125}I had thyroid burdens of 9–90 nCi (300–3,000 Bq) (Pomroy 1979).

Patients Undergoing Medical Treatment Involving Use of Iodinated Compounds. Patients undergoing treatment for hyperthyroidism and thyroid carcinoma typically receive between 10 and 150 mCi (370 and 5,500 MBq) of [131]I (Beierwaltes 1979). In addition to the radiation dose received by the thyroid from these loadings of ^{131}I, other tissue sites in the patient also receive a radiation dose, albeit a smaller dose. This is especially important when considering the impact that the radiation emitted from ^{131}I can have on bone marrow in a patient undergoing ^{131}I therapy to treat thyroid carcinomas. During these ^{131}I therapies, the bone marrow can receive a dose of 1–5 Gy (Tubiana 1982). This has been shown to lead to a 1% incidence of leukemia in these patients (Tubiana 1982).

There is also an exposure risk of a patient's immediate family to both the radiation and elemental ^{131}I that is emitted from the patient (Barrington et al. 1999). Patients are allowed to return home after the activity

of ^{131}I within them falls below 30 mCi (1.1 GBq). Due to differences in the excretion rate of radioiodine from patients, it is recommended that both biological clearance and physical decay be used in calculating the confinement time of a patient (Kaurin et al. 2000; North et al. 2001). However, the level of ^{131}I activity of 30 mCi (1.1 GBq) in a patient can produce an exposure rate to ionizing radiation (emitted in the decay of ^{131}I) of approximately 10 µGy/hour at a distance of 1 meter. Thus, family members are also at risk to exposures to ^{131}I that is emitted from the patients. The dose that a typical family member receives from a patient at home ranges between 0.17 and 126 µGy/day, as compared to the natural radiation background of 0.35 µGy/day (Jacobson et al. 1978). This can result in some family members exceeding the maximum allowable dose of mSv (1 mrem) per year. In another study, it was found that as many as 11% of children exposed to patients undergoing ^{131}I therapy exceeded the 1 mSv limit (Barrington et al. 1999). Activities of ^{131}I within the thyroids of family members of patients undergoing ^{131}I therapy were found to range from the detection limit of the measurement of 92–110,000 pCi, resulting in a dose of 4–1,330 mrem to the thyroid. Of special concern is the fact that the ^{131}I activity was highest in children (Jacobson et al. 1978).

A growing number of patients who are undergoing treatment for cancer are using alternative medicines and nutritional supplements (Cassileth 1999). It is believed that these alternative medicines and supplements will help to prevent the onset of a tumor, alleviate specific symptoms that are experienced as a consequence of their disease or treatment, or aid in the eradication of the tumor. The self administration of some of these alternative medicines and nutritional supplements can result in the intentional (e.g., elevated iodine intake to prevent breast cancer) or unintentional (e.g., when iodine is a natural component of a specific alternative medicine) elevation of the daily intake of iodine, especially when patients consume alternative medicines that contain salt water plants, such as kelp, or those individuals who take megavitamins or participate in orthomolecular therapy (Cassileth 1999). The amount of iodine intake will vary depending on the specific content of iodine in the supplement and the dosage, which could result in iodine intakes that approach toxic levels (e.g., >6 g/day). For most cancers, it is unclear what benefit increased iodine will have for the prognosis of the disease or how it can alter the incidence of a particular cancer (Cann et al. 2000; Cassileth 1999; Eskin 1970). However, there is evidence to suggest that elevated iodine intake, especially for populations where ambient iodine concentrations are low, can help to decrease the incidence of breast cancer and, in some cases, help to interfere with breast tumorogenesis (Cann et al. 2000; Eskin 1970).

Diseases/Predisposition to Iodine Toxicities. An increase in the availability of dietary iodine for a population may also cause difficulty in controlling Graves' disease with antithyroid drugs, decrease the

remission rates for those on antithyroid medication, and increase the dose of radioiodine required to induce euthyroidism (FDA 1989b). In the general population, between 0.2 and 33.3% of individuals develop goiter in response to excess iodine consumption, whereas an increase in the incidence of sensitivity or acute reactions was observed in <30% of individuals in the general population (FDA 1989b).

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 iodine and its radioisotopes is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (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 iodine and its radioisotopes.

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.   Adequate pertinent data on the physical and chemical properties of iodine and its radioisotopes and compounds are available in the literature.

Production, Import/Export, Use, Release, and Disposal.   Since iodine is not covered under Superfund Amendments and Reauthorization Acts (SARA), Title III, manufacturers and users are not required to report releases to the EPA. There is a lack of data on the release and disposal of iodine. There is a relatively good database on the release of radioiodine (Beals and Hayes 1995; DOE 1990, 1994; NCRP 1983; Patton and Cooper 1993), but only limited information is available on disposal and inventories of radioiodine in the disposal sites (DOE 1994).

Environmental Fate.   The major source of iodine on terrestrial surfaces originates from the volatilization of iodine from the ocean surface. Adequate information is available pertaining to the chemical species and reactions that take place at and above the ocean surface that are responsible for the production of volatile forms of iodine (Cox et al. 1999; Filistovic and Nedveckait 1998; Vogt et al. 1999; Whitehead 1984). Further work is needed to examine the organisms and microbial metabolic processes that are responsible to the formation of alkyl iodides, as well as the exact contribution of alkyl iodides, molecular iodine, and spray to the introduction of iodine into the atmosphere. There is a good body of literature on the photochemical reactions of iodine, both in the gaseous phase and in/on particulates or water droplets (Cox et al. 1999; Filistovic and Nedveckait 1998; Moran et al. 1999; Vogt et al. 1999; Whitehead 1984). The factors that are responsible for the retention of iodine in soil have also been examined extensively (Fawcett and Kirkwood 1953; Jirousek and Pritchard 1971; Sheppard et al. 1995; Whitehead 1984). However, more work is needed to characterize the interactions of iodine with organic components, especially with respect to the mechanisms of the binding and release of iodine from these organic components. The environmental fate of ^{129}I and ^{131}I has been examined extensively (AEC 1974; DOE 1978a, 1986; USNRC 1979, 1981).

Bioavailability from Environmental Media.   Adequate pertinent data for intake of iodine and radioiodine from inhalation, drinking water, and food intake are available (DOE 1993; NCRP 1983; USNRC 1979; Whitehead 1984).

Food Chain Bioaccumulation.   Concentrations of iodine in freshwater and marine fish have been determined (Poston 1986). Concentrations of iodine in aquatic plants have also been ascertained (NCRP 1983). Although aquatic plants and fish concentrate iodine in their tissues, there is little evidence for bioaccumulation of iodine in the food chain. Iodine and radioiodine concentrations have been measured in foods, especially in the context of milk and the transfer of radioiodine through the soil-plant-cow-milk pathway (AEC 1974; Kirchner 1994; Tracy et al. 1989; Voigt et al. 1988, 1989). Although some information is available, more information is needed on the uptake of iodine from the soil into plants (Burte et al. 1991; Moiseyev et al. 1984; Whitehead 1984).

Exposure Levels in Environmental Media.   Adequate pertinent data are available for current exposure of iodine in air, rainwater, surface water, groundwater, and soil (FDA 1974; Moran et al. 1999; USNRC 1979; Whitehead 1984; Yuita 1994a).

Exposure Levels in Humans.   A good database exists for exposure levels of the general population to iodine and its radioisotopes in various food types and drinking water (Allegrini et al. 1983; Bruhn et al. 1983; Dellavalle and Barbano 1984; Kidd et al. 1974; Pennington et al. 1986), including exposure levels in milk (Pennington 1988). Information for the average daily intakes of iodine based on diet and age groupings is available (Caplan et al. 1976; Pennington et al. 1984, 1986). Information on occupational exposures is available, especially for exposure of medical personnel to ^{131}I and laboratory workers to ^{125}I (Blum and Liuzzi 1967; Bogdanove and Strash 1975; de Groot 1979; Dunn and Dunscombe 1981; Krzesniak et al. 1979; Kwok and Hilditch 1982; Mountford and O'Doherty 1999; Pomroy 1979; Tubiana 1982). However, exposure data are currently not available for individuals who come in contact with, work in, or live in the vicinity of, clandestine methamphetamine production laboratories. This information is especially needed due to the potential for acute and chronic exposures to iodine. Data exist for the distribution of iodine in human and fetal tissues, but more information is needed (Dyer and Brill 1972; Hou et al. 1997b; von Zallinger and Tempel 1998).

Exposures of Children.   A good database exists for exposure levels of children to iodine and its radioisotopes in various environmental exposure pathways, including food types, drinking water, and especially milk and milk products (Cohn and Gusmano 1963; FDA 1974, 1989; Soldat 1976; Stather and Greenhalgh 1983; Tubiana 1982). Information for the average daily intakes of iodine for children based on age groupings is available (Pennington et al. 1984, 1986; Trowbridge et al. 1975). Information on *in utero* exposures to iodine and its radioisotopes is available (Bašič et al. 1988; Beierwaltes et al. 1963; Dyer and Brill 1972; Etling et al. 1979; Mountford and Steele 1995; von Zallinger and Tempel 1998). There is also some information on the exposure of children to ^{131}I, and the radiation that it emits, that occurs when children are in contact with, or in the vicinity of, individuals undergoing ^{131}I treatment (Barrington et al. 1999; Jacobson et al. 1978). However, more information is needed to adequately assess the risk of children to this exposure. Also, there will be a need to develop biomarkers to assess the low level exposures of children to ^{129}I. Improvements in analytical methods have provided an ability to detect the low concentrations of ^{129}I in tissues and, thus, given us an opportunity to reliably assess low level exposures of ^{129}I in children and adults. Yet, the development of biomarkers, such as the identification of DNA mutations that would be specifically formed as a consequence of ^{129}I exposure in a cell to monitor the possible biological effects of these low level exposures, have been lacking.

Child health data needs relating to susceptibility are discussed in Section 3.13.2 Identification of Data Needs: Children's Susceptibility.

Exposure Registries.   No exposure registries for iodine were located. This substance is not currently one of the compounds for which a subregistry has been established in the National Exposure Registry. Iodine will be considered in the future when chemical selection is made for subregistries 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

Two studies are currently underway (FEDRIP 2000) to study the interaction of iodine with differing soil components and the effect of iodine intake on the underlying mechanisms contributing to autoimmune thyroiditis. In the first study, Dr. R.L. Jones, at the Department of Natural Resources and Environmental Sciences, University of Illinois, Urbana, Illinois, is conducting a study of iodine in Illinois soils. The objective of this work is to determine the concentrations of iodine in a group of selected surface soils so that estimates of iodine concentrations can be made for major soil areas in Illinois. Analysis of soils as a function of depth and soil type will identify proportions of iodine in organic matter, and iron and aluminum fractions with the objective of identify whether differences occur between soils because of differences in soil development and genesis.

In another study, conducted by Dr. Carol L. Burek at Johns Hopkins University, Baltimore, Maryland, work is underway to determine whether increased intake of iodine contributes to an increase in the incidence of autoimmune thyroiditis (AT). The researchers intend to show that increases in iodine intake lead to an increased level of a highly iodinated form of thyroglobulin protein. It is thought that this highly iodinated form of thyroglobulin can act as an auto-immunogen and may be responsible for the T-cell mediated auto-immune response that is targeted against the thyroid gland. Using the NOD mouse model, the researchers will examine whether increased doses of iodine lead to an increase in the incidence of AT in these mice. Once this is established, the researchers will examine whether this increased incidence in AT is accompanied by an increase in the levels of highly iodinated thyroglobulin protein and increased activity of T-cells against this potential auto-immunogen.