6. POTENTIAL FOR HUMAN EXPOSURE

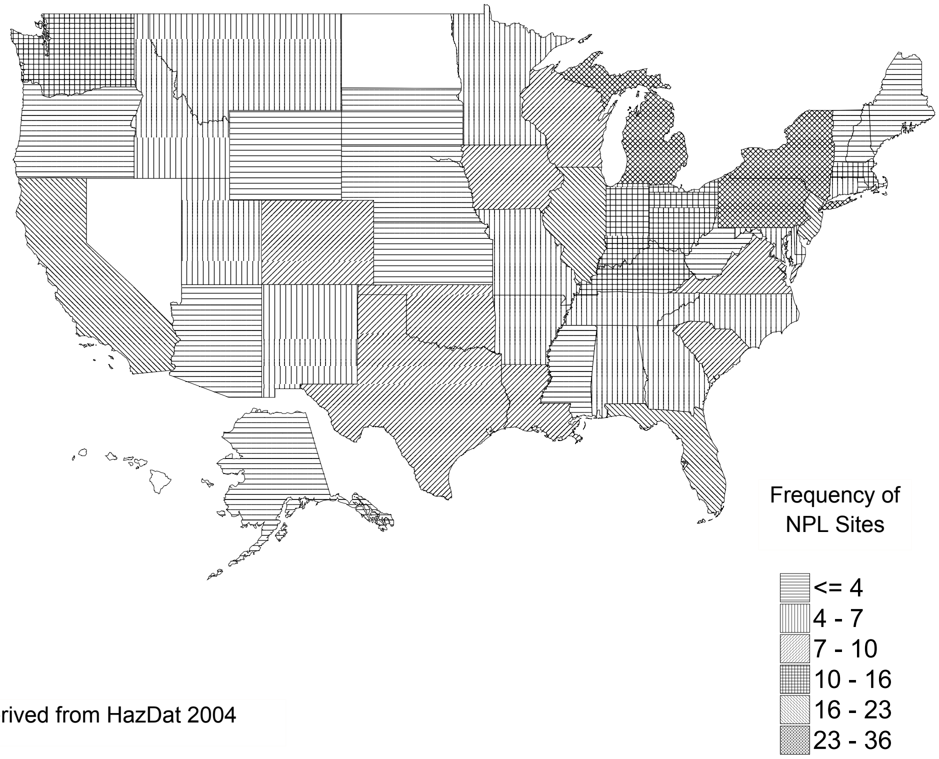
6.1 OVERVIEW

Stable cobalt has been identified in at least 426 of the 1,636 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 2004). Radioactive cobalt as ^{60}Co has been identified in at least 13 of the 1,636 hazardous waste sites that have been proposed for inclusion on the EPA NPL (HazDat 2004). However, the number of sites evaluated for stable cobalt and ^{60}Co is not known. The frequency of these sites can be seen in Figures 6-1 and 6-2, respectively. Of the cobalt sites, 421 are located within the United States, 1 is located in Guam (not shown), 3 are located in the Commonwealth of Puerto Rico (not shown), and 1 is located in the Virgin Islands (not shown). All of the sites at which ^{60}Co has been identified are located within the United States.

Cobalt occurs naturally in the earth's crust, and therefore, in soil. Low levels of cobalt also occur naturally in seawater and in some surface water and groundwater (Smith and Carson 1981). However, elevated levels of cobalt in soil and water may result from anthropogenic activities such as the mining and processing of cobalt-bearing ores, the application of cobalt-containing sludge or phosphate fertilizers to soil, the disposal of cobalt-containing wastes, and atmospheric deposition from activities such as the burning of fossil fuels and smelting and refining of metals (Smith and Carson 1981). Cobalt is released into the atmosphere from both anthropogenic and natural sources. However, emissions from natural sources are estimated to slightly exceed those from manufactured sources. Natural sources include windblown soil, seawater spray, volcanic eruptions, and forest fires. Primary anthropogenic sources include fossil fuel and waste combustion, vehicular and aircraft exhausts, processing of cobalt and cobalt-containing alloys, copper and nickel smelting and refining, and the manufacture and use of cobalt chemicals and fertilizers derived from phosphate rocks (Barceloux 1999; Lantzy and Mackenzie 1979; Nriagu 1989; Smith and Carson 1981). ^{60}Co and ^{58}Co, both radioactive forms of cobalt, may be released to the environment as a result of nuclear research and development, nuclear accidents, operation of nuclear power plants, and radioactive waste dumping in the sea or in radioactive waste landfills.

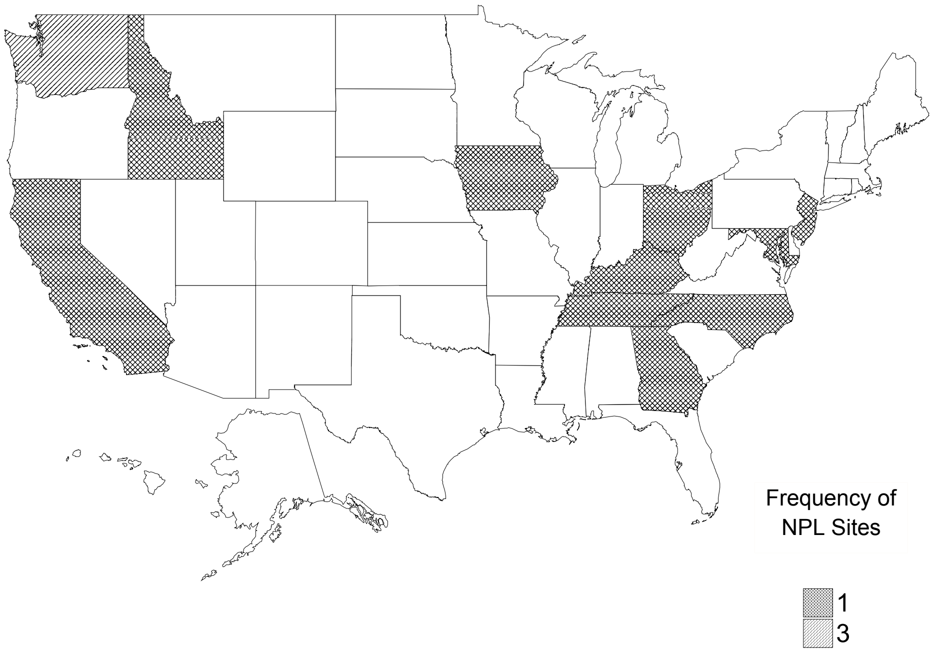
Cobalt compounds are nonvolatile and cobalt will be emitted to the atmosphere only in particulate form. Their transport in air depends on their form, particle size and density, and meteorological conditions. Cobalt so released will return to land or surface water as wet or dry deposition. Coarse particles, those

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



Derived from HazDat 2004

Figure 6-2. Frequency of NPL Sites with ^{60}Cobalt Contamination



Derived from HazDat 2004

with aerodynamic diameters >2 μm (such as those obtained during ore processing), may deposit within 10 km from the point of emission; finer particles (such as is obtained from thermal processes) may travel longer distances. It is generally assumed that anthropogenic cobalt originating from combustion sources exists primarily as the oxide; arsenides or sulfides may be released during mining and ore processing (Schroeder et al. 1987). Frequently, sediment and soil are the ultimate sinks for cobalt; however, this process is dynamic, and cobalt can be released into the water depending upon conditions. Soluble cobalt released into waterways will sorb to particles and may settle into the sediment or be sorbed directly by sediment. It may precipitate out as carbonates and hydroxides or with mineral oxides. It may also sorb to or complex with humic acid substances in the water. These processes are sensitive to environmental factors such as pH and the proportion of dissolved cobalt will be higher at low pH. In the case of ^{60}Co released into an experimental lake in northwestern Ontario, cobalt's half-life in the water column was 11 days; 5% of added ^{60}Co remained in the water after 100 days (Bird et al. 1998a). Cobalt can also be transported in dissolved form or as suspended sediment by rivers to lakes and the sea or by ocean currents. The proportion of cobalt transported in each form is highly variable (Smith and Carson 1981). In deep sediment where water is anoxic and hydrogen sulfide is present, some mobilization of cobalt from sediment may occur, probably due to the formation of bisulfides and polysulfides (Bargagli 2000; Brügmann 1988; Finney and Huh 1989; Glooschenko et al. 1981; Knauer et al. 1982; Nriagu and Coker 1980; Shine et al. 1995; Smith and Carson 1981; Szefer et al. 1996; Windom et al. 1989). Cobalt adsorbs rapidly and strongly to soil and sediment in which it is retained by metal oxides, crystalline minerals, and natural organic matter. The mobility of cobalt sediment depends on the nature of the soil or sediment; it increases with decreasing pH and redox potential (Eh) and in the presence of chelating/complexing agents (Brooks et al. 1998; Buchter et al. 1989; King 1988b; McLaren et al. 1986; Schnitzer 1969; Smith and Carson 1981; Swanson 1984; Yashuda et al. 1995).

While cobalt may be taken up from soil by plants, the translocation of cobalt from roots to above-ground parts of plants is not significant in most soils; the transfer coefficient (concentration in plant/concentration in soil) for cobalt is generally 0.01–0.3 (Mascanzoni 1989; Mermut et al. 1996, Smith and Carson 1981). However, in highly acidic soils (pH as low as 3.3) and in some higher plants (plants excluding algae), significantly higher transfer has been observed (Boikat et al. 1985; Francis et al. 1985; Jenkins 1980; Kloke et al. 1984; Mejstrik and Svacha 1988; Palko and Yli-Hala 1988; Tolle et al.1983; Watabe et al. 1984). The bioaccumulation factors (dry weight basis) for cobalt in marine fish and freshwater fish are ~100–4,000 and <10–1,000, respectively; accumulation is largely in the viscera and on the skin, as opposed to the edible parts of the fish. Cobalt does not biomagnify up the food chain (Barceloux 1999; Evans et al. 1988; Freitas et al. 1988; Smith and Carson 1981).

Atmospheric cobalt is associated with particulate matter. Mean cobalt levels in air at unpolluted sites are generally <1–2 ng/m^3. In several open-ocean environments, geometric mean concentrations ranged from 0.0004 to 0.08 ng/m^3 (Chester et al. 1991). However, in source areas, cobalt levels may exceed 10 ng/m^3; the highest average cobalt concentration recorded was 48 ng/m^3 at the site of a nickel refinery in Wales (Hamilton 1994; Smith and Carson 1981). By comparison, the Occupational Safety and Health Administration (OSHA) limit for airborne stable cobalt is 100,000 ng/m^3. While ^{60}Co has been detected in some air samples at the Hanford, Washington site and Oak Ridge National Laboratories, Tennessee, levels were not reported (HazDat 2004; PNNL 1996).

The concentrations of stable cobalt in surface and groundwater in the United States are generally low; <1 μg/L in pristine areas and 1–10 μg/L in populated areas (Hamilton 1994; Smith and Carson 1981). However, cobalt levels may be considerably higher in mining or agricultural areas. Cobalt concentrations in surface water and groundwater samples collected in 1992 from area creeks near the Blackbird Mine in Idaho, one of the large deposits of cobalt in North America where mining occurred from the late 1800s to 1982, were reported to range from <1 to 625,000 μg/L, and from not detected to 315,000 μg/L, respectively (ATSDR 1995). Cobalt levels in most drinking water is <1–2 μg/L although levels as high as 107 μg/L have been recorded (Greathouse and Craun 1978; Meranger et al. 1981; NAS 1977; Smith and Carson 1981).

Little data are available on the levels of ^{60}Co in water. In 1989, subsequent to the largest effluent discharge from the Steam Generating Heavy Water Reactor at Winfrith on the south coast of England, ^{60}Co levels in offshore seawater from 18 sites contained 0.06–2.22 mBq/L (1.6–69 fCi) of particulate ^{60}Co, 0.30–10.3 mBq/L (8–280 fCi) of soluble ^{60}Co(II), and 0.12–1.55 mBq/L (3.2–42 fCi) of soluble ^{60}Co(III) (Leonard et al. 1993a). The U.S. NRC discharge limit is 111,000 mBq/L (3x10^6 fCi/L) (USNRC 1991).

The average concentrations of cobalt in the earth's crust are 20–25 mg/kg (Abbasi et al. 1989; Merian 1985; Smith and Carson 1981). Most soils contain 1–40 mg cobalt/kg; the average cobalt concentration in U.S. soils is 7.2 mg/kg (Smith and Carson 1981). Soils containing <0.5–3 mg cobalt/kg are considered cobalt-deficient because plants growing on them have insufficient cobalt (<0.08–0.1 mg/kg) to meet the dietary requirements of cattle and sheep. Cobalt-deficient soils are found in some areas of the southeastern and northeastern United States. Soils near ore deposits, phosphate rocks, or ore smelting facilities, and soils contaminated by airport traffic, highway traffic, or other industrial pollution may contain high concentrations of cobalt; concentrations up to 800 mg/kg have been detected in such areas (Kloke et al. 1984; Smith and Carson 1981). Cobalt concentrations in 28 samples collected from surface deposits in the Big Deer and Blackbird Creek drainage basins near a site of former cobalt mining in Idaho ranged from 26.5 to 7,410 mg/kg (ATSDR 1995).

The level of cobalt in most foods is low. However, food is the largest source of exposure to cobalt in the general population. The estimated average daily dietary intake of cobalt in Canada was 11 μg/day. Food groups contributing most heavily to this intake were bakery goods and cereals (29.8%) and vegetables (21.9%) (Dabeka and McKenzie 1995). No estimates of the average dietary input of cobalt in the United States were located. People living near mining and smelting facilities or metal shops where cobalt is used in grinding tools may be exposed to higher levels of cobalt in air or soil. Similarly, people living near hazardous waste sites may be exposed to higher levels of cobalt in these media. Contaminated soils pose a hazardous exposure pathway to children because of both hand-to-mouth behavior and intentional ingestion of soil (pica) that contain metals and other contaminants (Hamel et al. 1998). However, much of the cobalt in soil may not be in a form that is available for uptake by the body. People who work in the hard metal industry, metal mining, smelting, and refining or other industries that produce or use cobalt and cobalt compounds may be exposed to substantially higher levels of cobalt, mainly from dusts or aerosols in air. Workers at nuclear facilities, irradiation facilities, or nuclear waste storage sites may be exposed to radioisotopes of cobalt. Exposure would generally be to radiation produced by these isotopes (e.g., gamma radiation from ^{60}Co).

6.2 RELEASES TO THE ENVIRONMENT

Stable cobalt has been identified in a variety of environmental media (air, surface water, leachate, groundwater, soil, and sediment) collected at 426 of 1,636 current or former NPL hazardous waste sites (HazDat 2004). ^{60}Co has been identified in a variety of environmental media (air, surface water, leachate, groundwater, soil, and sediment) collected at 13 of 1,636 current or former NPL hazardous waste sites (HazDat 2004).

According to the Toxic Chemical Release Inventory (TRI), in 2001, total releases of cobalt and cobalt compounds to the environment (including air, water, soil, and underground injection) from 605 reporting facilities that produced, processed, or used cobalt or cobalt compounds were 16,443,429 pounds (TRI01 2004). Table 6-1 lists amounts released from these facilities grouped by state. In addition, 1,619,874 pounds of cobalt and cobalt compounds were transferred offsite by these facilities (TRI01 2004). Starting in 1998, metal mining, coal mining, electric utilities, and Resource Conservation and Recovery Act (RCRA)/solvent recovery industries are required to report, to the TRI, industries with potentially large releases of cobalt and cobalt compounds. Industrial sectors producing, processing, or using cobalt that contributed the greatest environmental releases in 2001 were primary metals and RCRA/solvent recovery with 141,554 and 531,427 pounds, respectively. Industrial sectors producing, processing, or using cobalt compounds that contributed the greatest environmental releases in 2001 were metal mining and electrical utilities with 10,228,193 and 3,652,398, pounds, respectively. The TRI data should be used with caution because only certain types of facilities are required to report. This is not an exhaustive list.

6.2.1 Air

The sources of cobalt in the atmosphere are both natural and anthropogenic (Barceloux 1999). Natural sources include wind-blown continental dust, seawater spray, volcanoes, forest fires, and continental and marine biogenic emissions. The worldwide emission of cobalt from natural sources has been estimated to range from 13 to 15 million pounds/year (Lantzy and Mackenzie 1979; Nriagu 1989). The global atmospheric emission of cobalt from anthropogenic sources is an estimated 9.7 million pounds/year. Therefore, natural sources contribute slightly more to cobalt emissions in the atmosphere than anthropogenic sources (Lantzy and Mackenzie 1979). The primary anthropogenic sources of cobalt in the atmosphere are the burning of fossil fuels and sewage sludge, phosphate fertilizers, mining and smelting of cobalt-containing ores, processing of cobalt-containing alloys, and industries that use or process cobalt compounds. Small amounts of cobalt are found in coal, crude oils, and oil shales. Therefore, burning of these fossil fuels for power generation will emit cobalt into the atmosphere. The cobalt contents of the fly ash and flue gases of a coal-burning power plant are approximately 25 mg/kg and 100–700 μg/L, respectively. Gasoline contains <0.1 mg cobalt/kg, but catalytic converters may contain cobalt; therefore, emissions from vehicular exhaust are also a source of atmospheric cobalt (Abbasi et al. 1989; Holcombe

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

		Reported amounts released in pounds per year[b]						
State[c]	Number of facilities	Air[d]	Water	Under-ground injection	Land	Total on-site release[e]	Total off-site release[f]	Total on and off-site release
AK	2	23	0	16,000	546,463	562,486	0	562,486
AL	21	5,893	8,612	0	315,853	330,358	30,040	360,398
AR	9	921	142	0	8,301	9,364	2,015	11,379
AZ	12	1,029	0	0	1,061,035	1,062,064	2,266	1,064,330
CA	26	646	20	0	307,654	308,320	7,463	315,783
CO	1	3	1	0	12,026	12,030	0	12,030
CT	9	632	65	0	0	697	4,133	4,830
DE	2	1,265	52	0	52	1,369	27,444	28,813
FL	11	2,397	345	0	93,049	95,791	15,464	111,255
GA	17	3,508	268	0	282,610	286,386	12,461	298,847
IA	6	566	0	0	0	566	2,123	2,689
ID	2	74	5	0	395,424	395,503	0	395,503
IL	23	1,630	1,278	0	16,999	19,907	102,088	121,995
IN	42	7,005	351	0	279,122	286,478	64,293	350,771
KS	5	4,269	0	0	10,200	14,469	3,859	18,328
KY	22	3,184	542	0	478,855	482,581	13,269	495,850
LA	15	385	8,477	2,700	66,858	78,420	91,274	169,694
MA	11	794	780	0	5	1,579	17,403	18,982
MD	6	2,472	15	0	6,629	9,116	45,382	54,498
ME	2	66	0	0	0	66	700	766
MI	24	4,699	559	0	125,405	130,663	33,737	164,400
MN	5	255	No data	0	0	255	7,666	7,921
MO	5	1,457	8	0	559,401	560,866	0	560,866
MS	7	386	120	12,000	44	12,550	3,044	15,594
MT	1	250	No data	0	31,000	31,250	505	31,755
NC	24	6,593	8,257	0	194,974	209,824	216,849	426,673
ND	4	1,165	21	0	108,300	109,486	39,842	149,328
NE	1	0	27	0	0	27	3,982	4,009
NH	1	0	No data	0	0	0	No data	0
NJ	12	1,191	26	0	413	1,630	26,894	28,524
NM	6	498	1	0	4,257,140	4,257,639	69	4,257,708
NV	8	678	0	0	4,099,136	4,099,814	9,950	4,109,764
NY	12	755	44	0	11,843	12,642	14,322	26,964
OH	44	4,977	771	1,100	310,653	317,501	96,116	413,617
OK	13	1,357	158	0	5,677	7,192	20,760	27,952

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

		Reported amounts released in pounds per year[b]						
State[c]	Number of facilities	Air[d]	Water	Under-ground injection	Land	Total on-site release[e]	Total off-site release[f]	Total on and off-site release
OR	6	1,262	20	0	16,487	17,769	2,862	20,631
PA	44	6,169	3,176	0	51,350	60,695	221,662	282,357
PR	2	2	No data	0	0	2	2,871	2,873
RI	1	1	1	0	0	2	50	52
SC	25	1,579	10,970	0	43,488	56,037	70,316	126,353
SD	1	0	No data	0	0	0	0	0
TN	18	5,560	4,013	0	330,615	340,188	36,520	376,708
TX	44	8,126	784	3,730	150,470	163,110	95,840	258,950
UT	6	278	No data	0	23,350	23,628	126,502	150,130
VA	9	1,451	518	0	89,388	91,357	9,683	101,040
VI	1	0	0	0	0	0	0	0
WA	2	72	91	0	106,618	106,781	5,112	111,893
WI	20	1,098	5	0	8	1,111	95,996	97,107
WV	13	1,341	566	0	212,254	214,161	37,047	251,208
WY	2	898	No data	0	38,927	39,825	0	39,825
Total	605	88,860	51,089	35,530	14,648,076	14,823,555	1,619,874	16,443,429

Source: TRI01 2004

[a] The TRI data should be used with caution since only certain types of facilities are required to report. This is not an exhaustive list. Data are rounded to nearest whole number.
[b] Data in TRI are maximum amounts released by each facility.
[c] Post office state abbreviations are used.
[d] The sum of fugitive and stack releases are included in releases to air by a given facility.
[e] The sum of all releases of the chemical to air, land, water, and underground injection wells.
[f] Total amount of chemical transferred off-site, including to publicly owned treatment works (POTW).

et al. 1985; Ondov et al. 1982; Smith and Carson 1981). Cobalt has been detected in cigarette tobacco and therefore, smoking is a potential source of atmospheric cobalt that could impact on indoor air quality (Munita and Mazzilli 1986).

Stable cobalt has been identified in air samples collected at 5 of the 426 current or former NPL hazardous waste sites where it was detected in some environmental media (i.e., air, soil, sediment, or water) (HazDat 2004). ^{60}Co has been identified in air samples collected at 2 of the 13 current or former NPL hazardous waste sites where it was detected in some environmental media (HazDat 2004).

Air sampling data were used to estimate ^{60}Co release from the Savannah River Site (SRS) from the plant's start up in 1954 to 1989 (DOE 1991). From this monitoring, it was estimated that 0.092 Ci (3.4 GBq) of ^{60}Co was released to the atmosphere between 1968 and 1986. Total releases of ^{60}Co to the atmosphere from the SRS between 1968 and 1996 were 0.092 Ci (3.4 GBq) (DOE 1998). Data were not reported for all years in this interval. In 1999, atmospheric releases of ^{57}Co, ^{58}Co, and ^{60}Co as particulates were 4.71×10^{-8}, 1.27×10^{-4}, and 1.30×10^{-4} Ci (0.00174, 4.70, and 4.81 MBq), respectively (DOE 1999). The SRS was a major production facility to the U.S. defense program and included five nuclear reactors, a fuel fabrication plant, a naval fuel materials facility, two chemical separation plants, a heavy water production plant, and a laboratory. ^{60}Co has also been detected in air samples at the Hanford site and Oak Ridge National Laboratories (HazDat 2004; PNNL 1996).

According to the TRI, in 2001, releases of 88,860 pounds of cobalt and cobalt compounds to air from 605 reporting facilities accounted for 0.5% of the total onsite environmental releases of these substances (TRI01 2004). The industrial sectors contributing the largest release of cobalt and cobalt compounds to air were electrical utilities, chemicals, and primary metals. Table 6-1 lists the amounts of cobalt and cobalt compounds released to air from these facilities grouped by state. The TRI data should be used with caution, however, since only certain types of facilities are required to report. This is not an exhaustive list.

6.2.2 Water

Compounds of cobalt occur naturally in seawater and in some surface, spring, and groundwater (Smith and Carson 1981). Cobalt is also released into water from anthropogenic sources. While there has been no mine production of cobalt in the United States in recent years, cobalt is a byproduct or coproduct of the refining of other mined metals such as copper and nickel. Historic mining operations that processed cobalt containing ores may continue to release cobalt into surface water and groundwater. Waste water from the recovery of cobalt from imported matte or scrap metal, refining of copper and nickel, or during the manufacture of cobalt chemicals are sources of cobalt in water (Smith and Carson 1981). Process water and effluent from coal gasification and residue from solvent-refined coal contain cobalt. The accidental discharge of activated sludge and sewage may be important sources of cobalamins in waterways, together with bioconcentration by benthic organisms (Smith and Carson 1981). The discharge of waste water by user industries, such as paint and pigment manufacture, also contributes to the release of cobalt into water. In one case, manufacturers of nickel-cadmium batteries operating between 1953 and 1979 discharged cobalt from a battery factory to the Hudson River in Foundry Cove, New York, of which 1.2 metric tons are estimated to be present in the eastern cove (Knutson et al. 1987). Atmospheric deposition is an additional source of cobalt in water. Lake Huron receives an estimated 76% of its cobalt input from natural sources and 24% from anthropogenic sources. The corresponding estimated values for Lake Superior are 85.4 and 14.6% (Smith and Carson 1981). In these Great Lakes, it therefore appears that natural inputs of cobalt far exceed anthropogenic ones.

Cobalt has been identified in groundwater and surface water at 255 and 106 sites, respectively, of the 426 NPL hazardous waste sites, where it was detected in some environmental media (i.e., air, soil, sediment, or water) (HazDat 2004). ^{60}Co has been identified in groundwater and surface water at 4 and 2 sites, respectively, of the 13 NPL hazardous waste sites, where it was detected in some environmental media (HazDat 2004).

According to the TRI, in 2001, the reported releases of 51,089 pounds of cobalt and cobalt compounds to water from 605 reporting facilities accounted for 0.3% of the total onsite environmental releases of these substances (TRI01 2004). Table 6-1 lists the amounts of cobalt and cobalt compounds released to water from these facilities grouped by state. As of 1998, TRI no longer separately collects data on substances released indirectly to Publicly-Owned Treatment Works (POTWs), part of which may ultimately be released to surface waters. The TRI data should be used with caution, however, since only certain types of facilities are required to report. This is not an exhaustive list.

^{60}Co is present in the low-level aqueous radioactive waste discharges from many nuclear power plants. Alloys that contain stable cobalt (^{59}Co), such as stellite, used in piping of nuclear reactors corrode and may be activated, producing ^{60}Co, which accumulates in the reactor and must be periodically decontaminated. A common decontaminating agent includes a reducing metal ion (e.g., vanadium(II)) and a chelating agent (e.g., picolinate) resulting in low-level discharges of uncomplexed ^{60}Co(II) and complexed ^{60}Co(III). While soluble ionic and particulate forms predominate, at some sites stable, nonionic trivalent complexes of cobalt are present (Leonard et al. 1993a, 1993b; USNRC 2000d). For example, in 1987–1989 samples of treated effluent from the Steam Generating Heavy Water Reactor at Winfrith on the south coast of England, the percent of ^{60}Co as Co(III) picolinate ranged from 6.2 to 75.4%. Between 1978 and 1988, 12 TBq (320 Ci) of ^{60}Co was released into the Irish Sea by the British Nuclear Fuels reprocessing plant at Sellafield, United Kingdom (McCartney et al. 1994). These discharges are believed to be Co(II) (Leonard et al. 1993a). Both ^{58}Co and ^{60}Co are discharged into the Rhone River by the nuclear power plant at Bugey, France. This facility, which consists of a natural Uranium-Graphite-Gas unit and four pressurized water reactor (PWR) units, two of which are cooled by Rhone River water, discharged about 406 and 280 GBq (11.0 and 7.56 Ci) of ^{58}Co and ^{60}Co, respectively, in liquid waste during 1986–1990 (Beaugelin-Seiler et al. 1994).

Water sampling data were used to estimate effluent release from the SRS from the plant's start up in 1954 to 1989 (DOE 1991). From this monitoring, it was estimated that 17.8 Ci (659 GBq) of ^{60}Co were released into seepage basins and 66.4 Ci (2,460 GBq) were released into streams between 1955 and 1988. In addition, 2.7 Ci (100 GBq) of ^{58}Co were released into seepage basins between 1971 and 1988; no ^{58}Co was released into streams. Total releases of ^{60}Co to streams from the SRS for 1954–1995 were 66 Ci (2,400 GBq) (DOE 1998). No data were reported from 1985 to 1994. In 1999, 4.94×10^{-4} Ci (0.0183 GBq) of ^{60}Co was released to surface waters at the SRS (DOE 1999). ^{60}Co has also been reported in surface water at, Hanford, Washington, and Oak Ridge National Laboratories, and groundwater at Brook Industrial Park, New Jersey, the Hanford site and Oak Ridge National Laboratories, Tennessee (HazDat 2004). The Columbia River receives discharges from the unconfined aquifer underlying the Hanford Site via subsurface and surface (riverbank springs) discharges. This aquifer is contaminated by leachate from past waste-disposal practices at the site.

6.2.3 Soil

Cobalt occurs naturally in the earth's crust, and therefore, in soil. However, elevated levels of cobalt in soil may result from anthropogenic activities such as the mining and processing of cobalt-bearing ores, the application of cobalt-containing sludge or phosphate fertilizers to soil, the disposal of cobalt-containing wastes, and atmospheric deposition from activities such as burning of fossil fuels, smelting, and metal refining (Smith and Carson 1981).

Cobalt has been identified in soil at 219 sites and sediment at 143 sites collected from 426 NPL hazardous waste sites, where it was detected in some environmental media (i.e., air, soil, sediment, or water) (HazDat 2004). ^{60}Co has been identified in soil at 8 sites and sediment at 2 sites collected from 13 NPL hazardous waste sites, where it was detected in some environmental media (HazDat 2004). ^{60}Co has been detected onsite in soils at the Hanford Site, Washington; INEEL, Idaho; Lawrence Livermore National Laboratory, Main Site, California; and Robins Air Force Base, Georgia at maximum concentrations of 87.7, 570, 0.21, and 0.07 pCi/g (3.24, 21, 0.0078, and 0.003 Bq/g) (HazDat 2004).

According to the TRI, in 2001, reported releases of 14,646,076 pounds of cobalt and cobalt compounds to land from 605 reporting facilities accounted for 98.8% of the total onsite environmental releases of these substances (TRI01 2004). An additional 35,530 pounds, accounting for 0.2% of the total onsite environmental releases were injected underground (TRI01 2004). Industrial sectors contributing the largest releases of cobalt and cobalt compounds to land were metal mining and electrical utilities with 10,210,508 and 3,197,209 pounds, respectively. Table 6-1 lists the amounts of cobalt and cobalt compounds released on land from these facilities grouped by state. The TRI data should be used with caution, however, since only certain types of facilities are required to report. This is not an exhaustive list.

6.3 ENVIRONMENTAL FATE

6.3.1 Transport and Partitioning

Cobalt compounds are nonvolatile, and thus, cobalt is emitted to the atmosphere in particulate form. The transport of cobalt in air depends on its particle size and density, and meteorological conditions; it can be returned to land or surface water by rain or it may settle to the ground by dry deposition. In nonarid areas, wet deposition may exceed dry deposition (Arimoto et al. 1985; Erlandsson et al. 1983). Coarse particles, with aerodynamic diameters >2 μm (such as those obtained during ore processing), may deposit within 10 km from the point of emission; finer particles may travel longer distances. It is the larger particles that may be responsible for elevated local concentrations around emission sources. The mass median diameter for cobalt particles emitted from a power generator with a stack emission controlled by an electrostatic precipitator or scrubber ranged from <2 to 12 μm. The mass median diameter of cobalt in the ambient atmosphere is about 2.6 μm (Milford and Davidson 1985). Golomb et al. (1997) report average total (wet+dry) deposition rates of cobalt to Massachusetts Bay during the period September 15, 1992 to September 16, 1993. The total deposition rate was 58 μg/m^2-year, of which 47 μg/m^2-year was dry deposition and 12 μg/m^2-year was wet deposition. Total cobalt deposition flux at a site in the Rhone delta in southern France in 1988–1989 was 0.42±0.23 kg/km^2-year with 0.15 kg/km^2-year in the form of wet deposition (Guieu et al. 1991).

As with most metals, sediment and soil are frequently the final repository for cobalt released into the environment, although the process is dynamic, and cobalt can be released into the water depending upon conditions. Cobalt released into waterways may sorb to particles and settle into the sediment or be sorbed directly into the sediment. However, complexation cobalt to dissolved organic substances can significantly reduce sorption to sediment particles (Albrecht 2003). Studies by Jackman et al. (2001) suggest that interparticle migration of cobalt can influence the transport of metal ions, including cobalt, in sediments. For example, migration of a metal ion from a highly mobile sediment particle, such as clay, to less mobile gravels will slow the transport of that metal. Cobalt can also be transported in dissolved form or as suspended sediment by rivers to lakes and the sea or by ocean currents. Sediment in areas of active sedimentation would receive a large portion of the suspended sediment. In the case of the Peach Bottom Atomic Power Plant where ^{60}Co is released into the Conowingo Reservoir, an impoundment of the lower Susquehanna River, <20% of the radionuclide is trapped in the reservoir sediment, the rest being transported downstream and into the Chesapeake Bay (McLean and Summers 1990). It is often assumed that the primary mode of transport of heavy metals in aquatic systems is as suspended solids (Beijer and Jernelov 1986). However, in the case of cobalt, the percent that is transported by suspended solids is highly variable. Examples of the percentage of cobalt transported in suspended solids include (water body, percent): Main River (Germany), 33.4–42.2%; Susquehanna River (near its source in New York), 9%; New Hope River (North Carolina), 92%; Yukon River, >98%; Danube Rive (1961–1970), 27.4–85.9%; Columbia River (^{60}Co, downstream of the Hanford site), 95–98%; Strait of Juan de Fuca (Puget Sound, Washington), 11–15%; North Sea, 34%; and Lake Washington (Washington), 0% (Smith and Carson 1981).

In the oxic zones of many surface waters, dissolved cobalt levels decrease with increasing depth. This may be due to cobalt's continuous input into surface water from discharges or to increased adsorption and precipitation of the soluble forms with increasing depth. The fact that cobalt concentration profiles in deep water follow manganese and aluminum profiles strongly suggests that dissolved cobalt is precipitated in the adsorbed state with oxides of iron and manganese and with crystalline sediments such as aluminosilicate and goethite. A part of the cobalt may also precipitate out as carbonate and hydroxide in water. The higher concentration of organic pollutants in polluted water probably results in the formation of higher concentrations of soluble organic complexes. In a deep sediment where the water was anoxic and contained hydrogen sulfide, some mobilization of cobalt was observed, probably due to the formation of bisulfide and polysulfide complexes (Bargagli 2000; Brügmann 1988; Finney and Huh 1989; Glooschenko et al. 1981; Knauer et al. 1982; Nriagu and Coker 1980; Shine et al. 1995; Smith and Carson 1981; Szefer et al. 1996; Windom et al. 1989).

Cobalt strongly binds to humic substances naturally present in aquatic environments. Humic acids can be modified by UV light and bacterial decomposition, which may change their binding characteristics over time. The lability of the complexes is strongly influenced by pH, the nature of the humic material, and the metal-to-humic substance ratio. The lability of cobalt-humate complexes decreases in time ("aging effect") (Burba et al. 1994). The "aging effect" indicates that after a period of time (~12 hours), complexes that were initially formed are transformed into stronger ones from which the metal ion is less readily dislodged. In the Scheldt Estuary and the Irish Sea, between 45 and 100% of dissolved cobalt was found to occur in these very strong complexes (Zhang et al. 1990). Aquifer material from the contaminated aquifer at a low-level infiltration pit at the Chalk River Nuclear Laboratories in Canada was analyzed to assess the nature of the adsorbed ^{60}Co using sequential leaching techniques (Killey et al. 1984). Of the sediment-bound ^{60}Co, <10% was exchangeable, 5–35% was retained by iron oxide, and 55–>90% was fixed. Over 80% of the dissolved ^{60}Co was present as weakly anionic hydrophilic organic complexes. The average K_d for ^{60}Co between particulate matter and Po River (Italy) water was 451 m^3/kg over a 2-year monitoring period (Pettine et al. (1994). The mean K_d for ^{60}Co in Arctic surface sediment (Kara Sea) where large quantities of radioactive waste by the former Soviet Union was disposed was $1x10^5$ L/kg (range $1x10^3$–$7x10^5$), which is comparable to that in temperate coastal regions, $2x10^5$ L/kg (range, $2x10^4$–$1x10^6$) (Fisher et al. 1999).

The distribution coefficient of cobalt may vary considerably in the same sediment in response to conditions affecting the pH, redox conditions, ionic strength, and amount of dissolved organic matter

(Mahara and Kudo 1981b). Uptake of ^{60}Co from the water by sediment increased rapidly as the pH was increased from 5 to 7–7.5 and then slightly decrease (Benes et al. 1989a, 1989b). Therefore, pH would be an important factor affecting the migration of cobalt in surface water. Uptake was little affected by changes in liquid-to-solids ratio and ionic strength. ^{60}Co is more mobile in anaerobic marine aquatic environments than in freshwater aerobic ones (Mahara and Kudo 1981b). Therefore, ^{60}Co waste is most suitably stored underground in aerated zones away from possible seawater intrusions. In seawater-sediment systems under anaerobic conditions ^{60}Co was 250 times more mobile than ^{60}Co in freshwater-sediment systems under aerobic conditions. Under anaerobic conditions, 30% of the ^{60}Co added to a sediment-freshwater system was 'exchangeable' and therefore potentially mobile, while under aerobic conditions, 98% of the ^{60}Co was permanently fixed. Most of the mobile ^{60}Co produced under anaerobic conditions in seawater consisted of nonionic cobalt associated with low molecular weight organic substances that were stable to changes in pH; the exchangeable ^{60}Co appeared to be mostly ionic.

Bird et al. (1998b) added ^{60}Co to the anoxic hypolimnion of a Canadian Shield lake to simulate a nuclear waste scenario where radionuclides entered the bottom waters of a lake, and evaluated its behavior over 5 years. This situation was considered to be a likely pathway by which nuclear fuel waste stored deep underground in the plutonic (igneous) rock of this region would reach the surface environment via deep groundwater flow into the bottom waters of a lake. It was felt that adding a redox sensitive element such as cobalt to the anoxic hypolimnion might be different from adding it to the epilimnion. Monitoring vertical profiles in the lake established that the cobalt remained confined to the anoxic hypolimnion prior to the fall turnover (first 72 days) when mixing occurred throughout the water column. After 358 days, only about 4% of the ^{60}Co remained in the water. After the second year, approximately 2% of the ^{60}Co remained and after 5 years, only 0.4%. These results mirror previous experiments in which the ^{60}Co was added to the epilimnion, therefore establishing that there is little difference in the overall behavior of cobalt when added to the epilimnion or hypolimnion. The loss rate coefficient of ^{60}Co was 0.036/day (half-life=19 days) between days 90 and 131 (lake mixing) during which time, the cobalt sorbed to the suspended sediment and bottom sediment under anoxic conditions. Loss was to the sediment as there was no hydrological loss from the lake. In the previous experiment in which ^{60}Co was added to the epilimnion, the initial loss rate coefficient was somewhat higher, 0.056/day (half-life=12 days). Following the initial loss, ^{60}Co continued to be slowly removed from the water (loss rate coefficient 0.002/day; half-life=347 days); after 328 days, ^{60}Co was no longer detectable in the epilimnion. The half-life of ^{60}Co in the water column of an experimental lake in northwestern Ontario was 11 days; 5% of added ^{60}Co remained in the water after 100 days (Bird et al. 1998b). The redox potential also affects the behavior of cobalt in sediment. Under moderately reducing conditions, cobalt is released from sediment as Co^{2+} and forms CoS in the presence of sulfide. The concentration of cobalt in the bottom water increases as the water becomes more anoxic (Brügmann 1988; Smith and Carson 1981).

The mobility of cobalt in soil is inversely related to how strongly it is adsorbed by soil constituents. Cobalt may be retained by mineral oxides such as iron and manganese oxide, crystalline materials such as aluminosilicate and goethite, and natural organic substances in soil. Sorption of cobalt to soil occurs rapidly (within 1–2 hours). Soil-derived oxide materials were found to adsorb greater amounts of cobalt than other materials examined, although substantial amounts were also adsorbed by organic materials. Clay minerals sorbed relatively smaller amounts of cobalt (McLaren et al. 1986). In addition, little cobalt was desorbed from soil oxides while substantial amounts desorbed from humic acids and montorillonite. In clay soil, adsorption may be due to ion exchange at the cationic sites on clay with either simple ionic cobalt or hydrolyzed ionic species such as $CoOH^+$. Adsorption of cobalt onto iron and manganese increases with pH (Brooks et al. 1998). In addition, as pH increases, insoluble hydroxides or carbonates may form, which would also reduce cobalt mobility. Conversely, sorption onto mobile colloids would enhance its mobility. In most soils, cobalt is more mobile than lead, chromium (II), zinc, and nickel, but less mobile than cadmium (Baes and Sharp 1983; King 1988b; Mahara and Kudo 1981b; Smith and Carson 1981). In several studies, the K_d of cobalt in a variety of soils ranged from 0.2 to 3,800. The geometric mean, minimum, median, and maximum K_ds of ^{60}Co in 36 Japanese agricultural soils were 1,840, 130, 1,735, and 104,000 L/kg, respectively (Yasuda et al. 1995). The soil properties showing the highest correlation with K_d were exchangeable calcium, pH, water content, and cation exchange capacity (CEC). In 11 U.S. soils, the mean Freundlich K_F and n values were 37 L/kg and 0.754, respectively; K_F values ranged from 2.6 to 363 L/kg and correlated with soil pH and CEC (Buchter et al. 1989). In 13 soils from the southeastern United States whose soil pH ranged from 3.9 to 6.5, cobalt sorption ranged from 15 to 93%; soil pH accounted for 84–95% of the variation in sorption (King 1988b).

Organic complexing agents such as ethylenediaminetetraacetic acid (EDTA), which are used for decontamination operations at nuclear facilities, greatly enhance the mobility of cobalt in soil. Other organic complexing agents, such as those obtained from plant decay, may also increase cobalt mobility in soil. However, both types of complexes decrease cobalt uptake by plants (Killey et al. 1984; McLaren et al. 1986; Toste et al. 1984). Addition of sewage sludge to soil also increases the mobility of cobalt, perhaps due to organic complexation of cobalt (Gerritse et al. 1982; Williams et al. 1985).

Leaching of cobalt has been observed from municipal and low-level radioactive waste sites (Cyr et al. 1987; Czyscinski et al. 1982; Friedman and Kelmers 1988). The mobility of cobalt was assessed in two soils from the Cabriolet and Little Feller event sites at the Nevada Test site as a function of various parameters such as pH, ionic strength, cobalt concentrations, soil solids concentrations, and particle size distribution (DOE 1996). Cobalt was quantitatively sorbed on these soils (at least 90% sorbed) when the pH was above 7 and the solid concentration was at least 20 g/L. The experiments suggest that binding is principally on amphoteric surface-hydroxyl surfaces. Since the pH of these soils is around 8, cobalt would bind strongly under normal environmental conditions. Migration would be severely retarded under all but the most extreme conditions, e.g., pH of 4 or below and high ionic strength soil solutions (approximately 0.1 M). In addition, unrealistically large quantities of water would be needed to displace cobalt from the upper layers of the soil profile.

Cobalt may be taken up from soil by plants. Surface deposition of cobalt on leaves of plants from airborne particles may also occur. Elevated levels of cobalt have been found in the roots of sugar beets and potato tubers in soils with high cobalt concentrations (e.g., fly ash-amended soil) due to absorption of cobalt from soil. However, the translocation of cobalt from roots to above-ground parts of plants is not significant in most soils, as indicated by the lack of cobalt in seeds of barley, oats, and wheat grown in high-cobalt soil (Mermut et al. 1996; Smith and Carson 1981). Mermut et al. (1996) found 0.01–0.02 mg/kg in 10 samples of durum wheat grain from different areas of Saskatchewan where surface soil cobalt levels ranged from 3.7 to 16.4 mg/kg. The enrichment ratio, defined as the concentration in a plant grown in amended soil (fly ash) over the concentration in unamended soil, was about 1. Other authors have determined the transfer coefficient (concentration in plant/concentration in soil) for cobalt to be 0.01–0.3. The mean ^{57}Co soil-plant transfer factors obtained for clover from eight soils over a 4-year period ranged from 0.02 to 0.35, in good agreement with results of other investigators (Mascanzoni 1989). However, in highly acidic soil (pH as low as 3.3), significantly higher than normal concentrations of cobalt were found in rye grass foliage, oats, and barley. For example, cobalt concentrations in rye grass grown in unlimed soil (pH<5.0) was 19.7 mg/kg compared with 1.1 mg/kg in rye grass grown in limed soil (pH>5.0) (Boikat et al. 1985; Francis et al. 1985; Kloke et al. 1984; Mejstrik and Svacha 1988; Palko and Yli-Hala 1988; Tolle et al. 1983; Watabe et al. 1984). Soil and plant samples taken in the 30-km zone around Chernobyl indicated that ^{60}Co was not accumulated by plants and mushrooms (Lux et al. 1995). Transfer factors obtained in 1992 ranged from 0.005 to 0.16 and those obtained in 1993 ranged from <0.001 to 0.008. Studies investigating the uptake of ^{60}Co by tomato plants watered with ^{60}Co-contaminated water showed that tomato plants absorbed <2% of the activity available from the soil. The absorption was 6 times higher if the plants were watered aerially rather than ground watering. Using either watering method, >90% of the activity was absorbed by the stems and leaves (Sabbarese et al. 2002). Soil to plant transfer factors for ^{60}Co were determined for plants grown in containers with soil contaminated with ^{65}Zn and ^{60}Co over a 3-year period under outdoor tropical conditions. Average transfer factors for ^{60}Co over the 3-year period ranged from a high for spinach (1.030) to a low for rice (0.087) (Mollah and Begum 2001).

^{60}Co is taken up by phytoplankton and unicellular algae (*Senenastrum capricornutum*) with concentration factors (dry weight) ranging from 15,000 to 40,000 and 2,300 to 18,000, respectively (Corisco and Carreiro 1999). Elimination experiments with the algae indicate a two component biological half-life, 1 hour and 11 days, respectively, and suggest that the cobalt might be absorbed not only on the surface, but also intracellularly. Since these organisms are at the bottom of the food chain, they could play an important role in the trophic transfer of ^{60}Co released into waterways by nuclear facilities. However, cobalt levels generally diminish with increasing trophic levels in a food chain (Smith and Carson 1981).

The low levels of cobalt in fish may also reflect cobalt's strong binding to particles and sediment. The bioaccumulation factors (dry weight basis) for cobalt in marine and freshwater fish are ~100–4,000 and <10–1,000, respectively; accumulation in the muscle of marine fish is 5–500 (Smith and Carson 1981). Cobalt largely accumulates in the viscera and on the skin, as opposed to the edible parts of the fish. In carp, accumulation from water accounted for 75% of ^{60}Co accumulated from both water and food; accumulation from water and food was additive (Baudin and Fritsch 1989). Depuration half-lives were 53 and 87 days for fish contaminated from food and water, respectively. In the case of an accidental release of ^{60}Co into waterways, the implication is that effects would manifest themselves rapidly since the primary route of exposure is from water rather than food. Uptake of ^{60}Co by biota in lakes in northwestern Ontario was not affected by the tropic status of the lakes (Bird et al. 1998a). Uptake of ^{60}Co was very low in whitefish, with concentrations being highest in kidney and undetectable in muscle. Similarly, while accumulation of ^{60}Co by carp from food was dependent on food type, the transfer factor was very low, approximately 0.01, and no long-term bioaccumulation of the radionuclide occurred (Baudin and Fritsch 1987; Baudin et al. 1990). Accumulation of ^{60}Co from food for rainbow trout showed that after the 42-day exposure period, the highest concentrations of ^{60}Co were found in the kidneys, secondary gut, and viscera, and the trophic transfer factor was 0.0186. After 73 days of depuration, residual ^{60}Co concentrations were the highest in the kidneys, viscera, and fins (Baudin et al. 2000). In the experiment described above in which Bird et al. (1998a) added ^{60}Co to the anoxic hypolimnion of a Canadian Shield lake to simulate a nuclear waste scenario where radionuclides entered the bottom waters of a lake, ^{60}Co levels in biota were low because of the rapid loss of cobalt to the sediment. Levels in forage fish, minnows, and sculpins were low, <0.3 Bq/g (8 pCi/g) dry weight; an occasional high level, ~4 Bq/g (110 pCi/g) dry weight, in slimy sculpin was thought to reflect the presence of detritus in the gut of the fish. Epilimnion additions of ^{60}Co in an earlier study resulted in lower maximum concentrations in fish, 0.07, 0.11, and 0.01 Bq/g (2, 3.0, and 0.3 pCi/g) dry weight in pearl dace, fathead minnows, and slimy sculpins, respectively, when similar quantities of radioactive cobalt were added to the lake.

Concentration factors have also been reported for various other aquatic organisms. Freshwater mollusks have concentration factors of 100–14,000 (~1–300 in soft tissue). Much of the cobalt taken up by mollusks and crustacae from water or sediment is adsorbed to the shell or exoskeleton; very little cobalt is generally accumulated in the edible parts (Amiard and Amiard-Triquet 1979; Smith and Carson 1981). A concentration factor for ^{60}Co of 265 mL/g (wet weight) was determined for *Daphnia magna* in laboratory studies. The rapid decrease in radioactivity during the depuration phase indicated that adsorption to the surface was the major contamination process (Adam et al. 2001). However, the digestive glands of crustaceans, which are sometimes eaten by humans, may accumulate high levels of ^{60}Co. Five different species of marine mollusks had whole-body ^{60}Co concentration factors between 6.3 and 84 after 1-month exposure to ^{60}Co in seawater (Carvalho 1987). The shell accounted for more than half of the body-burden. Among the soft tissue, the gills and viscera had the highest concentrations factors and the muscle had the lowest. Fisher et al. (1996) studied the release of ^{60}Co accumulated in mussels from water and ingested phytoplankton. In each case, there was a slow and fast component to the release; the rapid release was in the form of fecal pellets if uptake was from food and from desorption from the shell if uptake was from the dissolved phase. Biological half-lives obtained in laboratory studies were about 12–21 days from both the shell and soft parts. Higher absorption efficiencies and lower efflux rates were obtained for cobalamins than for inorganic cobalt, suggesting that it is a more bioavailable form of cobalt for mussels. Cobalt from fecal pellets is rapidly released into the overlying water and may play a role in its geochemical cycling (Fisher et al. 1996). The concentration of cobalt in clams in the Indian River Lagoon, Florida did not correlate with levels found in either water or sediment (Trocine and Trefry 1996). Kinetics of bioaccumulation of ^{57}Co from water and depuration by starfish (*Asterias rubens*) were carried out in laboratory studies. After 32 days of exposure to seawater containing ^{57}Co, whole body uptake from seawater reached a concentration factor of 23 (wet weight). ^{57}Co was released with a half-life of 27 days after removal to uncontaminated water. Comparison of the kinetics of loss of ^{57}Co following exposure to

^{57}Co-contaminated food versus exposure from ^{57}Co-contaminated water indicate that *A. rubens* accumulates ^{57}Co predominately from seawater rather than from food (Warnau et al. 1999).

6.3.2 Transformation and Degradation

6.3.2.1 Air

There is a paucity of data in the literature regarding the chemical forms of cobalt in air and their transformations in the atmosphere. It is generally assumed that anthropogenic cobalt originating from combustion sources exists primarily as the oxide (Schroeder et al. 1987). In addition, cobalt may be released into the atmosphere as its arsenide or sulfide during ore extraction processes. It is not clear if these species are transformed in the atmosphere. Should a relatively insoluble species such as the oxide be transformed into a more soluble form such as the sulfate, one would expect greater quantities to be washed out of the atmosphere in rain.

6.3.2.2 Water

Many factors control the speciation and fate of cobalt in natural waters and sediments. These include the presence of organic ligands (e.g., humic acids, EDTA), the presence and concentration of anions (Cl^-, OH^-, CO_3^{-2}, HCO_3^-, SO_4^{-2}), pH, and redox potential (Eh). Modeling the chemical speciation of a metal in water depends upon the environmental factors assumed and the stability constants of the various complexes. Mantoura et al. (1978) predicted the equilibrium levels of Co^{2+} species in fresh water to follow the order: free $Co^{+2} \geq CoCO_3 > CoHCO_3^+ >> CoSO_4 \geq Co \bullet$humic acid. However, the mole percent of various cobalt species in a Welsh lake was found to be: free Co^{+2}, 76%; $CoCO_3$, 9.8%; $CoHCO_3^+$, 9.6%; humate complexes, 4.0%; and $CoSO_4$, 0.4%. The rank order of species concentration in seawater was estimated to be: $CoCO_3 >$ free $Co^{+2} > CoSO_4 \geq CoHCO_3^+$. In another model, the speciation of cobalt was completely different with $CoCl^+ >$ free $Co^{+2} > CoCO_3 > CoSO_4$ (Smith and Carson 1981). More recently, Tipping et al. (1998) estimated the equilibrium speciation of cobalt in riverine, estuarine, and marine surface water of the Humber system (England). In all but seawater, cobalt complexes with carbonate (HCO_3^- and CO_3^{2-}) constituted about 70% of dissolved cobalt while the free Co^{2+} ion, was a major species, ~25%, which is much lower than the 61% predicted by Mantoura et al. (1978). As the alkalinity of the water increases, the proportion of cobalt complexed with carbonate increases at the expense of free Co^{2+}. The proportion, but not the concentration, of cobalt that exists as the free ion and the carbonate complexes in river water is independent of the level of fulvic acid in the water. In seawater, the carbonate species and the free aqua species assume roughly equal importance. The proportion of dissolved cobalt complexed with fulvic acid decreased with increasing salinity. About 20% of cobalt in seawater was estimated to be present as complexes with sulfate. In a bioconcentration study in which $CoCl_2$ was initially added to the seawater, at month's end, the cationic form of cobalt was progressively converted into anionic and neutral forms, possibly as a result of complexation with organic ligands (Carvalho 1987). Addition of humic acid to natural waters may merely increase the concentration of colloidal dispersed metal rather than form truly soluble humic complexes. In water that contains high organic wastes such as was the case in the Rhone River in France, cobalt was almost completely complexed. A recent study determined that the distribution of ^{60}Co in the Rhone River sampled at Arles, France was 45% in the particulate phase, 30% in the dissolved phase, and 25% in the colloidal phase (Eyrolle and Charmasson 2001). Cobalt forms complexes with EDTA that are very stable environmentally. EDTA is often used in agriculture, food and drug processing, photography, and textile and paper manufacturing, and therefore, it is a likely constituent of industrial discharges.

Acidity and redox potential have an effect on the behavior of cobalt in water. The adsorption of cobalt by particulate matter decreases with decreasing pH, since the increasing H^+ concentration competes with metal binding sites. This may lead to increased concentrations of dissolved cobalt at low pH. The effect of Eh (redox potential) on the speciation of cobalt has been shown by the increase in the concentration of dissolved cobalt by orders of magnitude with increasing depth in certain parts of Baltic waters. The increase in the concentration of dissolved cobalt may be due to the formation of soluble bisulfide and polysulfide complexes in the anoxic zones. The residence time of soluble cobalt in seawater has been estimated to range from <1 to 52 years (Brugmann 1988; Knauer et al. 1982; Smith and Carson 1981).

Vitamin B_{12}, which contains cobalt, is synthesized by 58 species of seven genuses of bacteria as well as blue-green algae and actinomycetes (mold-like bacteria). Consequently, vitamin B_{12} levels in marine water range from very low levels in some open ocean water to much higher levels in some coastal waters. Freshwater environments have comparable levels of vitamin B_{12}. The high level of cobalamins in coastal water appears to be related to the occurrence of macrophytes in these areas with their high concentrations of vitamin B_{12}. Cobalamins are released into the water when the organisms die (Smith and Carson 1981).

Alkaline thermal groundwater in granitic areas have been studied as possible waste disposal sites for radioactive waste (Alaux-Negrel et al. 1993). Water in these areas is characterized by high pH, low CO_2 partial pressure, and generally low redox potential; sulfide concentrations are in the range of 10^{-4} to 10^{-3} mol/L. The solubility of cobalt is controlled by the solubility of CoS (log K_1 and log K_2 being 5.7 and 8.7 at 25°C) and therefore, levels of cobalt are very low, 10^{-8}–10^{-10} mol/L.

The ^{60}Co (III) picolinate complex that is released into water by some nuclear reactors does not break down immediately on release into seawater, but rather can coexist with the ^{60}Co (II) forms for lengthy periods in the environment (Leonard et al. 1993a, 1993b). Studies indicate that several processes occur to the Co(III) organic complexes, including reduction to the inorganic form, sorption of both species to particulate matter, and transformations of the uncomplexed species. It is possible that this more soluble and uncharged form of radioactive cobalt will increase the dispersion of ^{60}Co from its point of discharge.

6.3.2.3 Sediment and Soil

The speciation of cobalt in soil or sediment depends on the nature of the soil or sediment, concentration of chelating/complexing agents, pH, and redox potential (Eh) of the soil. Dissolved cobalt may be absorbed by ion exchange and other mechanisms, or may form complexes with fulvic acids, humic acid, or other organic ligands in soil. The humic and fulvic complexes of cobalt are not very stable compared with those of copper, lead, iron, and nickel. The speciation of cobalt in sediment from nine sites in the Red Sea, a sea that is unique in that it has no permanent streams flowing into it, was assessed using a sequential extraction technique (Hanna 1992). The mean percentages contained in the various fractions were: exchangeable, 5.5%; carbonate, 5%; Fe/Mn oxides, 24%; organic, 30.4%; sulfides, 13%; and lithogenous, 22%. While the mean concentration of cobalt in the sediment increased from 0.003 to 0.006 ppb between 1934 and 1984, its distribution among the different phases did not change appreciably.

The reduction of soil Eh, which may occur when soil is flooded or in deeper layers of soil that are oxygen-depleted, may change the speciation of cobalt. This may result in the reduction of soil iron and manganese and the subsequent release of adsorbed cobalt from the mineral oxides. Similarly, a decrease in soil pH may result in the solubilization of precipitated cobalt and desorption of sorbed cobalt, resulting in increased cobalt mobility (Smith and Carson 1981). Co^{2+} may also be oxidized to Co^{3+} by manganese oxides, a common component of soils and aquifer material, with subsequent surface precipitation (Brusseau and Zachara 1993). This process may affect transport of cobalt in the subsurface environment.

EDTA complexes of cobalt are very stable and are likely to form in soils containing EDTA. EDTA is widely used as a decontaminating agent at nuclear facilities. Although cobalt-EDTA complexes are adsorbed by some soils, the mobility of cobalt in soil may increase as a result of complex formation (Schnitzer 1969; Smith and Carson 1981; Swanson 1984). ^{60}Co that is disposed of in shallow land trenches have sometimes been found to migrate more rapidly than expected from the disposal sites. Organic chelating agents are frequently present at these sites and would possibly increase the solubility and transport of the radionuclide.

Bacterial action can affect the mobility of a substance by mediating reactions or by participating in reactions that lower the pH. Another way of influencing radionuclide mobility is by degrading complexing agents used in cleaning reactors (e.g., citric acid), thereby releasing the radionuclide. However, experiments on the fate and transport of cobalt released upon the biodegradation of the complexing ligand indicate that results are not always predictable; the means of ligand removal and the geochemical environment are important factors that must be considered (Brooks et al. 1998).

6.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

Cobalt concentrations in environmental media, including food and human tissue, have been exhaustively tabulated by Smith and Carson (1981) and Young (1979). The International Agency for Research on Cancer (IARC 1991) contains reviews of more recent studies, but is primarily focused on occupational exposures and body burdens of cobalt.

6.4.1 Air

Atmospheric cobalt is associated with particulate matter. Mean cobalt levels in air at unpolluted sites are generally <1–2 ng/m^3 (Hamilton 1994; Smith and Carson 1981). At the South Pole, cobalt levels of 0.00049±0.00015 ng/m^3 were recorded in 1974–1975 (Maenhaut et al. 1979). Geometric mean cobalt levels in several open-ocean environments ranged from 0.0004 to 0.08 ng/m^3 (Chester et al. 1991). The average annual PM-10 (particles with diameters ≤10 μm) cobalt concentration at Nahant, Massachusetts (near Boston) in 1992–1993 was 1.7 ng/m^3 (Golomb et al. 1997). Half of the cobalt was contained in fine particles (<2.5 μm) and half in coarse particles (2.5–10 μm). The mean cobalt level in southern Norway in 1985–1986 (n=346) was 0.10 ng/m^3 with 35% of the samples falling below the detection limit of 0.04 ng/m^3 (Amundsen et al. 1992). Atmospheric cobalt levels in industrial settings may exceed 10 ng/m^3. The highest recorded average cobalt concentration in air was 48 ng/m^3 in Clydach, Wales at the site, where nickel and cobalt were refined (Smith and Carson 1981). Some ambient atmospheric levels of cobalt are given in Table 6-2. These data show the contribution of anthropogenic sources in increasing the level of cobalt in the ambient air. Typical occupational cobalt levels are 1.0×10^4–1.7×10^6 ng/m^3 (Barceloux 1999; IARC 1991). While ^{60}Co has been detected in air samples at the Hanford site and Oak Ridge National Laboratories, levels were not reported (HazDat 2004; PNNL 1996). In 1995, the concentration of ^{60}Co in air at the Hanford site was below the detection limit in over 88% of the air samples.

6.4.2 Water

The concentrations of cobalt in surface water and groundwater in the United States are generally low, <1 μg/L in pristine areas and 1–10 μg/L in populated areas (Hamilton 1994; Smith and Carson 1981). However, cobalt levels may be considerably higher in mining or agricultural areas. Levels as high as

4,500 μg/L were reported in Mineral Creek, Arizona, near a copper mine and smelter; levels of 6,500 μg/L were reported in the Little St. Francis River, which receives effluent from cobalt mining and milling operations (Smith and Carson 1981). Mining at Blackbird Mine in Idaho, one of the large deposits of cobalt in North America, occurred from the late 1800s to 1982. Cobalt concentration in surface water and groundwater samples collected in 1992 from area creeks near this mine were reported to range from <1 to 625,000 μg/L, and from not detected to 315,000 μg/L respectively (ATSDR 1995), Eckel and Jacob (1988) analyzed U.S. Geological Survey (USGS) data for 6,805 ambient surface water stations and estimated the geometric mean and median dissolved cobalt concentration as 2.9 and 2.0 μg/L, respectively. Mean cobalt levels reported in seawater range from 0.078 μg/L in the Caribbean Sea to 0.39 μg/L in the Indian Ocean (Hamilton 1994). Vitamin B_{12} is synthesized by bacteria, macrophytes,

Table 6-2. Concentration of Cobalt in the Atmosphere

Location	Possible source/activity	Concentration[a]	Units	Type	Reference
Ambient levels—remote					
South Pole, 1974–1975	Crustal material	0.00049±0.00015	ng/m^3	Mean±SD	Maenhaut et al. 1979
Open-ocean		0.0004–0.08	ng/m^3	Geomean range	Chester et al. 1991
North Atlantic		0.006–0.09	ng/m^3	Range	Smith and Carson 1981
Baltic Sea, 1983		0.09, 0.01–0.43	ng/m^3	Mean, range	Hasanen etal. 1990
Remote sites		0.001–0.9	ng/m^3	Range	Schroeder et al. 1987
Ambient levels—rural/suburban/urban					
Rural sites		0.08–10.1	ng/m^3	Range	Schroeder et al. 1987
Massachusetts, Nahant, 1992–1993		1.7	ng/m^3	Annual mean	Golomb et al. 1997
Urban sites					Schroeder et al. 1987
United States		0.2–83	ng/m^3	Range	
Canada		1–7.9			
Europe		0.4–18.3			
Texas state average (1978–1982)		2.0	ng/m^3	Mean	Wiersema et al. 1984
Illinois, urban air (<2.5 μm; 2.5–10 μm)					Sweet et al. 1993
Bondville, Ill (rural)	Background	0.2; 0.1	ng/m^3	Mean (fine; coarse)	
Southeast Chicago	Steel mills	0.4; 0.4			
East St. Louis	Smelters	0.5; 0.4			
Washington, DC (1974)	Urban area	1.1	ng/m^3	Mean	Smith and Carson 1981
Ambient levels—industrial					
Maryland, Baltimore Harbor Tunnel (1973–1974)					Ondov et al. 1982
Air outside	Vehicular exhaust	0.8–1.9	ng/m^3	Range	
Air inside	Vehicular exhaust	2.2–5.3			
Ohio, Cleveland	Be-Cu alloy and other industrial activities	610	ng/m^3	Maximum	Smith and Carson 1981
Texas, El Paso (1978–1982)	Industrial	127	ng/m^3	Maximum	Wiersema et al. 1984

Table 6-2. Concentration of Cobalt in the Atmosphere

Location	Possible source/activity	Concentration[a]	Units	Type	Reference
Texas, Houston (1978–1982)	Urban area	81	ng/m^3	Maximum	Wiersema et al. 1984
Arizona, Tucson					Smith and Carson 1981
Urban	Copper smelting	1.9	ng/m^3	Mean	
Rural		0.7			
Maryland, Chalk Point Generator	Coal-burning power plant	3.86	ng/m^3	Mean	Smith and Carson 1981
Wales, Clydach	Nickel refining	48, 3–300	ng/m^3	Mean, range	Smith and Carson 1981
Wales, Llausamlet and Trebanos	Towns near Clydach	3.8		Mean	Smith and Carson 1981
Occupational air levels					
Northern Italy, exposure survey, 1991, area monitoring (n=259)	Diamond abrasive mfg.				Mosconi et al. 1994a
	Mould-filling	220, 47–960	ng/m^3	Median, range	
	Sintering	101.5, 32–240			
	Grinding	22, 15–45			
	Mechanical-working	20, 12–44			
	Grinding	5, 2.5–94			
	Tool production	6, 5–47			
	Hard metal alloy filing	2, 0.8–3			
	Other	2.7, 2.3–15			
Northern Italy, exposure survey, 1991, personal sampling (n=259)	Diamond abrasive mfg.				Mosconi et al. 1994a
	Mould-filling	382, 76–2,600	ng/m^3	Median, range	
	Sintering	309, 238–413			
	Grinding	230, 82–690			
	Mechanical-working	40, 7.1–65			
	Grinding	9.3, 1.5–178			
	Tool production	17, 4–28			
	Hard metal alloy filling	5, 1–107			
	Other	50, 10–290			

Table 6-2. Concentration of Cobalt in the Atmosphere

Location	Possible source/activity	Concentration[a]	Units	Type	Reference
Japan, personal sampling, hard metal tool manufacture, 8-hour TWA, 356 workers (n=935)	Powder preparation				Kumagai et al. 1996
	Rotation	459, 7–6,390	μg/m^3	Mean, range	
	Full-time	147, 26–378			
	Press				
	Rubber	339, 48–2,910			
	Steel	47, 6–248			
	Shaping	97, 4–1,160			
	Sintering	24, 1–145			
	Blasting	2, 1–4			
	Electron discharging	3, 1–23			
	Grinding	45, 1–482			

geomean = geometric mean; SD = standard deviation; TWA = time weighted average blue-green algae, and actinomycetes, and cobalt levels in oceans often correlate with biological productivity. In the Baltic Sea, dissolved cobalt levels that are 1.0 ng/L near the surface, increase to 71.0 ng/L at a depth of 200 m (Brügmann 1988). The rise in dissolved cobalt is coincident with the onset of anoxic conditions and the presence of hydrogen sulfide, indicating that soluble bisulfide and polysulfide complexes may be present. Some cobalt levels reported in water are given in Table 6-3.

In a 1962–1967 survey, cobalt was detected in 2.8% of 1,577 U.S. raw surface waters from which drinking water is derived; the detection limit was 1 μg/L and the maximum concentration was 48 μg/L (NAS 1977). Of 380 U.S. finished drinking waters, only 0.5% contained cobalt levels exceeding 1 μg/L; the maximum concentration found was 29 μg/L (NAS 1977). These values are higher than the respective median and maximum levels of <2.0 and 6.0 μg/L found in Canadian finished drinking water (Meranger et al. 1981). Meranger et al. (1981) tested source water and drinking water in 71 municipalities across Canada and concluded that, in general, both surface water and groundwater used for drinking water supplies contain negligible amounts of cobalt. Greathouse and Craun (1978) analyzed 3,834 grab samples of household tap water from 35 geographical areas in the United States for 28 trace elements. Cobalt was found in 9.8% of the samples at concentrations ranging from 2.6 to 107 μg/L. It is not clear whether these higher levels could indicate that cobalt was picked up in the distribution system. In the earlier National Community Water Supply Study (2,500 samples), 62% of the samples contained <1 μg Co/L; the average and maximum cobalt concentrations were 2.2 and 19 μg/L, respectively (Smith and Carson 1981). Cobalt was not detected (detection limit 8 μg/L) in a 1982–1983 survey of drinking water in Norway that covered 384 waterworks serving 70.9% of the Norwegian population (Flaten 1991).

The mean concentrations of cobalt in rain is around 0.03–1.7 μg/L, with levels generally ranging from 0.002 μg/L at Enewetak Atoll to about 2.9 μg/L in the Swansea Valley, Wales (Arimoto et al. 1985; Dasch and Wolff 1989; Hansson et al. 1988; Heaton et al. 1990; Helmers and Schrems 1995; Nimmo and Chester 1993; Nimmo and Fones 1997; Smith and Carson 1981). The highest recorded level of cobalt in precipitation was 68.9 μg/L in the vicinity of a nickel smelter in Monchegorsk in the Russian Arctic (Reimann et al. 1997). An analysis of rain in the Mediterranean and urban and coastal sites in northwest England showed that about 33–44% of the cobalt occurred as very stable dissolved organic complexes (Nimmo and Chester 1993; Nimmo and Fones 1997).

As it was pointed out in Section 6.3.2.2, ^{60}Co discharged from the Steam Generating Heavy Water Reactor at Winfrith on the south coast of England was shown to be largely in the form of the nonionic

Table 6-3. Cobalt Levels in Water

Nature/location of water	Level	Units	Type	Reference
Sea water				
Florida (Indian River Lagoon) (43 sites)	0.031, 50	µg/L	Mean, range	Trocine and Trefry 1996
California (Baja) 2–45 km offshore (n=11)	0.022–0.17	nM	Range	Sañudo-Wilhelmy and Flegal 1996
<100 m off shore (n=11)	0.11–0.59			
Agean Sea, 1994; 8 sites (dissolved)	0.168–0.632, 1.917	nM	Range of means, maximum	Voutsinou-Taliadouri 1997
Baltic Sea (Gotland Deep site)				Brügmann 1988
10 m	1.0	ng/L	Mean (dissolved Co)	
50 m	1.0			
100 m	3.5			
150 m	4.2			
200 m (anoxic)	71.0			
235 m (anoxic)	49.2			
Seawater background	0.04	µg/L		Bargagli 2000
Seawater	0.27	µg/L	Mean	Abbasi et al. 1989
Fresh surface water				
Freshwater background	0.05	µg/L		Bargagli 2000
U.S. ambient surface water (6,805 stations)	<2.9, 2.0	µg/L	Mean, median	Eckel and Jacob 1988
Five Great Lakes waters	ND–0.09	µg/L	Range	Rossmann and Barres 1988
Japan, unpolluted lake	<0.004	µg/L		Nojiri et al. 1985
Norway, 11 rivers	0.94	µg/L	Maximum	Flaten 1991
Streams near populated areas	1–10	µg/L	Range	Smith and Carson 1981
Streams in agricultural and mining areas	11–50	µg/L	Range	Smith and Carson 1981
Suspended solids in rivers	7–94	mg/kg	Range	Smith and Carson 1981
Groundwater				
Canada (Chalk River nuclear waste site)	0.0001–0.002	µg/L		Cassidy et al. 1982
Colorado (Denver)–shallow groundwater, (n=30)	<1 (<1–9)	µg/L	Median, range	Bruce and McMahon 1996

Table 6-3. Cobalt Levels in Water

Nature/location of water	Level	Units	Type	Reference
Drinking water				
Canadian drinking water (71 municipalities)				Meranger et al. 1981
Raw:	<2.0	μg/L	Median	
Treated:	<2.0			
Distributed:	≤2.0			
Precipitation				
Massachusetts, 1984 (12 events)	0.045 (0.008), 0.02–0.12	μg/L	Mean (SD), range	Dasch and Wolff 1989
Rhode Island (rain/snow), 1985 (n=269)	0.038 (0.067)	ppb	Median (mean)	Heaton et al. 1990
	0.001–0.80		Range	
Western Mediterranean, 1988–1989				Nimmo and Chester 1993
Total cobalt	0.029–0.134, 0.043	μg/L	Range, mean	
Labile cobalt	0.009–0.104, 0.025			
Organic cobalt	ND–0.613, 0.019			
Arctic (7 sites in Finland, Norway, Russia)	<0.02–1.07, 3.32	μg/L	Median range, maximum	Reimann et al. 1997
Russia (Monchegorsk), nickel smelter	11.8, 68.9		Median, maximum	

ND = not detected; SD = standard deviation trivalent complex, ^{60}Co(III) picolinate. The ^{60}Co(III) species is not immediately reduced to the more particle-reactive divalent form, and both oxidation states may coexist for long periods of time in the environment. The proportion of the more soluble and mobile ^{60}Co(III) would be expected to increase with time and distance from the point of discharge. Shoreline water samples (n=22) taken in 1987–1988 at two locations in the vicinity of the discharge from the Steam Generating Heavy Water Reactor at Winfrith contained 0.3–16.2 mBq/L (8–437 fCi/L) of particulate ^{60}Co, 2.8–44.4 mBq/L (76–1,200 fCi/L) of soluble ^{60}Co(II), and 0.2–4.8 mBq/L (5–130 fCi/L) of soluble ^{60}Co(III) (Leonard et al. 1993). The percent of the soluble ^{60}Co present as Co(III) ranged from 4.3 to 18.6%. In 1989, in conjunction with the largest discharge of effluent from the plant, offshore seawater samples from 18 sites contained 0.06–2.22 mBq/L (2–60 fCi/L) of particulate ^{60}Co, 0.30–10.3 mBq/L (8.1–278 fCi/L) of soluble ^{60}Co(II), and 0.12–1.55 mBq/L (3.2–41.9 fCi/L) of soluble ^{60}Co(III). The percent of the soluble ^{60}Co present as Co(III) ranged from 6.0 to 28.6%.

6.4.3 Sediment and Soil

Cobalt is the 33rd most abundant element in the earth’s crust. Its average concentrations in the earth's crust and in igneous rocks are 20–25 and 18 mg/kg, respectively (Abbasi et al. 1989; Merian 1985; Smith and Carson 1981). Trace metals in soils may originate from parent rock or from anthropogenic sources, primarily fertilizers, pesticides, and herbicides. Most soils contain 1–40 mg cobalt/kg. The average cobalt concentration in U.S. soils is 7.2 mg/kg (Smith and Carson 1981). Soils containing <0.5–3 mg cobalt/kg are considered cobalt-deficient because plants growing on them have insufficient cobalt (<0.08–0.1 mg/kg) to meet the dietary requirements of cattle and sheep. Cobalt-deficient soils include the humus podzols of the southeastern United States, and the podzols, brown podzolic soils, and humus groundwater podzols in the northeastern parts of the United States. (Podzols are generally coarse-textured soils.) The cobalt content of surface soils from 13 sites in the brown and dark brown soil zones of southwestern Saskatchewan ranged from 3.7 to 16.0 mg/kg and only in one case was the soil appreciably elevated above the corresponding parent material (Mermut et al. 1996). Fertilizers used in this agricultural area contained 0.12–102 mg Co/kg, with a median of 5.7 mg/kg.

Mean cobalt concentrations in surface soil from nine sites on two active volcanic islands off of Sicily ranged from 5.1 to 59.0 mg/kg (Bargagli et al. 1991). Soils near ore deposits, phosphate rocks, or ore smelting facilities, and soils contaminated by airport traffic, highway traffic, or other industrial pollution may contain much higher concentrations of cobalt; concentrations up to 800 mg/kg have been detected in such areas (Kloke et al. 1984; Smith and Carson 1981). Cobalt concentrations from 28 samples collected from surface deposits in the Big Deer and Blackbird Creek drainage basins in Idaho near the Blackbird Mine ranged from 26.5 to 7,410 mg/kg (Agency for Toxic Substances and Disease Registry 1995). Soils around the large copper-nickel smelters in Sudbury, Ontario have been shown to contain high levels of cobalt. Fifty kilometers from the smelters, cobalt levels in surface soil were 19 mg/kg. These levels increased to 48 mg/kg at 19 km, 33 mg/kg at 10 km, and 42–154 mg/kg between 0.8 and 1.3 km from the smelter (Smith and Carson 1981). Soils around a cemented tungsten carbide tool grinding factory contained cobalt levels as high as 12,700 mg/kg, almost 2,000 times the average in U.S. soils (Abraham and Hunt 1995). However, neighborhood soils between 30 and 160 meters from the factory only contained 12–18 mg Co/kg.

Unpolluted freshwater sediment contains about the same levels of cobalt as does cobalt-sufficient soil, generally <20 mg/kg (Smith and Carson 1981). In the Hudson River Estuary, cobalt levels in suspended sediment were an order of magnitude higher than in bottom sediment (Gibbs 1994). This can be attributed to the finer grain size of suspended sediment or local sources. Cobalt levels in core samples (surface to 42 cm deep) from the Upper St. Lawrence Estuary were independent of depth, indicating the lack of any recent significant anthropogenic releases (Coakley et al. 1993). Cobalt levels in sediment are shown in Table 6-4.

No broad-based monitoring studies of ^{60}Co or other radioactive cobalt isotopes in soil or sediment were found in the literature. Soil samples from the O-horizon taken from three sites in the 30-km zone around Chernobyl in 1992 and again in 1993 contained 14–290 and 4.5–245 Bq/kg (380–7,800 and 120–6,620 pCi/kg) dry weight of ^{60}Co, respectively (Lux et al. 1995). The Columbia River receives radiological contaminants along the Hanford Reach primarily through seepage of contaminated groundwater. The regional median concentration of ^{60}Co in sediment was highest along the Hanford reach, approximately 0.09 pCi/g (0.003 Bq/g) (PNNL 1996). ^{60}Co activity in a sediment cores in water off of Southampton in southern England contained up to 28 Bq/kg (760 pCi/kg) in the upper 3 cm; no activity was found below 12.5 cm (Croudace and Cundy 1995). Discharges of treated effluent occurred on closing a steam generating heavy water reactor west of where the sampling was done. The maximum discharge occurred in 1980–1981; however, no value was reported (Croudace and Cundy 1995).

Table 6-4. Cobalt Levels in Sediment

Nature/location of sediment	Level	Units	Type	Reference
Freshwater				
Polluted lakes and rivers	0.16–133	mg/kg	Range	Smith and Carson 1981
Lake Ontario near Miesissaqua, Canada	4.1–19.8	mg/kg	Range	Glooschenko et al. 1981
Hudson River, Foundry Cove, 1983, Ni-Cd battery plant, 1953–1979, surficial (0–5 cm) sediment, 16 sites	18–700	mg/kg	Range	Knutson et al. 1987
Estuaries and Marine				
Hudson River Estuary (0–80 km from ocean), 1991				Gibbs 1994
Bottom sediment	1–13	mg/kg	Range	
Suspended sediment (near surface)	30–140			
Upper St. Lawrence Estuary, 1989–1990				Coakley et al. 1993
Core C168	3.1 (0.6)	mg/kg	Mean (SD)	
Cores LE and LO	2.7 (0.5)			
Massachusetts, New Bedford Harbor- core (0–25 cm)				Shine et al. 1995
Outer Harbor	7.03, 3.64–9.79, range			
Inner Harbor	6.38, 2.62–10.52			
Buzzards Bay (control site)	4.76, 1.64–8.19			
Indian River Lagoon, Florida (43 sites)	2.3, 0.4–6.3	mg/kg	Mean, range	Trocine and Trefry 1996
Gulf of Mexico				Villanueva and Botello 1998
Coastal areas (11 sites)	12.30–36.26	mg/kg	Range of means	
Continental shelf (3 sites)	6.39–21.00			
Antarctica (Ross Sea) continental shelf (n=12)	19, 0.10–13	mg/kg	Mean, range	Bargagli 2000
Northern Arctic Alaska, continental shelf (n=136)	9, 3.3–18	mg/kg	Mean, range	Bargagli 2000
Chukchi Sea, northeast Alaska (31 stations, surficial sediment)	32.7, 19–74	mg/kg	Mean, range	Naidu et al. 1997
Baltic Sea, southern, off Poland (surficial sediment)	0.69–18.10	mg/kg	Range	Szefer et al. 1996
Baltic Sea (Gotland Deep site)	19, 11–33	mg/kg	Mean, range	Brügmann 1988

SD = standard deviation

Sediment samples were analyzed from the Peconic River system on Long Island, New York, downstream of Brookhaven National Laboratory (BNL). Near the sewage treatment plant, closest to the BNL, mean concentrations of ^{60}Co from three locations at the depth intervals 0.00–0.06, 0.06–0.15, 0.15–0.24, and 0.24–0.37 meters were 9.6, 6.7, 9.6, and 10.5 Bq/kg (0.25, 0.18, 0.25, and 0.28 nCi/kg) dry weight, respectively. At one location at the BNL property boundary, mean concentrations of ^{60}Co, using the same depth intervals, were 5.8 Bq/kg (0.16 nCi/kg) dry weight for the 0.00–0.06 m depth and <4 Bq/kg (<0.11 nCi/kg) dry weight for the remaining depth intervals. Sediment samples from a control river, Connetquot River, were <4 Bq/kg (<0.11 nCi/kg) in two locations at two depths (0.00–0.06 and 0.06–0.15 m) (Rapiejko et al. 2001).

Mururoa and Fangataufa Atolls were used for underground testing of nuclear weapons from 1975 to 1996. ^{60}Co was detected in the particle fraction of water in measurable levels at two of the nine Mururoa Atoll sites, Aristee and Ceto, at 0.58 and 1.06 mBq/L (0.016 and 0.029 pCi/L), respectively. ^{60}Co levels were found at levels below the detection limit, <0.1 mBq/L (<0.003 pCi/L), at the two Fangataufa Atoll sites and at the seven other Muruoa Atoll sites (Mulsow et al. 1999). Concentrations of ^{60}Co of soil samples used for growing onion, potatoes, tomatoes, cabbage, and maize in the Bulgarian village, Ostrov, in the vicinity (approximately 25–30 km) of the “Kozloduy” nuclear power plant were <8, 3, 320, 330, and 180 mBq/kg (2, 8.1, 8.6, 8.9, and 4.9 pCi/kg), respectively (Djingova and Kuleff 2002).

6.4.4 Other Environmental Media

The cobalt content of plants depends on the plant, the cobalt content of the soil, and numerous environmental factors. The mean cobalt concentration reported for terrestrial plants was 0.48 μg/g, while the mean and median levels for freshwater vascular plants were 0.48 and 0.32 μg/g, respectively (Outridge and Noller 1991). The median cobalt level in freshwater vascular plants from polluted waters was about the same as in unpolluted waters, 0.37 μg/g, although extremely high levels of cobalt, up to 860 μg/g, was reported in one species, *Myriophyllum verticillatum*, from central Ontario lakes. Grasses normally contain 0.2–0.35 μg/g of cobalt, but grasses from cobalt-deficient regions contain only 0.02–0.06 μg/g of cobalt (Hamilton 1994). Durum wheat grown in southeastern Saskatchewan contained 0.01–0.02 mg/kg dry weight (Mermut et al. 1996). In view of the cobalt content of the soil and the fact that almost half of the cobalt in fertilizers used in the area was in a readily available form, the uptake of cobalt by wheat was negligible.

^{60}Co levels in plants and mushrooms in the 30-km zone around Chernobyl were mostly below the detection limit in samples obtained in 1992 and 1993; the highest activity recorded was 3.9 Bq/kg (110 pCi/kg) dry weight in *Athyrium filix femina* (Lux et al. 1995).

Cobalt concentrations have been reported in various aquatic animals and seabirds. Eel and a freshwater fish from three Dutch polder lakes contained 2.5–25.0 and 2.50–5.63 mg cobalt/kg wet weight, respectively, (Badsha and Goldspink 1988). Muscle tissue of ocean fish and rock crabs caught near dump sites off New York City, New Haven, Connecticut, and Delaware Bay contained 10–40 and 16.0 μg/kg, respectively (Greig and Jones 1976). In a study of the levels and distribution of 14 elements in oceanic seabirds, the concentration of cobalt, an essential element, appeared to be highly regulated, with over 80% of the body burden residing in the skeleton. The mean cobalt concentration in the livers of 11 seabird species ranged from 0.048 to 0.078 μg/g dry weight, and cobalt had the lowest coefficient of variation in the different species of the elements studied (Kim et al. 1998a). In another study in Antarctica, mean cobalt levels in fish and amphipods were 0.11–0.14 and 1.01 μg/g dry weight, respectively, while those in the tissue of penguin and other sea birds ranged from 0.09 to 0.11 μg/g (Szefer et al. 1993). The concentration of cobalt in the tissue of 14 bluefin tuna caught by various commercial fishing vessels off Newfoundland was essentially the same, 0.01±0.004 μg/g (Hellou et al. 1992a). Similarly, in a broad survey of contaminant levels in nine species of fish and fiddler crabs from 11 sites in the lower Savannah River, Georgia and the Savannah National Wildlife Refuge, mean cobalt levels among different species and sites were statistically indistinguishable (Winger et al. 1990). These and other studies indicate that cobalt does not biomagnify up the food chain (Smith and Carson 1981). While high levels of cobalt were found in sediment from the Tigris River in Turkey and low levels in the water, cobalt was not detected in two species of fish, *Cyprinion macrostomus* and *Garra rufa* (Gümgüm et al. 1994). Cobalt was detected in two other species of fish collect between 1995 and 1996 in the upper Sakarya river basin, Turkey. Cobalt concentrations ranged from 0.038 to 0.154 μg/g dry weight for *Cyprinus caprio* and from 0.045 to 0.062 μg/g dry weight for *Barbus plebejus* (Barlas 1999).

^{60}Co was not detected in fish and mussel samples analyzed from the Peconic River system on Long Island, New York, downstream of the BNL. The lower detection limit for ^{60}Co was 0.4 Bq/kg (10 pCi/kg). ^{60}Co had been detected in sediment samples from this area (Section 6.4.3) (Rapiejko et al. 2001).

Some female birds sequester metals into their eggs under certain conditions, a phenomenon that may jeopardize the developing embryos. The geometric mean concentrations of cobalt in tern eggs collected from coastal New Jersey in 1971 and 1982 were 0.48 and 0.50 mg/kg, respectively. Unlike the levels of seven other common metals (e.g., mercury, cadmium, copper, lead, manganese, nickel, and zinc), the level of cobalt in tern eggs (and in the environment) showed no decline over the 11-year period (Burger and Gochfeld 1988).

Table 6-5 shows the levels of cobalt in food items and food categories from different countries. The level of cobalt in most Canadian foods was low; items with the highest concentrations in this study were waffles (0.076 μg/g), corn cereal (0.074 μg/g), and potato chips (0.070 μg/g) (Dabeka and McKenzie 1995). Green leafy vegetables and fresh cereals are the richest sources of cobalt (0.2–0.6 μg/g dry weight), while dairy products, refined cereals, and sugar contain the least cobalt (0.1–0.3 μg/g dry weight) (Barceloux 1999). The levels of cobalt were determined in 50 different food items, mainly meat, fish, fruit, vegetables, pulses, and cereals on the Swedish market during the years 1983–1990 (Jorhem and Sundström 1993). Beef liver and seeds were fairly high in cobalt and fish, fruit, and root and leafy vegetables were under 0.01 μg cobalt/g fresh weight. The cobalt levels in μg/g fresh weight were highest in alfalfa seeds, 0.86; linseed, 0.56; milk chocolate, 0.34; dark chocolate, 0.24; white poppy seeds, 0.30; blue poppy seeds, 0.15; soya beans, 0.084; green lentils, 0.054; and beef liver, 0.043. The cobalt content of 20 brands of alcoholic and nonalcoholic beer widely consumed in Spain ranged from 0.16 to 0.56 μg/L with a median of 0.39 μg/L (Cameán et al. 1998). Cobalt, which was at one time added to beer to increase the foam head, has been associated with cardiomyopathies (heart disease) in heavy beer drinkers.

A study of radionuclide levels in various foods and drinks in Hong Kong found that the ^{60}Co content in nearly all foods and drinks used in the study were below the minimal detection limit (Yu and Mao 1999). Analysis of wild plants in Bulgaria in villages near the “Kozloduy” nuclear power plant showed that the concentrations of ^{60}Co were below the detection limit. Mean activity concentrations of ^{60}Co in edible plants in this region were mostly <0.04 Bg/kg (<1 pCi/kg) (Djingova and Kuleff 2002).

Stable cobalt is present in various consumer products including cleaners, detergents, and soaps, which have resulted in dermatitis in sensitive individuals (Kokelj et al. 1994; Vilaplana et al. 1987). Tobacco contains about <0.3–2.3 μg Co/g dry weight and approximately 0.5% of the cobalt appears in mainstream smoke (Barceloux 1999; Munita and Mazzilli 1986; Ostapczuk et al. 1987; Stebbens et al. 1992).

Table 6-5. Cobalt Levels in Food

Food item	Level	Units[a]	Type	Reference
Infant formulas/milk				
Evaporated milk (n=21)	0.74, 0.52–2.6	μg/kg[b]	Median, range	Dabeka 1989
Ready-to-use formula (n=49)	0.53, 0.21–5.2	μg/kg[b]	Median, range	Dabeka 1989
Milk-based (n=33)	0.40, 0.21–0.99			
No added iron (n=6)	0.36, 0.21–0.61			
Added iron (n=27)	0.87, 0.41–0.99			
Soy-based (n=16)	2.27, 1.71–5.2			
Concentrated liquid formula (n=50)	2.27,0.25–11.8	μg/kg[b]	Median, range	Dabeka 1989
Milk-based (n=34)	1.57, 0.25–3.11			
No added iron (n=20)	1.06, 0.25–1.77			
Added iron (n=14)	2.59, 2.03–3.11			
Soy-based (n=16)	4.33, 2.7–11.8			
Powdered formula (n=64)	9.54, 2.6–53	μg/kg[b]	Median, range	Dabeka 1989
Milk-based (n=36)	4.96, 2.6–10.6			
No added iron (n=23)	4.24, 2.6–9.6			
Added iron (n=13)	8.26, 5.1–10.6			
Soy-based (n=28)	20.0, 10.6–53			
Agricultural crops				
Cabbage, United States	0.2	mg/kg[c]	Typical level	NAS 1977
Corn seed, United States	0.01	mg/kg[c]	Typical level	NAS 1977
Fruits, 12 types, Poland	0.01–0.02	mg/kg	Range	Bulinski et al. 1986
Lettuce, Sweden 1983–1990 (n=7)	0.002, 0.006	mg/kg	Mean, maximum	Jorhem and Sundström 1993
Lettuce, United States	0.2	mg/kg[c]	Typical level	NAS 1977
Onions, 11 Danish sites (n=110)	1.51, 0.119–5.1	μg/kg	Median, range	Bibak et al. 1998a
Peas, 10 Danish sites (n=93)	4.6, 0.57–17	μg/kg	Median, range	Bibak et al. 1998b
Potatoes, Sweden (n=8)	0.008, 0.017	mg/kg	Mean, maximum	Jorhem and Sundström 1993
Spinach, United States	0.4–0.6	mg/kg[c]	Typical range	NAS 1977
Strawberries, Sweden (n=10)	0.004, 0.010	mg/kg	Mean, maximum	Jorhem and Sundström 1993
Vegetables, 30 types, Poland	0.008–0.032	mg/kg	Range	Bulinski et al. 1986
White flour, United States	0.003	mg/kg[c]	Typical level	NAS 1977
Meat, fish, beverages				
Beef, Sweden (n=3)	0.001, 0.001	mg/kg	Range, maximum	Jorhem and Sundström 1993
Beef liver, Sweden (n=3)	0.043, 0.074	mg/kg	Range, maximum	Jorhem and Sundström 1993
Beef kidney, Sweden (n=3)	0.008, 0.010	mg/kg	Range, maximum	Jorhem and Sundström 1993

Table 6-5. Cobalt Levels in Food

Food item	Level	Units[a]	Type	Reference
Beer, Spain, 20 brands	0.39, 0.16–0.56	μg/L	Median, range	Cameán et al. 1998
Cocoa, Germany	1.31	mg/kg[c]		Ostapczuk et al. 1987
Coffee (whole), South Africa	0.93	mg/kg[c]		Horwitz and Van der Linden 1974
Coffee (whole), Germany (61% water extractable)	0.11–0.31	mg/kg[c]	Range	Ostapczuk et al. 1987
Fish, Sweden, 10 varieties (n=40)	<0.001–.008, 0.020	mg/kg	Range of mean, maximum	Jorhem and Sundström 1993
Pork, Sweden (n=36)	0.001, 0.012	mg/kg	Range, maximum	Jorhem and Sundström 1993
Pork liver, Sweden (n=36)	0.010, 0.023	mg/kg	Range, maximum	Jorhem and Sundström 1993
Pork kidney, Sweden (n=36)	0.004, 0.011	mg/kg	Range, maximum	Jorhem and Sundström 1993
Tea (whole), South Africa	0.2	mg/kg[c]		Horwitz and Van der Linden 1974
Tea (whole), Germany (40% water extractable)	0.18–6.7	mg/kg[c]	Range	Ostapczuk et al. 1987
Food categories				
Bakery good/ cereals, Canada (n=24)	10.9, 75.7	μg/kg	Median, maximum	Dakeba and McKenzie 1995
Beverages, Canada (n=7)	5.9, 9.1	μg/kg	Median, maximum	Dakeba and McKenzie 1995
Fats and oils, Canada (n=3)	<2.6, 37.6	μg/kg	Median, maximum	Dakeba and McKenzie 1995
Fish, Canada (n=6)	18.6, 14.3–29.4	μg/kg	Median, range	Dakeba and McKenzie 1995
Fruits and fruit juices, Canada (n=25)	<6.6, 35.7	μg/kg	Median, maximum	Dakeba and McKenzie 1995
Meat and poultry, Canada (n=18)	<5.5, 38.2	μg/kg	Median, maximum	Dakeba and McKenzie 1995
Milk and milk products, Canada (n=13)	<1.4, 18.9	μg/kg	Median, maximum	Dakeba and McKenzie 1995
Soups, Canada (n=4)	5.6, 8.5	μg/kg	Median, maximum	Dakeba and McKenzie 1995
Sugar and candy, Canada (n=7)	<0.4, 3.5	μg/kg	Median, maximum	Dakeba and McKenzie 1995
Vegetables, Canada (n=38)	2.4, 18.1	μg/kg	Median, maximum	Dakeba and McKenzie 1995

[a] Produce on a fresh weight basis, unless otherwise specified
[b] As sold
[c] Dry weight basis

The cobalt content of sewage sludge, incinerator ash, fertilizers, soil amendments, and other substances appears in Table 6-6. The concentration of cobalt in U.S. coal averages about 5 mg/kg, levels in crude oil and fuel oil are 0.001–10 and 0.03–0.3 mg/kg, respectively, and those in gasoline are <0.1 mg/kg (Smith and Carson 1981). Cobalt levels were below the detection limit of 0.05 ppm dry weight in all but 1 of 26 samples of composted yard waste, sewage sludge, and municipal solid waste samples nationwide in 1991. The one positive sample of composted yard waste contained 1.53 ppm of cobalt (Lisk et al. 1992).

6.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

Exposure of the general population to cobalt occurs through inhalation of ambient air and ingestion of food and drinking water. In general, intake from food is much greater than from drinking water, which in turn, is much greater than from air. From the limited monitoring data available, the average concentration of cobalt in ambient air in the United States is approximately 0.4 ng/m^3. However, levels may be orders of magnitude higher in source areas. Therefore, intake to cobalt in air will vary substantially from nonsource areas to areas with cobalt-related industries. Similarly, the median cobalt concentration in U.S. drinking water is <2.0 μg/L; however, values as high as 107 μg/L have been reported in surveys of water supplies (Smith and Carson 1981). Therefore, exposure from drinking water may vary considerably from one location to another. In Canada, the daily cobalt intake of the average adult from drinking water is ≤2.6 μg; this could increase to 10 μg for those living in areas with the highest cobalt levels (Meranger et al. 1981).

General population exposure to cobalt from food is highly variable and normally higher than intake from drinking water. Most of the cobalt ingested is inorganic; vitamin B_{12}, which occurs almost entirely in food of animal origin, constitutes only a very small fraction of cobalt intake. The cobalt intake in food has been estimated to be 5.0–40.0 μg/day (Jenkins 1980). The daily cobalt intake, including food, water, and beverages of two men that were followed for 50 weeks was much higher, 310 and 470 μg (Smith and Carson 1981). The estimated average daily cobalt intake from diet in Canada was 11 μg/day; the intake varied from 4 to 15 μg/day between the various age/sex groups (Table 6-7) (Barceloux 1999; Dabeka and McKenzie 1995). The contributions of various food groups to cobalt intake in this study were (category, contribution of dietary intake): bakery goods and cereals, 29.8%; vegetables, 21.9%; beverages, 9.8%; milk and milk products, 9.4%; meat and poultry, 9.1%; soups, 6.4%; fruit and fruit juices, 5.0%; sugar

Table 6-6. Cobalt Content of Miscellaneous Substances

Substance/source	Level	Units	Type	Reference
Bituminous coal used for power generation	6.4	mg/kg	Median	Rubin 1999
Coal, United States	~5	mg/kg	Mean	Smith and Carson 1981
Fly ash	~25	mg/kg	Mean	Smith and Carson 1981
MSW Incinerator ash, Mississippi				Buchholz and Landsberger 1995
Fly ash (n=30)	11.3–13.5	μg/g	Range	
Bottom ash (n=30)	65.2–90.3			
Combined ash (n=30)	24.8–30.5			
MSW Incinerator ash, United States, 1987				Mumma et al. 1990
Fly ash (n=5)	18.2–54.0	μg/g	Range	
Bottom ash (n=7)	13.5–35.1			
Combined ash (n=8)	11.2–43.4			
Compost, Toronto				Evans and Tan 1998
Residential compost	8.1, 3.2–12	mg/kg	Median, range	
Greenhouse finished compost	6.1±1.03		Mean ± SD	
Sewage sludge				
16 large U.S. cities	11.3, 6.08–29.1	mg/kg	Median, range	Gutenmann et al 1994
32 U.S. cities	7.2, 2.4–30.1	mg/kg	Median, range	Mumma et al. 1984
Cow manure (comparison)	6.1	mg/kg		Mumma et al. 1984
Miscellaneous soil amendments[a]				Raven and Loeppert 1997
Compost	3.55, 3.57	mg/kg	Individual means	
Diammonium phosphate	3.24, 0.68			
Dolomite	0.33			
Manure	2.23			
Monoammonium phosphate	0.78, 3.38			
Rock phosphate, Tilemsi	19.6			
Rock phosphate, North Carolina	<0.08			
Sewage sludge, Austinite	4.10			
Sewage sludge, Milorganite	4.07			
Triple superphosphate	6.61, 2.24			
Street dust, New York City	8.7–12.9	μg/g	Range	Fergusson and Ryan 1984

[a] The rest of the 24 fertilizers and soil amendments tested were below the detection limit (typically <0.07 ppm)

MSW = municipal solid waste; SD = standard deviation

Table 6-7. Mean Daily Dietary Intake of Cobalt for Selected Population Groups in Canada

Group	Mean daily intake (µg/day)
1–4 years	7
5–11 years	10
12–19 years; male	14
12–19 years; female	10
20–39 years; male	15
20–39 years; female	9
40–65 years; male	12
40–65 years; female	9
65+; male	10
65+; male	8

Source: Dabeka and McKenzie 1995

and candies, 2.8%; fish, 2.7%; fats and oils, 2.2%; and miscellaneous, 1.1%. The average daily intake of cobalt in France was estimated to be 29 μg/day (Biego et al. 1998). In this study, foods were divided into nine categories. The foods accounting for the greatest contributions of cobalt intake were milk and dairy products, fish-crustaceans, and condiments-sugar oil, respectively contributing 32, 20, and 16% to the daily intake. The U.S. Department of Agriculture (USDA) conducted a special exploratory study in 1985–1986 to determine the concentration of trace metals in tissue of health livestock and poultry randomly selected from those slaughtered. Between 0.6 and 5.9% of samples in the 11 production classes had levels of cobalt that exceeded the lowest reliable quantitation level of 0.15 ppm (0.15 mg/kg) and the mean of positive samples ranged from 0.20 to 0.23 ppm in all classes but heifer/steer, which had a level of 1.92 ppm (Coleman et al. 1992). Cobalt, which has been added to beer to increase the foam head, has been associated with cardiomyopathies (heart disease) in heavy beer drinkers. However, according to a recent Spanish study, the low levels of cobalt presently found in beer do not make a significant contribution to the total cobalt intake in heavy beer drinkers (Cameán et al. 1998). Smokers may be exposed to cobalt in mainstream smoke, but the level of exposure has not been assessed (Barceloux 1999).

Since cobalt and other heavy metals have been used on hand-painted china, a study was conducted to see whether these metals are released into food under acidic conditions. Forty-six samples of porcelain dinnerware from Europe or Asia that were manufactured before the mid-1970s and had hand-painted designs over the glaze were filled with 4% acetic acid to within 7 mm of the rim and analyzed after 24 hours (Sheets 1998). Of these, 36 samples released <0.02 μg/mL of cobalt and 10 released 0.020–2.9 μg/mL. The Food and Drug Administration (FDA) has not established dinnerware extraction limits for cobalt.

Data are lacking on the levels of cobalt in tissues and fluids of the general populations in the United States; values from various countries are given in Table 6-8. This table shows that cobalt concentrations are greatest in nail, hair, and bone. The differences in cobalt levels in similar human tissues (e.g., hair, nail) in different countries may be due to differences in dietary and living habits and levels of cobalt in food (Takagi et al. 1988). The total amount of cobalt in the body of an adult as vitamin B_{12} is about 0.25 mg, of which 50–90% in contained in the liver (IARC 1991).

A recent study in the United States determined the concentrations of trace metals in seminal plasma in industrial workers in a petroleum refinery, smelter, and chemical plant as compared with those of hospital

Table 6-8. Cobalt Levels in Human Tissues and Fluids

Tissue or fluid	Level	Units[a]	Type	Reference
Urine, U.S., NHANES, representative population (n=1007)	0.36, 0.11–0.89	μg/L	Geomean, 10th–90th percentile	CDC 2001
Urine, U.S., NHANES 1999–2000, Total, age 6 and older (n=2,465)	0.372, 0.130–1.32	μg/L	Geomean, 10th–95th percentile	CDC 2003
6–11 years (n=340)	0.498, 0.130–1.32			
12–19 years (n=719)	0.517, 0.200–1.52			
20 years and older (n=1,406)	0.339, 0.120–1.28			
Males (n=1,227)	0.369, 0.150–1.01			
Females (n=1,238)	0.375, 0.120–1.49			
Mexican Americans (n=884)	0.415, 0.130–1.47			
Non-Hispanic blacks (n=568)	0.433, 0.160–1.45			
Non-Hispanic whites (n=822)	0.365, 0.120–1.29			
Urine, The Netherlands	<0.2–1.2	μg/L	Range	Bouman et al. 1986
Urine, Sweden	0.5, 0.1–2.2	μg/L	Mean, range	Alexandersson 1988
Urine, Denmark (3 reference groups)				Poulsen et al. 1994
Unexposed control females (n=46)	1.5, LOD–20.5	nmol[b]	Mean, range	
Unexposed males (n=12)	0.9, LOD–2.31			
Unexposed females (n=11)	5.9, LOD–25.02			
Urine, hip arthroplasty patients, observed 7–15 years (n=17)	0.9–1.05	μg/L	Range	IARC 1991
Urine, hip arthroplasty patients, observed 5–15.5 years (n=10)	3.8	μg/L	Mean	IARC 1991
Urine, 48 metal sharpening workers in 12 Italian factories	0–40.3, 86	μg/L	Range of means, maximum	Imbrogno et al. 1994
Urine, 12 female cobalt powder sintering workers, Italy				Ferdenzi et al. 1994
Monday, before shift	25, 1–51	μg/L	Mean, range	
Friday, before shift	29, 3–159			
Friday, end-of shift	85, 6–505			
After 3-week holiday	11, 4–34			
Urine, Italian workers wet grinding of hard metal tools (end of shift)				Sesana et al. 1994
Factory A no local exhausts (n=3)	138.3 (108), 123.7 (74)	μg/L	Mean (SD) Monday, Friday	
Factory B local exhausts (n=5)	15.3 (7.7), 24.4 (14.1)			
Factory C local exhausts (n=3)	48.2 (7.3), 74.7 (13)			

Table 6-8. Cobalt Levels in Human Tissues and Fluids

Tissue or fluid	Level	Units[a]	Type	Reference
Urine, Northern Italy, 1991, occupational exposure survey, 314 exposed people				Mosconi et al. 1994
Diamond abrasive production				
Mould-filling	320, 587, 39–2,100	µg/L	Median, mean, range	
Sintering	168, 193, 02–390			
Grinding	61, 151, 34–520			
Mechanical-working	50, 67, 143–165			
Grinding	15, 32, 0.8–730			
Tool production	12, 19, 0.8–100			
Hard metal alloy filling	5, 5, 0.8–18			
Other	1, 2.9, 0.8–72			
Blood, Denmark, porcelain factory				Raffn et al. 1988
Plate painters, off work for 6 weeks (n=46)	8.05, 1.70–22.1	nmol/L	Mean, range	
Plate painters, working 4 weeks (n=46)	36.7, 3.40–407			
Top glaze painters (unexposed) (n=51)	4.04, <1.70–10.2			
Urine, Denmark, porcelain factory				Raffn et al. 1988
Plate painters, off work for 6 weeks (n=46)	81.8, <1.70–445	nmol/L	Mean, range	
Plate painters, working 4 weeks (n=46)	1,308, 37.4–14,397			
Top glaze painters (unexposed) (n=51)	16.0, <1.70–234			
Plasma, Sweden	0.1–1.2	µg/L	Range	Alexandersson 1988
Whole Blood, Denmark (3 Reference groups)				Poulsen et al. 1994
Unexposed control females (n=46)	4.1, <1.7–10.2	nmol/L	Mean, range	
Unexposed males (n=12)	3.1, <1.7–6.8			
Unexposed females (n=11)	7.6, <1.7–30.5			
Lung, Sweden				Gerhardsson et al. 1988
Rural	0.007	mg/kg	Mean	
Urban	0.011			
Liver Tissue, United Kingdom, newborns and infants that died from SIDS (n=157)	17.4±11.3 (15.9)	ng/g wet mass	mean±SD (median)	Patriarca et al. 1999
Liver, New Zealand (n=96)	0.120	mg/kg	Mean	IARC 1991

Table 6-8. Cobalt Levels in Human Tissues and Fluids

Tissue or fluid	Level	Units[a]	Type	Reference
Tissue, Japan				Yamagata et al. 1962
Pectoral muscle	0.016	mg/kg	Mean	
Rib bone	0.036			
Stomach	0.021			
Liver	0.017			
Brain	0.0055			
Urinary bladder	0.0055			
Kidney	0.012			
Aorta	0.021			
Nails				Takagi et al. 1988
Canada (n=40)	0.09	mg/kg	Mean	
India (n=100)	0.06			
Japan (n=252)	0.17			
Poland (n=49)	0.04			
U.S. (n=71)	0.06			
Adipose tissue	0.035–0.078	mg/kg	Range	EPA 1986
Hair				Takagi et al. 1986
Canada (n=92)	0.043	mg/kg	Mean	
India (n=255)	0.051			
Japan (n=457)	0.18			
Poland (n=46)	0.022			
United States (n=55)	0.047			
Hair, Italy				Vienna et al. 1995
Male biology students (n=20)	0.007, 0.001–0.07	mg/kg	Geomean, range	
Female biology students (n=20)	0.017, 0.001–0.28			
Hair, Pakistan				Ashraf et al. 1995
Rural (n=28)	2.05, 0.10–4.80	mg/kg	Mean, range	
Urban (n=39)	3.86, 1.10–5.90			

[a] fresh weight, unless otherwise specified
[b] creatinine basis

geomean = geometric mean; LOD = limit of detection; NHANES = Nation Health and Nutrition Examination Survey; SD = standard deviation; SIDS = sudden infant death syndrome workers (control group). There were four groups each with 50 adult men. The mean cobalt concentrations (μg/dL), including standard errors, were determined to be 31±2 (hospital workers), 25±0.8 (metal ore smelter workers), 19±0.6 (petroleum refinery workers), and 22±1 (chemical workers) (Dawson et al. 2000).

Surgical implants for knee and hip replacements often use cobalt-containing alloys, which may lead to elevated cobalt levels in body fluids. Indeed, cobalt levels in serum and urine have been used as an index of prosthesis wear. In some cases, significant increases in cobalt levels have been observed, while in other cases, elevations were much lower or only sporadic (IARC 1991). These differences have been ascribed to greater release rates from metal to metal than metal to polyethylene articular surfaces as well to differences in the cobalt-containing alloys.

There are several reports of cobalt exposure among occupational groups. The concentrations of cobalt in the air of hard metal manufacturing, welding, and grinding factories may range from 1 to 300 μg/m^3, compared to normal atmospheric levels of 0.4–2.0 ng/m^3 (Burr and Sinks 1989; Haddad and Zikovsky 1985; Koponen et al. 1982; Lichtenstein et al. 1975). The maximum OSHA permissible level is 100 μg/m^3. The concentration of cobalt in the dust of an electric welding factory was 4.2 μg/g compared to its normal dust level of 0.1–1.0 μg/g (Baumgardt et al. 1986). The higher rate of exposure to cobalt for occupational groups is also reflected in the higher cobalt content in tissues and body fluids of living and deceased workers in this group. The levels of cobalt in the urine of workers in the hard metal industry varied with the levels of cobalt concentration in the working atmosphere. At a concentration of 0.09 mg/m^3, the urinary excretion of cobalt exceeded normal values by orders of magnitude. When the cobalt concentration in the working atmosphere was 0.01 mg/m^3 or lower, urinary cobalt excretion was 4–10 times higher than normal level (Alexandersson 1988; Scansetti et al. 1985). At high exposure levels, the cobalt concentration in blood was 20 times higher than normal; in the low exposure group, it was only slightly higher than in the control group (Alexandersson 1988).

An extensive survey of workers potentially exposed to cobalt in the Bergamo Province in northern Italy in 1991 identified 403 exposed workers in different production areas (Mosconi et al. 1994a). Significant cobalt exposure occurred especially for operators working in diamond abrasive production, and in particular, in mold filling and sintering units where environmental limits are regularly exceeded. Exposure in tool production, tool sharpening, and hard metal alloy filling is much more restrained.

Occupational cobalt air levels and urinary excretion levels recorded in the survey appear in Tables 6-2 and 6-8.

Several studies of cobalt concentrations in air in the hard metal industry have been reported. In the hard metal industry in Japan, Kumagai et al. (1996) found that mean 8-hour time weighted averages (TWAs) of airborne cobalt were >50 μg/m^3 for workers involved in powder preparation (rotation), powder preparation (full-time), rubber press, and shaping operations; mean atmospheric concentrations were 459, 147, 339, and 97 μg/m^3, respectively. Workers involved in the manufacture and maintenance of hard metal and stellite blades in Finland were exposed to breathing zone cobalt concentrations ranging from 2 to 240 μg/m^3, with a geometric mean of 17 μg/m^3 (Linnainmaa et al. 1996). The average proportion of water soluble cobalt in airborne cobalt was 68% (range 14–100%). Wet grinding was not sufficient to adequately control cobalt levels and coolant cobalt levels were high. In a group of 12 factories in Italy in which 48 workers were tested who had been exposed to cobalt in operations such as sharpening with diamond grinding stones, the mean concentration of cobalt in air was 21.2 and 137.7 μg/m^3 (Permissible exposure limit [PEL]-TWA 100 μg/m^3) in work places with and without dust ventilation, respectively (Imbrogno et al. 1994).

Urine concentrations have been used to monitor workers' exposure to airborne cobalt. Ferdenzi et al. (1994) obtained a correlation between Friday TWA air cobalt levels and Friday end-of-shift urine levels among women in the powder sintering industry. Median urinary cobalt concentrations were 25 (range: 1–51) and 29 (3–159) μg/L, on Monday and Friday before the shift, respectively, and 85 (6–505) μg/L on Friday after the shift. Imbrogno and Alborghetti (1994) evaluated the levels of occupational exposure to cobalt during dry and/or wet hard metal sharpening. The mean urine cobalt level in the workers in 12 factories was found to range from 0 to 40.3 μg/L and the maximum was 86 μg/L. The average urinary cobalt level among workers using wet/mixed sharpening methods was 4 times higher than those using dry sharpening methods; 21.38 μg/L as compared to 5 μg/L, respectively. Gallorini et al. (1994) found that the ratio of inorganic to organic cobalt in the urine of hard metal workers was 2.3 compared to 1.01 in controls; the ratio was constant over the range of urinary cobalt levels analyzed (180–1,254 μg/L). Exposure to cobalt during the wet grinding of hard metal tools (Widia tools) used in the wood industry produced exposure to cobalt above the PEL-TWA of 100 μg/m^3 (Sesana et al. 1994). However, exhausts near the grinding wheels were shown to substantially reduce exposure levels (see Table 6-8). In the processing department of a small company producing carbide tip saw blades for the woodworking industry, area air sampling showed that exposure levels were low in all departments except tip grinding where wet and dry tip grinding areas contained 55 and 21 $\mu g/m^3$ of cobalt, respectively, for the total collection method (Stebbins et al. 1992). For the method collecting respirable particles, cobalt levels ranged from 2 to 28 $\mu g/m^3$. Wet grinding is a traditional method for controlling dust during grinding. However, some coolants may contain significant concentrations of cobalt (in this case, 61–538 mg/mL) that can contribute to exposure during grinding (Stebbins et al. 1992). Among cobalt blue dye plate painters in a porcelain factory in Denmark, the blood and urine cobalt levels were, respectively, 2–4 and 5–15 times higher than in control groups (Raffn et al. 1988). Similarly, lungs taken from deceased, occupationally exposed workers also had higher levels of cobalt than lungs from control groups. Lungs of deceased hard metal industry workers in Sweden contained 2.5–4 times higher levels of cobalt than control lungs (Gerhardsson et al. 1988). Similarly, the lungs of coal miners from England contained 6 times higher cobalt levels than control lungs (Hewitt 1988).

Exposure to radioactive cobalt can occur through various means. Workers at nuclear facilities, irradiation facilities, or nuclear waste storage sites may be accidentally exposed to radioisotopes of cobalt. Also, workers using cobalt isotopes in tracer studies, in calibration or other devices, or ^{57}Co in Mössbauer spectroscopy, may be exposed to radioactive cobalt. Exposure would generally be to radiation produced by these isotopes (e.g., gamma radiation from ^{60}Co). Patients receiving ^{60}Co radiotherapy will obviously be exposed to its radiation. According to the USNRC (1999), the collective intake of ^{60}Co by ingestion and inhalation at power reactors in 1998 was 352 μCi (13 MBq) for 25 intake records and 27,000 μCi (1,000 MBq) for 281 intake records (USNRC 1999). The collective intake at fuel fabrication facilities was 0.486 μCi (0.180 MBq) for 502 intake records. The USNRC occupational inhalation annual limits of intake (ALIs) for ^{60}Co are 200 μCi (7.4 MBq) for all compounds, except oxides, hydroxides, halides, and nitrates, and 30 μCi (1.1 MBq) for compounds of oxides, hydroxides, halides, and nitrates (USNRC 2001k).

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).

As with adults, most children are exposed to cobalt largely through their diet. Dabeka and McKenzie (1995) estimated that the dietary cobalt intake by Canadian children ages 1–19 ranged from 7 to 14 mg/day (see Table 6-7). Milk constitutes a larger part of children's diets than that of adults, and infants may consume infant formula. Cobalt concentrations ranging from 0.3 to 0.8 ng/g in cow's milk were reported by Iyengar (1982). The levels of cobalt in human milk from Nigeria, Zaire, Guatemala, Hungary, Philippines, and Sweden ranged from 150 (Hungary) to 1,400 ng/g (Philippines), median 320 ng/g (Nriagru 1992). Garg et al. (1993) reported much lower cobalt levels in three samples of human milk in India, 2.42 ng/g, and reported a cobalt concentration of 5.07 ng/g in cow's milk in India. Dakeba (1989) determined cobalt levels in various infant formulas (see Table 6-5). Milk-based infant formulas and evaporated milk contained <1 ng/g of cobalt on a "ready-to-use" basis. Milk-based formulas with added iron contained about twice the cobalt as those with no added iron and soy-based formulas contained about 5 times more cobalt. The influence of added iron suggests that the cobalt in formula is not primarily from vitamin B_{12}. Using literature values of cobalt in food, Dakeba also estimated that infants 0–12 months old ingest an average of 0.52 μg Co/kg-day (3.93 μg/day) from food and water and that for an infant, 0–12 months old, the total dietary cobalt intake would range from 0.42 μg/kg-day (3.39 μg/day) for a breast or milk-based formula fed infant to 1.0 μg/kg-day (7.33 μg/day) for an infant fed soy-based formula powder. The recommended dietary allowance for Canadian infants is 0.012 μg/day cobalt as vitamin B_{12}. In a 1967 study of the total dietary intake of some trace elements, excluding drinking water, of institutionalized children aged 9–12 in 28 U.S. cities, cobalt intake ranged from 0.297 to 1.767 mg/day with a mean value of 1.024 mg/day (Murthy et al. 1971).

Exposure to stable cobalt in communities near mining and smelting facilities or metal shops where cobalt is used in grinding tools is a public health concern, especially for infants and children. Since cobalt remains in the surface soil indefinitely and long past land uses may be forgotten, people may not realize that they are living in areas where high levels of cobalt may occur in soil. Contaminated soils pose a particular hazard to children because of both hand-to-mouth behavior and intentional ingestion of soil that contain metals and other contaminants (Hamel et al. 1998). In these communities, cobalt may have been tracked in from outdoors and contaminate carpeting. Cobalt-containing dust may be brought home in the clothing of parents working in industries where they are exposed to cobalt. Children may be exposed to this cobalt while crawling around or playing on contaminated carpeting. Exposure may also result from dermal contact with soil, or by inhaling dust and then swallowing it after mucociliary transport up out of the lungs. Because there is little absorption of cobalt through the skin following dermal exposure, and because much of the cobalt in soil is embedded in or adsorbed to soil particles or insoluble, it may not be in a form accessible for uptake by the body, and therefore may not pose a serious health hazard.

6.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

In addition to workers in the hard metal industry (tool production, grinding, etc.) and industries such as coal mining, metal mining, smelting and refining, cobalt dye painters, and the cobalt chemical production, the general population living near these industrial sites may be exposed to high levels of cobalt in air and in soil. Exposure to cobalt during the wet grinding of hard metal tools is especially high when local exhausts are not in use (Sesana et al. 1994). People living near hazardous waste sites may be exposed to cobalt by inhaling dust from contaminated sites or through dermal contact with cobalt-contaminated soil. In the case of children playing in and around unrestricted landfill sites, exposure via dermal and ingestion routes is possible. The general populations in agricultural areas that use sewage sludge or cobalt-containing fertilizers or other soil amendments may be exposed to higher levels of cobalt via inhalation of dust or dermal contact with the soil. However, no experimental evidence of higher than normal exposures for these population groups was found in the literature. People who live in areas that naturally contain higher levels of cobalt minerals may also be exposed to higher levels of cobalt from both the inhalation and dermal contact routes.

The higher exposure of cobalt in patients with cobalt-chromium knee implants has been demonstrated by the slightly higher levels of cobalt in whole blood, serum, and urine, and by very high levels of cobalt in bone of these patients (IARC 1991; Ostapczuk et al. 1985; Sunderman et al. 1989). Prosthetic devices that contain polyethylene components to avoid metal-to-metal contact do not appear to cause elevated levels of cobalt in tissues and body fluids (IARC 1991; Ostapczuk et al. 1985; Sunderman et al. 1989). People who use cobalt supplements as a treatment for anemia and those who take large amounts of vitamin B-12 as a dietary supplement would have higher intakes of cobalt than the general population.

Workers at nuclear facilities and nuclear waste storage sites may be exposed to potentially high levels of radiation exposure from ^{60}Co and ^{58}Co. Workers at irradiation facilities using ^{60}Co may be exposed to potentially high levels of gamma radiation exposure from this isotope. Patients receiving ^{60}Co radiotherapy will intentionally be exposed to high levels of gamma radiation.

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 cobalt 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 cobalt.

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, Chemical and Radiological Properties. As can be seen from Table 4-2 and Section 4.2, the relevant physical and chemical properties of cobalt and its compounds are sufficiently known to enable prediction of environmental fate and transport of cobalt compounds (Budavari 1996; Lide 1994; Stokinger 1981; Weast 1985). Information on the radiological properties of important cobalt isotopes are also well known (see Table 4-3) (ICRP 1983; Lide 1994). No data needs were identified.

Production, Import/Export, Use, Release, and Disposal. Information on the production, import/export, use, release, and disposal of a chemical is important because it is an indicator of possible environmental contamination and human exposure. Large releases and consumer use would indicate higher general population exposure from environmental sources (e.g., air, drinking water, and food) and use of consumer products. Occupational exposure may also increase with increased production and use. U.S. production of cobalt is derived primarily from scrap (secondary production). Information is available on cobalt consumption derived from secondary production, import/export, and release of cobalt from the National Defense Stockpile (USGS 1998, 1999, 2002). However, production volumes of individual cobalt compounds are not available and information on the production of individual compounds would be useful in assessing exposure to specific cobalt compounds. Radioactive cobalt isotopes, primarily ^{60}Co and ^{57}Co, are not commercially produced in the United States, but rather are imported from Canada and the United Kingdom; consumption amounts are not available. Information on the uses of cobalt is available (Cobalt Development Institute 2004; Donaldson 1986; Hodge 1993; IARC 1991; Richardson 1993; USGS 1998, 2002). Cobalt-containing products are mostly used in the workplace, although some consumer products contain cobalt.

According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit chemical release and offsite transfer information to the EPA. The TRI for 2001 is currently available (TRI01 2004). Starting in 1998, metal mining, coal mining, electric utilities, and RCRA/solvent recovery industries were required to report to the TRI. These sectors include those contributing greatest environmental releases of cobalt and cobalt compounds, giving us a much more complete picture of cobalt releases to the environment. The TRI also contains information on the onsite and offsite disposal and management of wastes (e.g., recycling, treatment, transfer to publicly owned treatment works [POTWs]). EPA guidelines address the disposal of hazardous cobalt wastes. The TRI database will be updated yearly and provides a list of industrial production facilities and emissions. The TRI data should be used with caution since the 1987 data represent first-time reporting by these facilities. Only certain types of facilities were required to report. This is not an exhaustive list.

Environmental Fate. There are data that permit assessment of the environmental fate and transport of cobalt in water and soil (Section 6.3). Frequently, sediment and soil are the ultimate sinks for cobalt; however, this process is dynamic, and cobalt can be released into the water depending upon conditions. There is a paucity of data in the literature regarding the chemical forms of cobalt released to the atmosphere and their transformations in air and this information would facilitate the determination of the transport and persistence of cobalt in the atmosphere. Additional data elucidating the mode of speciation of cobalt in water and soil would also be desirable. For example, under what circumstances Co(III) compounds might be formed in the environment and how long.

Bioavailability from Environmental Media. Absorption by the inhalation and oral routes in humans has been studied, but the results vary considerably (see Section 3.5.1) (Foster et al. 1989; Harp and Scoular 1952; Sedlet et al. 1958; Sorbie et al. 1971; Valberg et al. 1969). These variations were attributed to differences in the types and doses of cobalt compounds given, to the nutritional status of the subjects following oral exposure, and to particle size differences following inhalation exposure. Additional data assessing the absorption of cobalt following soil ingestion by children may be helpful. Data in animals are plentiful for both inhalation and oral routes and correlate well with the human data (Andre et al. 1989; Bailey et al. 1989; Collier et al. 1989; Kreyling et al. 1986; Patrick et al. 1989; Talbot and Morgan 1989). Data in animals following dermal exposure suggested that cobalt is not absorbed well through intact skin, but is rapidly taken up through damaged skin. Data regarding the bioavailability of cobalt following dermal exposure are important because dermal exposure to cobalt in the workplace is probable.

Food Chain Bioaccumulation. Bioaccumulation in the food chain is important in assessing the human exposure to cobalt from the consumption of food. Data are available that indicate that cobalt is not taken up appreciably by plants and does not biomagnify up the food chain (Baudin and Fritsch 1987; Baudin et al. 1990; Boikat et al. 1985; Francis et al. 1985; Kloke et al. 1984; Lux et al. 1995; Mascanzoni 1989; Mejstrik and Svacha 1988; Mermut et al. 1996; Palko and Yli-Hala 1988; Smith and Carson 1981; Tolle et al. 1983; Watabe et al. 1984).

Exposure Levels in Environmental Media. Monitoring data on levels of cobalt in air, water, and food permits the estimation of exposure from these sources. Data are available on the cobalt levels in ambient air (Golomb et al. 1997; Hasanen et al. 1990; Schroeder et al. 1987; Smith and Carson 1981; Sweet et al. 1993; Wiersema et al. 1984). However, the data are not sufficiently recent or broad-based for estimating the current levels of exposure to cobalt in the general U.S. population and particularly those living near cobalt-containing hazardous waste sites. In addition, in only isolated studies was there an assessment of the concentration of cobalt associated with coarse and fine particles (Sweet et al. 1993) or an average annual level obtained at a site (Golomb et al. 1997). Similarly, levels of cobalt in ambient water, while generally low, are also not sufficiently broad-based or recent to be satisfactory (Bargagli 2000; Bruce and McMahon 1996; Cassidy et al. 1982; Eckel and Jacob 1988; Flaten 1991; Nojiri et al.

1985; Rossmann and Barres 1988; Smith and Carson 1981). This deficiency may be satisfied when the EPA's improved and updated STORET database becomes available. Cobalt levels in Canadian drinking water are ≤2.0 mg/L (Meranger et al. 1981). However, U.S. drinking water levels have not been reported and would be useful. The levels of cobalt in sediment are available (Bargagli 2000; Coakley et al. 1993; Gibbs 1994; Glooschenko et al. 1981; Knutson et al. 1987; Naidu et al. 1997; Shine et al. 1995; Smith and Carson 1981; Trocine and Trefry 1996; Villanueva and Botello 1998), but more data on levels in soil and in the vicinity of industrial and hazardous waste sites would be useful. Few data on the levels of cobalt in U.S. foods are available, although studies from Canada and Sweden are available that indicate that cobalt levels in food items are generally low (Barceloux 1999; Dabeka and McKenzie 1995; Jorhem and Sundström 1993). In particular, total diet studies of cobalt in U.S. food is lacking. A Canadian total diet study estimated average daily cobalt intake to range from 7 to 15 μg/day for different age-sex groups (Dabeka and McKenzie 1995).

Few data are available on levels of ^{60}Co and other cobalt isotopes in environmental media.

Exposure Levels in Humans. The levels of cobalt in hair, nail, and adipose tissues of the general U.S. population are known (EPA 1986; Takagi et al. 1986, 1988). No reliable data on the levels of this substance in blood (or plasma) and urine of the general U.S. population were found, although such data are available for certain European populations including occupationally-exposed groups (Table 6-8). These data may be important for establishing the background exposure level of cobalt. No data on the levels of cobalt in any body tissue or fluid for populations living near hazardous waste sites are available. Such data would be important in assessing the exposure levels of this group of people.

Exposures of Children. Dabeka (1989) reported the levels of cobalt in various formulas and milk products consumed by children in Canada, and Dabeka and McKenzie (1995) determined the mean dietary intake of Canadian children as young as 1–4 years of age. Nriagru (1992) reported levels of cobalt in human milk from several countries. No analogous U.S. studies were found. Cobalt levels is the tissue and body fluids of children have not been found.

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

Exposure Registries. No exposure registries for cobalt were located. This compound is not currently one of the compounds for which a subregistry has been established in the National Exposure Registry. The compound 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 the exposure to cobalt and its compounds.

6.8.2 Ongoing Studies

The Federal Research in Progress (FEDRIP 2002, 2004) database provides additional information obtainable from a few ongoing studies that may fill in some of the data needs identified in Section 6.8.1. These studies are summarized in Table 6-9.

Remedial investigations and feasibility studies conducted at the NPL sites known to be contaminated with cobalt, such as the Blackbird Mine in Idaho, will add to the available database on exposure levels in environmental media, exposure levels in humans, and exposure registries, and will increase the current knowledge regarding the transport and transformation of cobalt in the environment.

The Cobalt Development Institute (CDI) is implementing a research program to assess environmental risks posed by the manufacture and use of cobalt and cobalt compounds. Studies that are underway include the assessment of seasonal and background variability of cobalt compounds in aquatic environment and a literature survey for existing data on the effects of cobalt and cobalt compounds in soils and sediment. Environmental studies proposed for 2002 included the assessment of seasonal and background variability of cobalt compounds in soils and sediments and a literature survey for existing data on the effects of cobalt and cobalt compounds on marine environments.

Table 6-9. Ongoing Studies on Cobalt

Investigator	Affiliation	Research description	Sponsor
Hamilton, JW	Dartmouth College, Hanover, New Hampshire	The overall goal of the Dartmouth Superfund Basic Research Program (SBRP) Project, Toxic Metals in the Northeast: From Biological to Environmental Implications is to determine the impact of toxic metals found at waste sites, including Superfund sites on human health and the environment. The program-wide focus of this research program is on toxic metals, particularly on arsenic, and also chromium, nickel, cadmium, mercury, cobalt, and lead.	NIH
Jones, BT	Wake Forest University, Winston-Salem, North Carolina	The investigators developing a novel, low-cost, portable instrument for the simultaneous determination of trace radioactive elements in nuclear forensic samples. At issue is the routine, inexpensive sampling for radioactivity that could be released on transport or storage of potential "dirty bomb" material. The instrument to be developed is expected to provide analytical figures comparable to inductively coupled plasma mass spectrometry, but the instrument is much lower cost and more portable. The specific objectives of the project include determination of the analytical figures of merit for elements including cobalt, cesium, and strontium, and analysis of real samples such as soil, urban dust, water and agricultural materials.	NSF
Kpomblekou-Ademawou, K Ankumah, RO	Tuskegee University, Tuskegee, Alabama	This project will investigate if excessive accumulation of some trace elements, added to poultry diet and excreted through feces, affects nitrogen transformation in broiler (chicken) litter amended soils and if this compromises safe food and feed production. The goals of this work are (1) to study the effects of concentrations of key trace elements (e.g., As, Cd, Co, Cr, Cu, Mn, Ni, Pb, Se, and Zn) found in broiler litter on nitrogen transformation in litter amended soils, (2) to assess the effects of temperature on the nitrogen transformation in the presence of trace elements and (3) to assess the fate of trace elements in sudax (Sorghum bicolor) grown in trace element-enriched broiler litter amended soils.	USDA
Longnecker, M	NIEHS, NIH	Evaluate the use of toenail levels as a measure of exposure by analyzing toenail and whole-diet homogenates by neutron activation analysis. Toenails reflect exposure over a longer period of time than do blood or urine measures, and are less likely to be influenced by contamination than hair.	NIEHS

Table 6-9. Ongoing Studies on Cobalt

Investigator	Affiliation	Research description	Sponsor
Saito, MA	Woods Hole Oceanographic Institution, Woods Hole, MA	This research will examine the influence of cobalt and cadmium speciation on *Synechococcus* and *Crocosphaera* at two sites in the Pacific Ocean. In addition, the distribution of cobalt across transects in the Eastern Equatorial Pacific will be determined to improve understanding of the global biogeochemical cycle of cobalt.	NSF
Tavlarides, LL	Syracuse University, Syracuse, New York	This work will be towards the development of sol-gel synthesis methods for organo-ceramic adsorbants for the extraction of toxic and valuable metal ions, such as cobalt, chromium, and arsenic ions from aqueous streams.	NSF

NIEHS = National Institute of Environmental Health Sciences; NIH = National Institute of Health; NSF = National Science Foundation USDA = U.S. Department of Agriculture; USDOE = U.S. Department of Energy

7. ANALYTICAL METHODS

The purpose of this chapter is to describe the analytical methods that are available for detecting, measuring, and/or monitoring cobalt, its metabolites, and other biomarkers of exposure and effect to cobalt. The intent is not to provide an exhaustive list of analytical methods. Rather, the intention is to identify well-established methods that are used as the standard methods of analysis. Many of the analytical methods used for environmental samples are the methods approved by federal agencies and organizations such as EPA and the National Institute for Occupational Safety and Health (NIOSH). Other methods presented in this chapter are those that are approved by groups such as the Association of Official Analytical Chemists (AOAC) and the American Public Health Association (APHA). Additionally, analytical methods are included that modify previously used methods to obtain lower detection limits and/or to improve accuracy and precision.

7.1 BIOLOGICAL MATERIALS

Entry of cobalt and its radioisotopes into the human body can be gained through ingestion, inhalation, or penetration through skin. The quantities of cobalt within the body can be assessed through the use of bioassays that are comprised of either *in vivo* and/or *in vitro* measurements. *In vivo* measurements can be obtained through techniques that directly quantitate internally deposited cobalt using, for example, whole body counters. These *in vivo* measurement techniques are commonly used to measure body burdens of cobalt radioisotopes (i.e., ^{60}Co), but cannot be used to assess the stable isotope of cobalt (^{59}Co). Instead, *in vitro* measurements provide an estimate of internally deposited cobalt (both the stable and radioactive isotopes), utilizing techniques that measure cobalt in body fluids, feces, or other human samples. Examples of these analytical techniques are given in NRCP Report No. 87 (1987) and are also listed in Tables 7-1 and 7-2.

7.1.1 Internal Cobalt Measurements

In vivo measurement techniques are the most direct and widely used approach for assessing the burden of cobalt radioisotopes within the body. The *in vivo* measurement of these radioisotopes within the body is

Table 7-1. Analytical Methods for Determining Stable Cobalt in Biological Materials

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Urine	Direct injection	GF-AAS with Zeeman background correction	0.3 μg/L	101% at 40μg/L	Bouman et al. 1986
	Addition of magnesium nitrate and nitric acid matrix modifiers and equal volume dilution of sample with water	GF-AAS with Zeeman background correction	2.4 μg/L	107.6% at 50 μg/L	Kimberly et al. 1987
	Sample chelated with dithiocarbamic acid derivative, solvent extracted	GF-AAS with Zeeman background correction	0.1 μg/L	No data	Alexandersson 1988; Ichikawa et al. 1985
	Sample wet digested with acid and chelated with 2,3-butanedion dioxide and complex preconcentrated at hanging mercury drop electrode	DPCSV	0.2 μg/L	No data	Heinrick and Angerer 1984
	Direct injection	GF-AAS with Zeeman background correction	0.1 μg/L	No data	Sunderman et al. 1989
Whole blood	Sample diluted with a homogenizer	GF-AAS with D_2 background correction	2 μg/L	No data	Heinrick and Angerer 1984
	Sample wet digested with acid and chelated with 2,3-butanedion dioxine and complex preconcentrated at hanging mercury drop electrode	DPCSV	0.8 μg/L	No data	Heinrich and Angerer 1984
	Sample acid digested, complexed with thiocyanate and N-phenylcinnamohydroxamic acid and extracted into ethyl acetate	Colorimetric	0.15 mg/L	No data	Afeworki and Chandravanshi 1987
Serum	Direct injection	GF-AA with Zeeman background correction	0.02 μg/L	No data	Sunderman et al. 1989

Table 7-1. Analytical Methods for Determining Stable Cobalt in Biological Materials

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Blood or tissue	Acid digestion	ICP-AES (NIOSH method 8005)	10 μg/g (blood); 0.2 μg/g (tissue)	81% at 110 μg/L (blood)	NIOSH 1984

D_2 = deuterium; DPCSV = differential pulse cathodic stripping voltammetry; GF-AAS = graphite furnace atomic absorption spectrometry; ICP-AES = inductively coupled plasma-atomic emission spectrometry; NIOSH = National Institute for Occupational Safety and Health

Table 7-2. Analytical Methods for Determining Radioactive Cobalt in Biological Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit[a]	Percent recovery	Reference
Urine	Direct count of sample	γ-spectrometry with NaI detector	No data (<MDL)	No data	Miltenberger et al. 1981
Soft tissue	Sample wet-ashed	γ-spectrometry (NaI)	No data	No data	Baratta et al. 1969
	Sample directly counted in detector	γ-spectrometry	5 pCi/g	No data	Rabon and Johnson 1973
	Sample digested in acid, oxidized with $HClO_4$, concentrated by precipitation with AMP, purified by resin column, precipitated with hexachloroplatinic acid	-counter	0.1 pCi/g	40–85%	Nevissi 1992
Feces	Direct count of sample	γ-spectrometry	No data	No data	Smith et al. 1972
Blood	Red cells separated from plasma and washed	γ-spectrometry with NaI detector	No data	No data	Smith et al. 1972

[a] 1 Bq=2.7×10^{-11} Ci=27 pCi

AMP = ammonium molybdophosphate; MDL = minimum detectable level; NaI = sodium iodide performed with various radiation detectors and associated electronic devices that are collectively known as whole body counters. These radiation detectors commonly utilize sodium iodide (NaI), hyperpure germanium, and organic liquid scintillation detectors to measure the 1,172 and 1,332 keV gamma rays from the decay of ^{60}Co. Because of the relatively low attenuation of the high energy gamma rays emitted from ^{60}Co by most tissues, cobalt radioisotopes can easily be detected and quantified using whole body counting techniques (Lessard et al. 1984; NCRP 1987; Raghavendran et al. 1978; Smith et al. 1972; Sun et al. 1997). Many configurations of the whole body counter and scanning methods have been utilized, ranging from unshielded single-crystal field detectors to shielded, multi-detector scanning detectors (IAEA 1962, 1970, 1972, 1976, 1985; NCRP 1987). Where appropriate, shielding of the room that houses the whole body counter and/or the detector is often used to increase the detection sensitivity of the equipment by minimizing background radiation. Additionally, care must be exercised to insure that external contamination with radioactive cobalt or other gamma-emitting radioisotopes on the clothing or skin of the individual to be scanned has been removed. Also, *in vitro* measurements of cobalt (see Section 7.1.2) are often used in conjunction with whole body counting when monitoring individuals working with cobalt, especially in conjunction with the assessment of individuals who have experienced accidental exposures to cobalt (Bhat et al. 1973).

Calibration of whole body counters is achieved through the use of tissue-equivalent phantoms. These phantoms are constructed to mimic the shape and density of the anatomical structure using tissue equivalent materials such as water-filled canisters or masonite (Barnaby and Smith 1971; Bhat et al. 1973; Sun et al. 1997). For example, the bottle mannequin absorber (BOMAB) consists of a series of water-filled polyethylene canisters constructed into seated or reclined human forms (Sun et al. 1997). ^{60}Co standards are measured either as point sources along the phantom or dissolved within the water-filled canisters. Comparisons of the actual counts obtained from the phantom to the known activity of the cobalt standards are used to determine the efficiency of the counting technique and, thus, provide the basis for calibrating the technique. Even so, differences in whole body measurement techniques, calibration methods, and background radiation count calculations between different laboratories can complicate the direct comparisons of body burden measurements and clearance rates for cobalt radioisotopes and should be taken into consideration when comparing data obtained from independent laboratories.

7.1.2 External Measurements

In vitro analyses of cobalt are routinely performed in situations where *in vivo* analyses can not be obtained or in support of an *in vivo* monitoring program. Urine and feces are the preferred samples for *in vitro* analyses of cobalt, although other sample types, such as tissue, bone, or blood, can also be used on a more limited basis. Urine provides for an analysis of soluble (inorganic) cobalt, fecal analysis can be used to assess the cobalt (organic) that is eliminated into the gut or the fraction of ingested cobalt not absorbed by the gut, and tissue/blood/bone are used to assess whole or regional body burdens of cobalt (NCRP 1987; Smith et al. 1972).

The analytical methods for determining the stable cobalt isotope, ^{59}Co, in biological matrices are given in Table 7-1. For accurate determination of cobalt, contamination of samples during sample collection, storage, and treatment must be avoided, particularly for biological samples containing low levels of cobalt. Cobalt contamination in blood samples has been reported from disposable syringes and technical-grade anticoagulants. Menghini needles, often used for liver biopsy, and mortar, pestles, and grinding devices used for homogeneous mixing may contaminate samples. Other sources of contamination may be collection and storage containers and chemical reagents used for preparing samples. In fact, sample contamination was responsible for erroneous reports in the earlier literature of grossly high levels of cobalt in biological specimens of unexposed persons. Therefore, blanks should always be run with the samples.

The commonly used classical methods for determining stable cobalt in biological samples are polarographic and colorimetric methods. Details about these methods are given by Saltzman and Keenan (1957). Since these older methods have interference problems and are unsuitable for determining low levels of cobalt in many biological samples, the samples are pretreated before quantification. Precipitation, chelation, chromatography, and ion-exchange are some of the methods used for this purpose. In recent years, the two single-element instrumental techniques most frequently used methods for determining cobalt are graphite furnace-atomic absorption spectrometry (GF-AAS) (also called electrothermal atomic absorption spectrometry) and differential pulse anodic stripping voltammetry (DPAVS). Multi-element techniques commonly used for cobalt determination are neutron activation analysis and inductively coupled plasma-atomic emission spectrometry (ICP-AES). Several other methods are available for determining stable cobalt in biological samples; these include x-ray fluorescence and Spark source mass spectrometry (Adeloju et al. 1985; Smith and Carson 1981).

For the *in vitro* analysis of cobalt radioisotopes in human samples, the majority of the analytical methods measure the cobalt radioisotopes directly in the samples, without the requirement for an extensive sample preparation procedure, using gamma spectrometry techniques. Of the cobalt radioisotopes that have been detected in the environment (e.g., ^{57}Co, ^{58}Co, and ^{60}Co), ^{60}Co is the most common. Consequently, most of the analytical methods that will be described in this chapter are those developed for the detection and quantitation of ^{60}Co in biological (see Table 7-2) and environmental samples (see Table 7-4).

The radiochemical analysis of ^{60}Co in urine has been used in conjunction with whole body scanning methods to assess acute and long-term body burdens of this isotope. The analysis of ^{60}Co in urine is the same as that described for a standardized method of analysis of cesium radioisotopes in urine (Gautier 1983). A urine sample of approximately 2 L is collected (either over 24 hours or before and after bedtime) and a 1-L aliquot is transferred to a Marinelli beaker for counting in a gamma-ray spectrometer (Gautier 1983). This simple procedure offers high recoveries of cobalt (98%) and the minimum detection sensitivity (100 pCi/L [3.7 Bq/L]) that is required to evaluate individuals for exposures to radioactive cobalt (Gautier 1983). Direct counting methods are also used for the analysis of cobalt radioisotopes in tissues, feces, and blood (Smith et al. 1972, Table 7-2). However, some of these methods may require sample preparation to reduce volume or increase concentration.

Accuracy of *in vivo* and *in vitro* measurements of cobalt is determined through the use of standard, certified solutions or radioactive sources with known concentrations or activities of cobalt. Certified standards for stable cobalt can be obtained through a number of commercial sources. The primary source of certified cobalt radioisotope standards is the National Institute of Standards and Technology (NIST). Gamma ray point sources for ^{60}Co (SRM 4200, 60,000 Bq [1.6 μCi] and SRM 4207, 300,000 Bq [56 μCi]) and standard solutions of ^{60}Co (SRM 4233, 600,000 Bq/g [16 μCi/g]) are available from NIST. Also, the determination of accuracy of a method often requires standard reference materials (SRMs). Unfortunately, very few biological SRMs are available. An SRM for cobalt in animal muscle is available from the International Atomic Energy Agency (IAEA), Vienna; an SRM for bovine liver (SRM-1577) is available from NIST (formerly the National Bureau of Standards) (Adeloju et al. 1985; Smith and Carson 1981).

7.2 ENVIRONMENTAL SAMPLES

There are two common approaches for measuring cobalt in the environment. Cobalt radioisotopes can either be measured directly in the field (*in situ*) using portable survey instruments or samples can be procured from the field and returned to the laboratory for quantitation. However, quantitation of the stable cobalt isotope ^{59}Co in environmental samples is generally conducted in the laboratory.

7.2.1 Field Measurements of Cobalt

In situ measurement techniques are extremely useful for the rapid characterization of radionuclide contamination in the environment, such as soils, sediments, and vegetation, or when monitoring personnel for exposure to radionuclides. The measurement of gamma-ray-emitting radionuclides, like cobalt, in the environment is conducted with portable survey instruments such as Gieger-Mueller detectors, sodium iodide scintillation detectors, and gamma-ray spectrometers. However, the use of gamma-ray spectrometers in field survey equipment is preferred for measuring cobalt in the field because of its selectivity and sensitivity. The relatively high energy and penetrability of the gamma ray that is emitted during the decay of ^{60}Co provides an advantage for assessing the level of cobalt both on and below the surface using portable field survey instruments such as the gamma-ray spectrometer. These gamma-ray spectrometers are equipped with a high purity germanium detector that is able to selectively and sensitively differentiate the 1,173 and 1,332 keV gamma rays emitted from ^{60}Co from the gamma-rays emitted from other radionuclides, for example ^{40}K or ^{137}Cs (USNRC 1997). Minimum detectable activities (MDAs) of 0.005 Bq/g (0.05 pCi/g) for ^{60}Co are routinely achieved using p-type germanium gamma-ray spectrometers with 10-minute counting times (USNRC 1997). However, counting errors can occur where the simultaneous detection of the 1,173 and 1,332 keV gamma rays produces a sum peak at 2,505 keV or a count in the continuum between the individual peaks and the sum peak (APHA 1998; USNRC 1997). These errors can be minimized by changing the geometry of the detector or the distance of the detector from the source of radioactivity. Computational methods have been derived to aid in determining the concentrations and distributions of ^{60}Co in different soil types and depths (USNRC 1997). The concentrations and distributions of ^{60}Co that have been derived from the computational analysis of the survey data are often verified by laboratory-based analyses of soil samples procured from the survey area.

7.2.2 Laboratory Analysis of Environmental Samples

Analytical methods for quantifying stable cobalt and cobalt radioisotopes in environmental samples (e.g. air, water, soil, and biota) are summarized in Tables 7-3 (^{59}Co) and 7-4 (^{60}Co). The methods that are commonly used in the analysis of stable cobalt are based on instrumental analytical techniques, such as atomic absorption spectrometry (AAS), instrumental neutron activation analysis (INAA), and mass spectrometry (MS). The analysis of ^{60}Co can be determined either as total mass or total activity, depending on the analytical technique that is used. Typically, radiochemical methods of analysis employing gamma-ray spectrometry techniques are used to quantitate ^{60}Co in environmental samples.

Analytical methods for determining stable cobalt in environmental samples are given in Table 7-3. Since cobalt exists in the particulate form in the atmosphere, it is sampled by drawing air through a metal-free filter (usually cellulose ester membrane), and the metal is quantified in the collected particles. Sample treatment prior to quantification is important for environmental samples. For example, the use of sodium carbonate for dry ashing plant materials results in poor cobalt recovery. Low-temperature ashing may be inadequate for some samples, and losses may occur during rigorous dry ashing. Wet ashing is the preferred method when sample treatment is necessary. Wet extraction with dilute nitric acid is most suitable for analyzing cobalt in dust samples. In some samples, the determination of soluble and insoluble cobalt is important, and analytical methods used to determine cobalt in filtered and unfiltered samples are available for this purpose.

As in the case of biological samples, contamination of environmental samples during sample collection, storage, and treatment should be avoided. Loss of cobalt from aqueous samples due to adsorption on storage containers should be avoided by using polyethylene or similar containers and acidifying the solution to the proper pH (Smith and Carson 1981). Because of its rapidity, accuracy, and low detection limit, GF-AAS with Zeeman background correction is the most commonly used method for quantifying cobalt in environmental samples. To meet the detection limits of the available analytical methods, preconcentration prior to quantification may be necessary for some samples (e.g., seawater). A few commonly used methods for determining cobalt in environmental samples are given in Table 7-3. Other less frequently used methods are inductively coupled plasma-mass spectrometry (ICP-MS) (Henshaw et al. 1989; McLaren et al. 1985), gas, liquid, and ion chromatography with colorimetric, electron capture, and electrochemical detection (Bond and Wallace 1984; Carvajal and Zienius 1986; Cheam and Li 1988; King and Fritz 1987; Schaller and Neeb 1987), photoacoustic spectroscopy with colorimetry (Kitamori

Table 7-3. Analytical Methods for Determining Stable Cobalt in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Air (workplace)	Weighed filter irradiated in a reactor	INAA	0.17 μg/m^3	No data	Haddad and Zikovsky 1985
	Sample filter digested by wet acid ashing	Flame-AAS with background correction (NIOSH method 7027)	0.4 μg/m^3	98% with 12–96 μg spiked filter	NIOSH 1984
	Sample filter digested by wet acid ashing	ICP-AES (NIOSH method 7300)	0.5 μg/m^3	95–100% with 2.5–1,000 spiked filter	NIOSH 1984
Water (low ionic strength)	Direct injection	GF-AAS with Zeeman or deuterium background correction	<0.5 μg/L	93–115% at 8.5–30 μg/L	Fishman et al. 1986
Lake water	Sample complexed with 8-hydroxyquinoline absorbed on a column, desorbed and digested with acid	ICP-AES	<0.004 μg/L	No data	Nojiri et al. 1985
Rainwater	Sample preconcentrated onto polystyrene films by spray-drying	PIXE	0.08 μg/L	No data	Hansson et al. 1988
Seawater	Sample complexed with 8-hydroxyquinoline absorbed on a column, desorbed and digested with acid	GF-AAS with Zeeman background correction	0.0002 μg/L	90%	Nakashima et al. 1988
Water and waste water	Direct aspiration of sample	Flame-AAS (EPA method 219.1)	0.05 mg/L	97–98% at 0.2–5.0 mg/L	EPA 1983
	Direct injection	GF-AAS with background correction (EPA method 219.2)	1 μg/L	No data	EPA 1983
Groundwater or leachate	Direct aspiration	Flame-AAS with background correction (EPA method 7200)	0.05 mg/L	97–98% at 0.2–5.0 mg/L	EPA 1986b
Groundwater or leachate	Direct injection	GF-AAS with background correction (EPA method 7201)	1 μg/L	No data	EPA 1986b

Table 7-3. Analytical Methods for Determining Stable Cobalt in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Food	Sample digested with acid	GF-AAS with background correction	1.88 μg/L in dissolved extract	100–107% at 0.2–0.6 mg/kg (leaves, liver)	Barbera and Farre 1988
Milled Wheat	Wet ashing (HNO_3), preconcentration and chelation	ET-AAS	20 ng/L	approximately 100%	González et al. 2000

AAS = atomic absorption spectrometry; EPA = Environmental Protection Agency; ET-AAS = electrothermal atomic absorption spectrometry; GF-AAS= graphite furnace atomic absorption spectrometry; ICP-AES = inductively coupled plasma-atomic emission spectrometry; INAA = instrumental neutron activation analysis; NIOSH = National Institute for Occupational Safety and Health; PIXE = photon induced x-ray emission

Table 7-4. Analytical Methods for Determining Radioactive Cobalt in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit[a]	Percent recovery	Reference
Air	Direct count of sample collected on paper filter	γ-spectrometry with Ge/Li detector	0.001 pCi/m^3	No data	USAEC 1974a
Air	Sample filter ashed	Scintillation counter with NaI detector	No data	No data	De Franceschi et al. 1974
Drinking water	Direct count of sample	γ-spectrometry with Ge detector	<2 pCi/L	99%	APHA 1998
Drinking water	Direct count of sample	γ-spectrometry	2 pCi/L	No data	USAEC 1974b
Water	Direct count of sample	γ-spectrometry with Ge/Li detector	2 pCi/L	No data	ASTM 1999
Water	Direct count of sample	γ-spectrometry	10 pCi/L	No data	Cahill et al. 1972
Seawater	Sample concentrated using continuous-flow coprecipitation-flotation separation technique	Scintillation detector	50 fCi/L	92–95%	Hiraide et al. 1984
Sediments	Sample dried and ground	γ-spectrometry	0.04 pCi/g	No data	Cahill et al. 1972
Fish	Samples dried and ashed	γ-spectrometry	0.001 pCi/g (DW)	No data	Cushing et al. 1981
Mollusc	Samples dried and ashed	γ-spectrometry	<0.01 pCi/g	No data	De Franceschi et al. 1976

[a] 1 Bq=$2.7\text{x}10^{-11}$ Ci=27 pCi

DW = dry weight; Ge/Li = lithium drifted geranium; NaI = sodium iodide et al. 1986), electrothermal vaporization with ICP-AES (Malinski et al. 1988) and chemiluminescence with spectrofluorimetry (Jones et al. 1989).

Analytical methods for determining cobalt radioisotopes in the environment are shown in Table 7-4. The analysis of cobalt in air is based on quantifying cobalt within aerosols or particles that become trapped on cellulose (paper) or glass fiber filters after a calibrated amount of air is passed through the filters. Since the cobalt radioisotopes do not occur naturally, but may be released as a result of nuclear weapons testing (which has been discontinued for several years), neutron-activation of specific materials (e.g., cobalt containing alloys used in piping of nuclear reactors), or a severe core damage accident in a nuclear plant, the amounts of these isotopes within the ambient environment are near or below the minimum detectable levels for these isotopes (DOE 1995). However, trace amounts of ^{60}Co can be detected in air, water, and sediments within or near nuclear weapons or fuel production facilities, nuclear reactors, and nuclear waste storage sites (DOE 1995; Boccolini et al. 1976; USAEC 1973). Analysis of cobalt radioisotopes in air filters, water, sediments, vegetation, and biota can be performed directly using gamma-ray spectrometry, or following some sample preparation (e.g., drying, ashing, or extraction) (Boccolini et al. 1976; Cahill et al. 1972; Cushing 1981; Hiraid et al. 1984; Windham and Phillips 1973).

The detection limits, accuracy, and precision of any analytical methodology are important parameters in determining the appropriateness of a method for quantifying a specific analyte at the desired level of sensitivity within a particular matrix. The Lower Limit of Detection (LLD) has been adopted to refer to the intrinsic detection capability of a measurement procedure (sampling through data reduction and reporting) to aid in determining which method is best suited for the required sample quantitation (USNRC 1984). Several factors influence the LLD, including background, size or concentration of sample, detector sensitivity and recovery of desired analyte during sample isolation and purification, level of interfering contaminants, and, particularly, counting time. Because of these variables, the LLDs between laboratories, utilizing the same or similar measurement procedures, will vary.

The accuracy of a measurement technique in determining the quantity of a particular analyte in environmental samples is greatly dependent on the availability of standard reference materials. Several SRMs for cobalt in environmental samples are also available. Some of these are coal, fly ash, diet, and orchard leaf SRMs available from NIST. The Community Bureau of Reference, European Communities offers SRMs for cobalt in sludges, and an SRM for cobalt in thin polymer films is available from NIST for x-ray fluorescence analysis in aerosol particle samples (Dzubay et al. 1988; Miller-Ihli and Wolf

1986; Schramel 1989; Smith and Carson 1981; Tinsley et al. 1983). Gamma ray point sources for ^{60}Co (SRM 4200, 60,000 Bq [1.6 µCi] and SRM 4207, 300,000 Bq [56 µCi]) and standard solutions of ^{60}Co (SRM 4233, 600,000 Bq/g [16 µCi/g]) are available from NIST.

7.3 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 cobalt 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 cobalt.

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.

7.3.1 Identification of Data Needs

Methods for Determining Biomarkers of Exposure and Effect. Cobalt concentrations in blood or urine can serve as exposure indicator (Alexandersson 1988; Ichikawa et al. 1985; Scansetti et al. 1985). The available analytical methods are capable of determining the levels of cobalt in both the blood and urine of normal and occupationally exposed persons (Table 7-1). For the quantitation of cobalt radioisotopes, whole body counters can be used to assess radioactive cobalt body burdens that have occurred both from acute and chronic exposures to cobalt radioisotopes (Bhat et al. 1973; NCRP 1987). *In vitro* analytical methods are available for analyzing cobalt radioisotopes in urine, feces, and tissues obtained from normal and occupationally exposed persons (Table 7-2).

Sensitive serum protein responses were found in animals exposed to cobalt at levels below those that produce hematopoietic effects. This unique serum protein response to cobalt exposure includes an increase in alpha globulin fractions of serum proteins and associated serum neuraminic acid. Details of this effect are given in Chapters 2 and 3. If similar changes occur in humans, this measurement may provide the earliest indications of effects of cobalt exposure. The available analytical methods are capable of determining these effects of cobalt exposure.

Methods for Determining Parent Compounds and Degradation Products in Environmental Media. Analytical methods with good sensitivity and specificity are available for determining cobalt in air, water, soil, and other environmental media (Table 7-3). Analytical methods for cobalt, like those for most metals, measure total metal content rather than the particular compound. Therefore, analytical methods do not generally differentiate between the parent compound and a transformation product as would be the case, for example, were cobalt oxide to be converted to cobalt sulfate. (An exception to this would be the case of radioactive decay in which the parent could be readily distinguished from the decay product.) Analytical methods with the capability of distinguishing between different cobalt species would be important an important tool for assessing the fate of cobalt compounds in the environment. However, methods for quantifying specific cobalt compounds were not found in the literature.

The levels of the parent compound or its reaction products in different environmental media can be used to assess the exposure to cobalt by humans through the inhalation of air and ingestion of food and drinking water. In the case of cobalt, a correlation between its levels in environmental media (e.g., occupational air) and in biological tissues and body fluids has been found (Alexandersson 1988; Ichikawa et al. 1985; Scansetti et al. 1985). Therefore, it is possible to estimate the total body burden of cobalt in workers exposed to airborne cobalt vapor and fumes from its concentration in workplace air.

For cobalt radioisotopes, analytical methods also exist that have good sensitivity and specificity for determining radioactive cobalt in air, water, soil, and other environmental media are available (Table 7-4). Because ^{60}Co decays to the stable element ^{60}Ni, there is no need to develop methods to detect and quantify the decay products.

7.3.2 Ongoing Studies

Two studies involving analytical techniques for cobalt was listed in the Federal Research in Progress database (FEDRIP 2002, 2004). N.J. Miller-Ihli and co-workers of the Agricultural Research Service in Beltsville, Maryland are developing single and multielement methods for the determination of trace elements of nutritional and health concern. This work will develop new/improved methods permitting direct analysis of solids by GF-AAS and ICP-MS, as well as methods for the determination of different chemical forms of these elements by coupling capillary zone electrophoresis with inductively coupled plasma mass spectrometry (ICP-MS). This research is supported by the U.S. Department of Agriculture (USDA) Agricultural Research Service. B.T. Jones of Wake Forest University in Winston-Salem, North Carolina along with C. Calloway of Winthrop College, South Carolina, are working to develop a novel, low-cost, portable instrument for the simultaneous determination of trace radioactive elements in nuclear forensic samples. The instrument to be developed is expected to provide analytical figures comparable to ICP-MS, but the instrument is much lower cost and more portable. The specific objectives of the project include determination of the analytical figures of merit for elements including cobalt, cesium, and strontium, and analysis of real samples such as soil, urban dust, water, and agricultural materials.

8. REGULATIONS AND ADVISORIES

International and national guidelines and state regulations regarding exposure to stable cobalt and its compounds are summarized in Table 8-1. The regulations regarding radioactive cobalt are summarized in Table 8-2.

Stable Cobalt. An MRL of $1\text{x}10^{-4}$ mg cobalt/m^3 has been derived for chronic-duration inhalation exposure. The MRL is based on a NOAEL of 0.0053 mg cobalt/m^3 for decreased respiratory function in exposed workers (Nemery et al. 1992). An MRL of $1\text{x}10^{-2}$ mg/kg-day has been derived for intermediate-duration oral exposure, based on a LOAEL of 1 mg/kg-day for polycythemia in human volunteers (Davis and Fields 1958). No other inhalation or oral MRLs were derived.

The EPA has not derived an RfC or RfD for cobalt and compounds. Similarly, no cancer classification has been performed by the EPA (IRIS 2000). The American Conference of Governmental Industrial Hygienists (ACGIH) has given cobalt a classification of A3, *confirmed animal carcinogen with unknown relevance to humans*, and established an 8-hour time-weighted average (TWA) of 0.02 mg/m^3 for occupational exposure (ACGIH 2000). The Occupational Safety and Health Administration (OSHA) has promulgated an 8-hour permissible exposure limit (PEL) of 0.1 mg/m^3 (OSHA 2001e), and the National Institute for Occupational Safety and Health (NIOSH) recommends an 8-hour TWA of 0.05 mg/m^3 (NIOSH 2001). IARC (2001b) reports that cobalt and cobalt compounds are *possibly carcinogenic to humans* (Group 2B), based on sufficient evidence for cobalt metal and cobalt oxides and limited evidence for cobalt chloride and cobalt sulfate.

Cobalt and its compounds are regulated by the Clean Water Effluent Guidelines for the following industrial point sources: nonferrous metal manufacturing, asbestos, timber products processing, paving and roofing, paint formulating, ink formulating, gum and wood, carbon black, and battery manufacturing (EPA 1988).

Radioactive Cobalt. No MRLs were derived for inhalation or oral exposure to radioactive cobalt. MRLs for acute and chronic exposure to ionizing radiation exist (Agency for Toxic Substances and Disease Registry 1999) and are applicable to cobalt. The EPA has not derived an RfC or RfD for radioactive

Table 8-1. Regulations and Guidelines Applicable to Stable Cobalt

Agency	Description	Information	Reference
INTERNATIONAL Guidelines:			
IARC	Carcinogenicity classification		IARC 2001b
	Cobalt and cobalt compounds[a]	Group 2B[b]	
NATIONAL Regulations and Guidelines:			
a. Air			
ACGIH	TLV-TWA		ACGIH 2000
	Cobalt, elemental, and inorganic compounds (as Co)	0.02 mg/m^3	
NIOSH	REL (TWA)		NIOSH 2001
	Cobalt metal, dust, and fumes (as Co)	0.05 mg/m^3	
	IDLH		
	Cobalt metal, dust, and fumes (as Co)	20 mg/m^3	
OSHA	PEL (8-hour TWA) for general industry		OSHA 2001e
	Cobalt metal, dust, and fumes (as Co)	0.1 mg/m^3	29CFR1910.1000 Table Z
	PEL (8-hour TWA) for construction industry		OSHA 2001d 29CFR1926.55
	Cobalt metal, dust, and fumes (as Co)	0.1 mg/m^3	
	PEL (8-hour TWA) for shipyard industry		OSHA 2001c 29CFR1915.1000
	Cobalt metal, dust, and fumes (as Co)	0.1 mg/m^3	
USC	HAP (cobalt compounds)		USC 2001a 42USC7412
b. Water			
EPA	NPDES permit application testing requirements; conventional and nonconventional pollutants required to be tested by existing dischargers if expected to be present		EPA 2001g 40CFR122 Appendix D Table IV
	BPT effluent limitations		EPA 2001b
	Maximum for 1 day	$3x10^{-4}$ kg/kkg	40CFR415.652
	Average of daily values for 30 consecutive days	$1.2x10^{-4}$ kg/kkg	
	Groundwater monitoring		EPA 2001d
	Suggested method	PQL	40CFR264
	6010	70 µg/L	Appendix IX
	7200	500 µg/L	
	7201	10 µg/L	

Table 8-1. Regulations and Guidelines Applicable to Stable Cobalt

Agency	Description	Information	Reference
NATIONAL (cont.)			
c. Food			
FDA	Drug products withdrawn or removed from the market for reasons of safety or effectiveness	All drug products containing cobalt salts (except radioactive forms of cobalt and its salts and cobalamin and its derivatives)	FDA 2000a 21CFR216.24
	New drug status accorded through rulemaking procedures	Cobalt preparations intended for use by man	FDA 2000b 21CFR310.502 (a)(7)
	Over-the-counter drugs; recommended warning and caution statement for cobalt as a cobalt salt	Required on articles containing ≥0.5 μg per dose and ≥2 μg per 24-hour period	FDA 2000e 21CFR369.20
	Substances generally recognized as safe; trace minerals added to animal feeds	Cobalt acetate Cobalt carbonate Cobalt chloride Cobalt oxide Cobalt sulfate	FDA 2000f 21CFR582.20
	Substances prohibited from use in human food	Cobaltous salts and its derivatives	FDA 2000g 21CFR189.120
d. Other			
ACGIH	Carcinogenicity classification		ACGIH 2000
	Cobalt, elemental, and inorganic compounds (as Co)	A3[c]	
	BEI		
	Cobalt in urine—end of shift at end of workweek	15 μg/L	
	Cobalt in blood—end of shift at end of workweek	1 μg/L	
EPA	Carcinogenicity classification RfC RfD	No data	IRIS 2000
	Toxic chemical release reporting; Community Right-to-Know; effective date	01/01/87	EPA 2001c 40CFR372.65(a)
	Hazardous waste; identification and listing	Contain ≤1 ppmv in synthesis gas fuel generated from hazardous waste	EPA 2001e 40CFR261.38 (b)(5)
	TSCA; health and safety data reporting		EPA 2001j 40CFR716.120
EPA	Municipal solid waste landfills; hazardous constituent for detection monitoring		EPA 2001f 40CFR258 Appendix I and II
	Suggested method	PQL	
	6010	70 μg/L	
	7200	500 μg/L	
	7201	10 μg/L	
	Reportable quantity (cobalt compounds)	1 pound	EPA 2001h 40CFR302.4

Table 8-1. Regulations and Guidelines Applicable to Stable Cobalt

Agency	Description	Information	Reference
NATIONAL (cont.)			
USC	Superfund imposition of tax on cobalt	\$4.45 per ton	USC 2001c 26USC4661
	Exemption of tax imposed on recycled cobalt		USC 2001b 26USC4662
STATE Regulations and Guidelines			
a. Air			
Alabama	HAP (cobalt compounds)		BNA 2001
Alaska	Air contaminant standard (TWA)		BNA 2001
	Cobalt metal, dust, and fumes	0.05 mg/m^3	
California	Airborne contaminant (cobalt metal, dust, and fumes)		BNA 2001
	HAP (cobalt compounds)		BNA 2001
	Toxic air contaminant (cobalt compounds)		CA Air Resources Board 2000
Colorado	HAP (cobalt metal, dust, and fumes)		BNA 2001
	“High-concern” pollutant (cobalt and compounds)		BNA 2001
	Reportable pollutants (cobalt metal, dust, and fumes)		CO Dept. of Public Health and Environment 2000
Connecticut	HAP—hazard limiting value (cobalt metal, dust, and fumes)		BNA 2001
	8 hours	2 μg/m^3	
	30 minutes	10 μg/m^3	
Delaware	Reportable quantities		DE Air Quality Management 2000
	Cobalt carbonyl	1 pound	
	Cobaltous sulfamate	1,000 pounds	
	Cobalt, ((2,2'-(ethane-diylbis(nitrilomethylidyne)	1 pound	
Hawaii	Air contaminant limit (PEL-TWA)		BNA 2001
	Cobalt metal, dust, and fumes	0.05 mg/m^3	
	HAP (cobalt compounds)		BNA 2001
Idaho	TAP non-carcinogenic increments		ID Dept. of Environmental Quality 2000
	Cobalt carbonyl and cobalt hydrocarbonyl (as Co)		
	OEL	1×10^{-1} mg/m^3	
	EL	7×10^{-3} pounds/hour	
	AAC (24-hour average)	5×10^{-3} mg/m^3	
	Cobalt metal, dust, and fumes		
	OEL	5×10^{-2} mg/m^3	
	EL	3.3×10^{-3} pounds/hour	
	AAC (24-hour average)	2.5×10^{-3} mg/m^3	
Illinois	Toxic air contaminant (cobalt)		IL EPA 2000a
Kansas	HAP (cobalt compounds)		KS Dept. of Health and Environment 2000

Table 8-1. Regulations and Guidelines Applicable to Stable Cobalt

Agency	Description	Information	Reference
STATE (cont.)			
Kentucky	HAP (cobalt compounds)		BNA 2001
Louisiana	Toxic air pollutant (cobalt compounds		BNA 2001
Maine	Emissions standards	2,000 pounds	BNA 2001
Maryland	Toxic air pollutant (cobalt compounds)		BNA 2001
Michigan	High concern toxic air pollutants (cobalt compounds)		BNA 2001
Minnesota	HAP threshold (cobalt metal and cobalt carbonyl)	0.1 tons/year	BNA 2001
Missouri	HAP (cobalt compounds)		BNA 2001
Montana	Occupational air contaminant (cobalt metal, dust, and fumes)	0.1 mg/m^3	BNA 2001
Nebraska	HAP (cobalt compounds and cobalt)		BNA 2001
New Mexico	Toxic air pollutant (cobalt metal, dust, and fumes [as Co])		BNA 2001
	OEL	1x10^{-1} mg/m^3	
	Emissions	6.67x10^{-3} pounds/hour	
New York	Annual guideline concentrations	5x10^{-3} μg/m^3	NYS Dept. of Environmental Conservation 2000
	Dangerous air contaminants (TLV) for cobalt metal, dust, and fumes	0.1 mg/m^3	BNA 2001
	HAP (cobalt compounds)		BNA 2001
	Transition limits (PEL)		BNA 2001
	Cobalt metal, dust, and fumes	0.1 mg/m^3	
	Final rule limits (TWA)		
	Cobalt metal, dust, and fumes	0.05 mg/m^3	
North Carolina	PEL-TWA (cobalt metal, dust, and fumes)	0.05 mg/m^3	BNA 2001
Ohio	TRI		Ohio EPA 2000
Oregon	Air contaminant (cobalt metal, dust, and fumes)	0.1 mg/m^3	BNA 2001
Rhode Island	HAP (cobalt compounds)		BNA 2001
South Carolina	Toxic air emissions (MAC) for cobalt compounds	0.25 μg/m^3	BNA 2001
Texas	HAP (cobalt metal, dust, and fumes)	0.1 mg/m^3	BNA 2001
Vermont	HAP (cobalt compounds)		BNA 2001
	Hazardous ambient air standards		BNA 2001
	Cobalt compounds		
	Annual average	0.12 μg/m^3	
	Averaging time	24 hours	
	Action level	6.2x10^{-3} pounds/8 hours	
Washington	Class B TAP and ASIL (24-hour average)		WA Dept. of Ecology 2000
	Cobalt metal, dust and fumes	0.17 μg/m^3	
	Cobalt carbonyl and cobalt hydrocarbonyl	0.33 μg/m^3	

Table 8-1. Regulations and Guidelines Applicable to Stable Cobalt

Agency	Description	Information	Reference
STATE (cont.)			
	Thresholds for HAPs		BNA 2001
	Cobalt carbonyl	0.1 tons/year	
	Cobalt metal, dust, and fumes	0.1 tons/year	
Wisconsin	HAP—existing sources		WI Dept. of Natural Resources 1999
	AAC <25 feet	4.08x10^{-3} pounds/hour	
	AAC ≥25 feet	1.704x10^{-2} pounds/hour	
b. Water			
Alabama	Groundwater monitoring (cobalt)		BNA 2001
	Suggested methods	PQL	
	6010	70 µg/L	
	7200	500 µg/L	
	7201	10 µg/L	
Arizona	Drinking water guideline	0.70 µg/L	FSTRAC 1999
Arkansas	Groundwater monitoring (cobalt)		BNA 2001
	Suggested methods	PQL	
	6010	70 µg/L	
	7200	500 µg/L	
	7201	10 µg/L	
California	Chemicals known to cause cancer or reproductive toxicity; date of initial appearance on the list		Cal/EPA 2000
	Cobalt metal powder		
	Cobalt[II] oxide	07/01/92	
	Cobalt sulfate heptahydrate	07/01/92	
		06/02/00	
Colorado	Groundwater standard (cobalt)	0.05 mg/L	BNA 2001
Delaware	Groundwater monitoring (cobalt)		BNA 2001
	Suggested methods	PQL	
	6010	70 µg/L	
	7200	500 µg/L	
	7201	10 µg/L	
Illinois	Groundwater quality standards for Class II	1 mg/L	IL EPA 2000b
Kentucky	Hazardous waste constituent for groundwater monitoring (cobalt)		BNA 2001
Louisiana	Groundwater monitoring (cobalt)		BNA 2001
	Suggested methods	PQL	
	6010	70 µg/L	
	7200	500 µg/L	
	7201	10 µg/L	
Massachusetts	Groundwater monitoring (cobalt)		BNA 2001
	Suggested methods	PQL	
	6010	70 µg/L	
	7200	500 µg/L	
	7201	10 µg/L	
Minnesota	Drinking water guideline	2 µg/L	FSTRAC 1995
	Groundwater protection hazardous constituent for cobalt (total)		BNA 2001

Table 8-1. Regulations and Guidelines Applicable to Stable Cobalt

Agency	Description	Information	Reference
STATE (cont.)			
Missouri	Water quality standards		BNA 2001
	Livestock, wildlife watering	$1x10^3$ µg/L	
	Groundwater	$1x10^3$ µg/L	
New Mexico	Standards for groundwater of 10,000 mg/L TDS concentration or less (cobalt)	0.05 mg/L	BNA 2001
New York	Groundwater monitoring (cobalt)		BNA 2001
	Suggested methods	PQL	
	6010	70 µg/L	
	7200	500 µg/L	
	7201	10 µg/L	
Tennessee	Effluent limitations—daily maximum concentration (cobalt)	10 mg/L	BNA 2001
Wisconsin	Drinking water guideline	40 µg/L	FSTRAC 1999
	Groundwater standards (cobalt)		BNA 2001
	Enforcement standard	40 µg/L	
	Preventive action limit	8 µg/L	
c. Food		No data	
d. Other			
Alabama	Detection limit values for comparable fuel specification for cobalt; concentration limit	4.6 mg/kg at 10,000 BTU/pound	BNA 2001
Arizona	Soil remediation levels (cobalt)		BNA 2001
	Residential	$4.6x10^3$ mg/kg	
	Non-residential	$9.7x10^4$ mg/kg	
Arkansas	Detection limit values for comparable fuel specification for cobalt; concentration limit	4.6 mg/kg at 10,000 BTU/pound	BNA 2001
	Solid waste management (cobalt)		BNA 2001
	Suggested methods	PQL	
	6010	70 µg/L	
	7200	500 µg/L	
	7201	10 µg/L	
California	Characteristics of toxicity for cobalt and cobalt compounds		BNA 2001
	STLC	80 mg/L	
	TTLC	8,000 mg/kg (wet-weight)	
	Chemicals known to cause cancer or reproductive toxicity (cobalt metal powder); initial appearance on the list	07/01/92	BNA 2001
	Hazardous substance (cobalt, cobalt carbonyl, and cobalt hydrocarbonyl)		BNA 2001
Delaware	Detection limit values for comparable fuel specification for cobalt; concentration limit	4.6 mg/kg at 10,000 BTU/pound	BNA 2001
Florida	Toxic substance in the workplace (cobalt metal, dust, and fumes)		BNA 2001
Georgia	Soil concentration (cobalt)	20 mg/kg	BNA 2001

Table 8-1. Regulations and Guidelines Applicable to Stable Cobalt

Agency	Description	Information	Reference
STATE (cont.)			
Illinois	Analytical parameters and required quantitation limits for cobalt		BNA 2001
	Water		
	Soil	50 µg/L	
	Method	10 mg/kg	
		6010A	
Indiana	Constituent subject to assessment monitoring (cobalt [total and dissolved])		BNA 2001
Maine	Screening standards for beneficial use (cobalt)	5,875 mg/kg (dry weight)	BNA 2001
Michigan	Identification and listing of hazardous waste (cobalt)	When in the form of 100 microns or less	BNA 2001
Minnesota	Hazardous substance		BNA 2001
	Cobalt metal, dust, and fumes (as Co)		
	Cobalt carbonyl (as Co)		
	Cobalt, elemental and inorganic compounds (as Co)		
	Cobalt hydrocarbonyl (as Co)		
Missouri	Hazardous constituent (cobalt [total])		BNA 2001
New Jersey	Hazardous substance		BNA 2001
	Cobalt		
	Cobalt carbonyl		
	Cobalt compounds		
New York	Occupational lung disease; hard metal disease	Cobalt	BNA 2001
Ohio	Toxic release inventory		BNA 2001
Oklahoma	Fertilizer labels and labeling; minimum percentage accepted for registration (cobalt)	5×10^{-4} percent	BNA 2001

Table 8-1. Regulations and Guidelines Applicable to Stable Cobalt

Agency	Description	Information	Reference
Oregon	Toxic substance (cobalt)		BNA 2001
Pennsylvania	Hazardous substance (cobalt and cobalt fumes)		BNA 2001

[a] Cobalt compounds: includes cobalt(II) carbonate, cobalt(II) chloride, cobalt(II) nitrate, cobalt(II) oxide, cobalt(II,III) oxide, cobalt(III) oxide, and cobalt(II) sulfate
[b] Group 2B: possibly carcinogenic to humans
[c] A3: confirmed animal carcinogen with unknown relevance to humans

AAC = acceptable ambient concentrations; ACGIH = American Conference of Governmental Industrial Hygienists; ASIL = acceptable source impact level; BEI = biological exposure indices; BNA = Bureau of National Affairs; BPT = best practicable control technology; BTU = British thermal unit; CFR = Code of Federal Regulations; EL = emissions levels; EPA = Environmental Protection Agency; FDA = Food and Drug Administration; FSTRAC = Federal-State Toxicology and Risk Analysis Committee; HAP = hazardous air pollutant; IARC = International Agency for Research on Cancer; IDLH = immediately dangerous to life and health; IRIS = Integrated Risk Information System; MAC = maximum allowable concentration; NIOSH = National Institute for Occupational Safety and Health; NPDES = National Pollutant Discharge Elimination System; OEL = occupational exposure limit; OSHA = Occupational Safety and Health Administration; PEL = permissible exposure limit; PQL = practical quantitation limit; REL = recommended exposure limit; RfC = reference concentration; RfD = reference dose; STLC = soluble threshold limit concentrations; TAP = toxic air pollutant; TDS = total dissolved solids; TLV = threshold limit value; TRI = Toxic Release Inventory; TSCA = Toxic Substances Control Act; TTLC = total threshold limit concentrations; TWA = time-weighted averages; USC = United States Code

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information	Reference
INTERNATIONAL Guidelines:			
IARC	Carcinogenicity classification	Group 1 (carcinogenic to humans)	IARC 2001b
ICRP	Occupational dose limits; effective dose	20 mSv per year, averaged over defined periods of 5 years	ICRP 1991
	Annual equivalent dose		
	Lens of the eye	150 mSv	
	Skin	500 mSv	
	Hands and feet	500 mSv	
	General population dose limits; effective dose	1 mSv in a year	ICRP 1991
	Annual equivalent dose		
	Lens of eye	15 mSv	
	Skin	50 mSv	
WHO	Drinking water quality	No data	
NATIONAL Regulations and Guidelines:			
a. Air			
ACGIH	All radiation exposures must be kept as low as reasonably achievable		ACGIH 2000
	Effective dose		ACGIH 2000
	Any single year	50 mSv	
	Averaged over 5 years	20 mSv per year	
	Annual equivalent dose		
	Lens of the eye	150 mSv	
	Skin	500 mSv	
	Hands and feet	500 mSv	
	Embryo-fetus exposures once the pregnancy is known		
	Monthly equivalent dose	0.5 mSv	
	Dose to the surface of women's abdomen (lower trunk)	2 mSv for the remainder of the pregnancy	
	Intake of radionuclide	1/20 of the ALI	

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information		Reference
NATIONAL *(cont.)*				
DOE	Radiation standards			DOE 2000
	Inhalation DAC (µCi/mL)	Class Wa	Class Yb	10CFR835
	^{55}Co	1x10^{-6}	1x10^{-6}	Appendix A
	^{56}Co	1x10^{-7}	8x10^{-8}	
	^{57}Co	1x10^{-6}	3x10^{-7}	
	^{58}mCo	4x10^{-5}	3x10^{-5}	
	^{58}Co	5x10^{-7}	3x10^{-7}	
	^{60}mCo	2x10^{-3}	1x10^{-3}	
	^{60}Co	7x10^{-8}	1x10^{-8}	
	^{61}Co	3x10^{-5}	2x10^{-5}	
	^{62}mCo	7x10^{-5}	7x10^{-5}	
	Radiation standards for air immersion DACc (µCi/mL) for ^{60}mCo	1x10-3		DOE 2000 10CFR835 Appendix C
NIOSH	REL	No data		
USNRC	Effluent concentrations—air			USNRC 2001k
	^{55}Co	ALI (µCi/mL)		10CFR20
	Class Wd	4x10^{-9}		Appendix B
	Class Ye	4x10^{-9}		Table 2
	^{56}Co			
	Class Wd	4x10^{-10}		
	Class Ye	3x10^{-10}		
	^{57}Co			
	Class Wd	4x10^{-9}		
	Class Ye	9x10^{-10}		
	^{58}Co			
	Class Wd	2x10^{-9}		
	Class Ye	1x10^{-9}		
	^{58}mCo			
	Class Wd	1x10^{-7}		
	Class Ye	9x10^{-8}		
	^{60}Co			
	Class Wd	2x10^{-10}		
	Class Ye	5x10^{-11}		
	^{60}mCo			
	Class Wd	6x10^{-6}		
	Class Ye	4x10^{-6}		
	^{61}Co			
	Class Wd	9x10^{-8}		
	Class Ye	8x10^{-8}		
	^{62}mCo			
	Class Wd	2x10^{-7}		
	Class Ye	2x10^{-7}		

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information		Reference
NATIONAL *(cont.)*				
USNRC	Occupational values			USNRC 2001k
	Inhalation			10CFR20
	^{55}Co	ALI (µCi)	DAC (µCi/mL)	Appendix B
	Class Wd	3x10^{3}	1x10^{-6}	Table 1
	Class Ye	3x10^{3}	1x10^{-6}	
	^{56}Co			
	Class Wd	3x10^{2}	1x10^{-7}	
	Class Ye	2x10^{2}	8x10^{-8}	
	^{57}Co			
	Class Wd	3x10^{3}	1x10^{-6}	
	Class Ye	7x10^{2}	3x10^{-7}	
	^{58}Co			
	Class Wd	1x10^{3}	5x10^{-7}	
	Class Ye	7x10^{2}	3x10^{-7}	
	^{58}mCo			
	Class Wd	9x10^{4}	4x10^{-5}	
	Class Ye	6x10^{4}	3x10^{-5}	
	^{60}Co			
	Class Wd	2x10^{2}	7x10^{-8}	
	Class Ye	3x10^{1}	1x10^{-8}	
	^{60}mCo			
	Class Wd	4x10^{6}	2x10^{-3}	
	Class Ye	3x10^{6}	1x10^{-3}	
	^{6}1Co			
	Class Wd	6x10^{4}	3x10^{-5}	
	Class Ye	6x10^{4}	2x10^{-5}	
	^{62}mCo			
	Class Wd	2x10^{5}	7x10^{-5}	
	Class Ye	2x10^{5}	6x10^{-5}	
OSHA	Safety and health regulations for construction—ionizing radiation			OSHA 2001e 29CFR1926.53
	Toxic and hazardous substances—ionizing radiation			OSHA 2001d 29CFR1910.1096
b. Water				
EPA	Drinking water standards			EPA 2000
	Beta particle and photon activity (formerly man-made radionuclides)			
	MCL	4 mrem		
	Caner risk at 10^{-4}	4 mrem/year		
	Gross alpha particle activity			
	MCL	15 pCi/L		
	Caner risk at 10^{-4}	15 pCi/L		
	Carcinogenic classification	Group A (human carcinogen)		

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information	Reference
NATIONAL *(cont.)*			
USNRC	Effluent concentrations		USNRC 2001k
	Water		10CFR20
	^{55}Co	ALI (µCi/mL)	Appendix B
	Class Wd	2x10^{-5}	Table 2
	^{56}Co		
	Class Wd	6x10^{-6}	
	^{57}Co		
	Class Wd	6x10^{-5}	
	^{58}Co		
	Class Wd	2x10^{-5}	
	^{58}mCo		
	Class Wd	8x10^{-4}	
	^{60}Co		
	Class Wd	3x10^{-6}	
	^{60}mCo		
	Class Wd	2x10^{-2}	
	^{61}Co		
	Class Wd	3x10^{-4}	
	^{62}mCo		
	Class Wd	7x10^{-4}	
	Releases to sewers—monthly average concentration		USNRC 2001k 10CFR20
	^{55}Co	ALI (µCi/mL)	Appendix B
	Class Wd	2x10^{-4}	Table 3
	^{56}Co		
	Class Wd	6x10^{-5}	
	^{57}Co		
	Class Wd	6x10^{-4}	
	^{58}Co		
	Class Wd	2x10^{-4}	
	^{58}mCo		
	Class Wd	8x10^{-3}	
	^{60}Co		
	Class Wd	3x10^{-5}	
	^{60}mCo		
	Class Wd	2x10^{-1}	
	^{61}Co		
	Class Wd	3x10^{-3}	
	^{62}mCo		
	Class Wd	7x10^{-3}	
c. Food and Drug			
FDA	Ionizing radiation for the treatment of poultry feed and poultry feed ingredients (energy sources)	Ioninzing radiation is limited to gamma rays from sealed units of ^{60}CO	FDA 1999 21CFR579.40
	Requirements regarding certain radioactive drugs for ^{58}Co or ^{60}Co	Labeled cyanocobalamin for use in intestinal absorption studies	FDA 2000d 21CFR310.503(c)

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information			Reference
NATIONAL *(cont.)*					
FDA	Sources of radiation used for inspection of food, packaged food, and controlling food processing				FDA 2000c 21CFR179.21 (a)(2)
d. Other					
DOE	Values for establishing sealed radioactive source accountability and radioactive material posting and labeling requirements	Activity (µCi)			DOE 2000 10CFR835 Appendix E
	^{56}Co	4.0x10^{1}			
	^{57}Co	2.3x10^{2}			
	^{58}Co	1.4x10^{2}			
	^{60}Co	1.8x10^{1}			
DOT	Activity values (Ci)	A1	A2		DOT 2001a 49CFR173.435 Table
	^{55}Co	13.5	13.5		
	^{56}Co	8.11	8.11		
	^{57}Co	216	216		
	^{58}mCo	1080	1080		
	^{58}Co	27.0	27.0		
	^{60}Co	10.8	10.8		
	Superfund, reportable quantity (Ci) (pounds)				DOT 2001b 49CFR172.101 Appendix A Table 2
	^{55}Co	10			
	^{56}Co	10			
	^{57}Co	100			
	^{58}Co	10			
	^{58}mCo	1,000			
	^{60}Co	10			
	^{60}mCo	1,000			
	^{61}Co	1,000			
	^{62}mCo	1,000			
EPA	Carcinogenicity classification RfC RfD	No data			IRIS 2000
	Annual possession quantities for environmental compliance (Ci/year)	Gas	Liquid/ Powder	Solid	EPA 2001a 40CFR61 Appendix E Table 1
	^{56}Co	2.3x10^{-6}	2.3x10^{-3}	2.3	
	^{57}Co	1.8x10^{-2}	1.8x10^{1}	1.8x10^{4}	
	^{58}Co	2.5x10^{-6}	2.5x10^{-3}	2.5	
	^{58m}Co	2.3x10^{-6}	2.3x10^{-3}	2.3	
	^{60}Co	4.6x10^{-2}	4.6x10^{1}	4.6x10^{4}	
	^{60m}Co	7.0	7.0x10^{3}	7.0x10^{6}	
	^{61}Co	9.8x10^{-1}	9.8x10^{2}	9.8x10^{5}	

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information	Reference
NATIONAL *(cont.)*			
EPA	Concentration levels for environmental compliance (Ci/m^3)		EPA 2001a 40CFR61 Appendix E Table 2
	^{56}Co	1.8×10^{-13}	
	^{57}Co	1.3×10^{-12}	
	^{58}Co	6.7×10^{-13}	
	^{58m}Co	1.2×10^{-10}	
	^{60}Co	1.7×10^{-14}	
	^{60m}Co	4.3×10^{-9}	
	^{61}Co	4.5×10^{-9}	
	Carcinogenicity—slope factors		EPA 2002
	Lifetime risk per pCi—ingestion		EPA 2002
	Water		
	^{57}Co	1.04×10^{-12}	
	^{58m}Co	2.95×10^{-12}	
	^{58}Co	1.26×10^{-13}	
	^{60}Co	1.57×10^{-11}	
	Lifetime risk per pCi—ingestion		EPA 2002
	Food		
	^{57}Co	1.49×10^{-12}	
	^{58m}Co	4.18×10^{-12}	
	^{58}Co	1.83×10^{-13}	
	60Co	2.23×10^{-11}	
	Lifetime risk per pCi—ingestion		EPA 2002
	Soil		
	^{57}Co	2.78×10^{-12}	
	^{58}mCo	7.44×10^{-12}	
	^{58}Co	3.47×10^{-13}	
	^{60}Co	4.03×10^{-11}	
	Lifetime risk per pCi—inhalation		EPA 2002
	^{57}Co	2.09×10^{-12}	
	^{58}mCo	5.99×10^{-12}	
	^{58}Co	6.88×10^{-14}	
	^{60}Co	3.58×10^{-11}	
	External exposure—risk/year per pCi/g soil		EPA 2002
	^{57}Co	3.55×10^{-7}	
	^{58}mCo	4.48×10^{-6}	
	^{58}Co	1.00×10^{-12}	
	^{60}Co	1.24×10^{-5}	

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information		Reference
NATIONAL *(cont.)*				
EPA	Superfund, reportable quantities (Ci) (pounds)			EPA 2001i 40CFR302.4 Appendix B
	^{55}Co	10		
	^{56}Co	10		
	^{57}Co	100		
	^{58}mCo	1,000		
	^{58}Co	10		
	^{60}mCo	1,000		
	^{60}Co	10		
	^{61}Co	1,000		
	^{62}mCo	1,000		
NCRP	Occupational exposures			NCRP1993
	Effective dose limits			
	Annual	50 mSv		
	Cummulative	10 mSv x age		
	Equivalent dose annual limits	150 mSv		
	Lens of eye	500 mSv		
	Skin, hands, and feet			
	Public exposures (annual)			
	Effective dose limits, continuous or frequent exposure	1 mSv		
	Effective dose limits, infrequent exposures	5 mSv		
	Equivalent dose limits			
	Lens of eye	15 mSv		
	Skin, hands, and feet	50 mSv		
	Embryo and fetus exposures (monthly)			
	Effective dose limit	0.5 mSv		
USNRC	Activity values for radionuclides (Ci)	A1	A2	USNRC 2001a 10CFR71
	^{55}Co	13.5	13.5	
	^{56}Co	8.11	8.11	
	^{57}Co	216	216	
	^{58}mCo	1080	1080	
	^{58}Co	27.0	27.0	
	^{60}Co	10.8	10.8	
	Byproduct material listing; exempt concentrations			USNRC 2001e 10CFR30.70 Schedule A
	Liquid and solid concentration (µCi/mL^2)			
	^{57}C	5x10^{-3}		
	^{58}C	1x10^{-3}		
	^{60}C	5x10^{-4}		

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information		Reference
<u>NATIONAL</u> *(cont.)*				
USNRC	Byproduct material listing (µCi)			USNRC 2001b
	^{58}mCo			10CFR30.71
	^{58}Co	10		Schedule B
	^{60}Co	10		
		1		
	Byproduct material listing (Ci)	Column If	Column IIg	USNRC 2001c
	^{58}mCo	100	1.0	10CFR33.100
	^{58}Co	1.0	0.01	Schedule A
	^{60}Co	0.1	$1\text{x}10^{-4}$	
	Items containing byproduct material listing—^{60}Co (µCi)			USNRC 2001d 10CFR30.15(a)(8)
	Electron tubes	1.0		
	Spark gap irradiators	1.0		
	Medical use—^{60}Co as a source for brachytherapy	As a sealed source in needles and applicator cells for topical, interstitial, and intracavitary treatment of cancer		USNRC 2001h 10CFR35.400
	Occupational values—oral ingestion			USNRC 2001k 10CFR20
	^{55}Co	ALI (µCi)		Appendix B
	Class Wd	$1\text{x}10^{3}$		Table 1
	^{56}Co			
	Class Wd	$5\text{x}10^{2}$		
	Class Ye	$4\text{x}10^{2}$		
	^{57}Co			
	Class Wd	$8\text{x}10^{3}$		
	Class Ye	$4\text{x}10^{3}$		
	^{58}Co			
	Class Wd	$2\text{x}10^{3}$		
	Class Ye	$1\text{x}10^{3}$		
	^{58}mCo			
	Class Wd	$6\text{x}10^{4}$		
	^{60}Co			
	Class Wd	$5\text{x}10^{2}$		
	Class Ye	$2\text{x}10^{2}$		
	^{60}mCo			
	Class Wd	$1\text{x}10^{6}$		
	St. wall	$1\text{x}10^{6}$		
	^{61}Co			
	Class Wd	$2\text{x}10^{4}$		
	Class Ye	$2\text{x}10^{4}$		
	^{62}mCo			
	Class Wd	$5\text{x}10^{4}$		
	St. wall	$4\text{x}10^{4}$		

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information	Reference
NATIONAL *(cont.)*			
USNRC	Quantities of radioactive material requiring labeling (µCi)		USNRC 2001g 10CFR30 Appendix B
	^{58}mCo	10	
	^{58}Co	10	
	^{60}Co	1	
	Quantities of licensed material requiring labeling (µCi)		USNRC 2001i 10CFR20 Appendix C
	^{55}Co		
	^{56}Co	100	
	^{57}Co	10	
	^{58}mCo	100	
	^{58}Co	1,000	
	^{60}mCo	100	
	^{60}Co	1,000	
	^{61}Co	1	
	^{62}mCo	1,000	
		1,000	
	Quantities of radioactive materials requiring need for an emergency plan		USNRC 2001j 10CFR30.72 Schedule C
	Release fraction	0.001%	
	Quantity (Ci)	5,000	
	Radioactive waste classification		USNRC 2001l 10CFR61.55
	Class A (Ci/m3)		
	60Co	≤700	
	Reports of individual monitoring—processing or manufacturing for distribution, byproduct material in quantities exceeding		USNRC 2001f 10CFR20.2206 (a)(7)
	^{60}Co (Ci)	1.0	
STATE Regulations and Guidelines:			
a. Air			
Alabama	HAP—radionuclides		BNA 2001
California	HAP—radionuclides		BNA 2001
Hawaii	HAP—radionuclides		BNA 2001
Illinois	Toxic air contaminant—radionuclides		BNA 2001
Kansas	HAP—radionuclides		BNA 2001
Kentucky	HAP—radionuclides		BNA 2001
Minnesota	HAP—radionuclides		BNA 2001
Missouri	HAP—radionuclides		BNA 2001

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information	Reference
STATE *(cont.)*			
Nebraska	HAP—radionuclides		BNA 2001
New York	HAP—radionuclides		BNA 2001
Rhode Island	HAP—radionuclides		BNA 2001
Wyoming	HAP—radionuclides		BNA 2001

[a] Class W: refers to the approximate length of retention in the pulmonary region which is 10–100 days for this class
[b] Class Y: refers to the approximate length of retention in the pulmonary region which is greater than 100 days for this class
[c] Air immersion DAC values: based on a stochastic dose limit of 5 rems (0.05 Sv) per year or a nonstochastic (organ) dose limit of 50 rems (0.5 Sv) per year
[d] Class W: all compounds except those given for Y
[e] Class Y: oxides, hydroxides, halides, and nitrates
[f] Column I: gas concentration
[g] Column II: liquid and solid concentration

ACGIH = American Conference of Governmental Industrial Hygienists; ALI = annual limits on intake; BNA = Bureau of National Affairs; CFR = Code of Federal Regulations; DAC = derived air concentrations; DOE = Department of Energy; DOT = Department of Transportation; EPA = Environmental Protection Agency; FDA = Food and Drug Administration; IARC = International Agency for Research on Cancer; ICRP = International Commission on Radiological Protection; IRIS = Integrated Risk Information System; mSv = millisievert; NCRP = National Council on Radiation Protection; NIOSH = National Institute for Occupational Safety and Health; OSHA = Occupational Safety and Health Administration; PEL = permissible exposure limit; REL = recommended exposure limit; RfC = reference concentration; RfD = reference dose; TLV = threshold limit value; TWA = time-weighted averages; USNRC = U.S. Nuclear Regulatory Commission; WHO = World Health Organization cobalt (IRIS 2000). Slope factors have been derived for exposure to cobalt radioisotopes (EPA 2002). The slope factors for ^{60}Co are 1.57x10^{-11}, 2.23x10^{-11}, and 4.03x10^{-11}/pCi for ingestion of water, food, and soil, respectively. The slope factor for inhalation exposure is 3.58x10^{-11}/pCi, and 1.24x10^{-5}/year/pCi/g soil for external exposure. The slope factors for ^{58}Co are 1.26x10$^{-13,}$, 1.83x10$^{-13,}$, and 3.47x10^{-13}/pCi for ingestion of water, food, and soil, respectively. The slope factor for inhalation exposure is 6.88x10^{-14}/pCi for inhalation exposure, and 1.00x10^{-12}/year/pCi/g soil for external exposure. The slope factors for ^{58m}Co are 2.95x10$^{-12,}$, 4.18x10$^{-12,}$, and 7.44x10^{-12}/pCi for ingestion of water, food, and soil, respectively. The slope factor for inhalation exposure is 5.99x10^{-14}/pCi for inhalation exposure, and 4.48x10^{-6}/year/pCi/g soil for external exposure. The slope factors for ^{57}Co are 1.04x10^{-12}, 1.49x10^{-12}, and 2.78x10^{-12}/pCi for ingestion of water, food, and soil, respectively. The slope factor for inhalation exposure is 2.09x10^{-12}/pCi for ingestion, and 3.55x10^{-7}/year/pCi/g soil for external exposure.

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10. GLOSSARY

Some terms in this glossary are generic and may not be used in this profile.

Absorbed Dose, Chemical—The amount of a substance that is either absorbed into the body or placed in contact with the skin. For oral or inhalation routes, this is normally the product of the intake quantity and the uptake fraction divided by the body weight and, if appropriate, the time, expressed as mg/kg for a single intake or mg/kg/day for multiple intakes. For dermal exposure, this is the amount of material applied to the skin, and is normally divided by the body mass and expressed as mg/kg.

Absorbed Dose, Radiation—The mean energy imparted to the irradiated medium, per unit mass, by ionizing radiation. Units: rad (rad), gray (Gy).

Absorbed Fraction—A term used in internal dosimetry. It is that fraction of the photon energy (emitted within a specified volume of material) which is absorbed by the volume. The absorbed fraction depends on the source distribution, the photon energy, and the size, shape and composition of the volume.

Absorption—The process by which a chemical penetrates the exchange boundaries of an organism after contact, or the process by which radiation imparts some or all of its energy to any material through which it passes.

Absorption Coefficient—Fractional absorption of the energy of an unscattered beam of x- or gamma-radiation per unit thickness (linear absorption coefficient), per unit mass (mass absorption coefficient), or per atom (atomic absorption coefficient) of absorber, due to transfer of energy to the absorber. The total absorption coefficient is the sum of individual energy absorption processes (see Compton Effect, Photoelectric Effect, and Pair Production).

Absorption Coefficient, Linear—A factor expressing the fraction of a beam of x- or gamma radiation absorbed in a unit thickness of material. In the expression $I=I_oe^{-\mu x}$, I_o is the initial intensity, I the intensity of the beam after passage through a thickness of the material x, and μ is the linear absorption coefficient.

Absorption Coefficient, Mass—The linear absorption coefficient per cm divided by the density of the absorber in grams per cubic centimeter. It is frequently expressed as μ/ρ, where μ is the linear absorption coefficient and ρ the absorber density.

Absorption Ratio, Differential—Ratio of concentration of a nuclide in a given organ or tissue to the concentration that would be obtained if the same administered quantity of this nuclide were uniformly distributed throughout the body.

Activation—The process of making a material radioactive by bombardment with neutrons or protons.

Activity—The number of radioactive nuclear transformations occurring in a material per unit time (see Curie, Becquerel). The term for activity per unit mass is specific activity.

Activity Median Aerodynamic Diameter (AMAD)—The diameter of a unit-density sphere with the same terminal settling velocity in air as that of the aerosol particle whose activity is the median for the entire size distribution of the aerosol.

Acute Exposure, Chemical—Exposure to a chemical for a duration of 14 days or less, as specified in the Toxicological Profiles.

Acute Exposure, Radiation—The absorption of a relatively large amount of radiation (or intake of a radioactive material) over a short period of time.

Acute Radiation Syndrome—The symptoms which taken together characterize a person suffering from the effects of intense radiation. The effects occur within hours or days.

Ad libitum—Available in excess and freely accessible.

Adsorption Coefficient (K_{oc})—The ratio of the amount of a chemical adsorbed per unit surface area or per unit weight of organic carbon of a specific particle size in the soil or sediment to the concentration of the chemical in solution at equilibrium.

Adsorption Ratio (K_d)—See Distribution Coefficient.

Alpha Particle—A positively charged particle ejected spontaneously from the nuclei of some radioactive elements. It is identical to a helium nucleus, i.e., 2 neutrons and two protons, with a mass number of 4 and an electrostatic charge of +2.

Alpha Track—The track of ionized atoms (pattern of ionization) left in a medium by an alpha particle that has traveled through the medium.

Annihilation (Positron-Electron)—An interaction between a positive and a negative electron in which they both disappear; their rest mass, being converted into electromagnetic radiation (called annihilation radiation) with two 0.51 MeV gamma photons emitted at an angle of 180° to each other.

Annual Limit on Intake (ALI)—The derived limit for the amount of radioactive material taken into the body of an adult worker by inhalation or ingestion in a year. It is the smaller value of intake of a given radionuclide in a year by the reference man that would result in a committed effective dose equivalent of 5 rem or a committed dose equivalent of 50 rem to any organ or tissue.

Atom—The smallest particle of an element that cannot be divided or broken up by chemical means. It consists of a central core called the *nucleus*, which contains *protons* and *neutrons* and an outer shell of *electrons*.

Atomic Mass (u)—The mass of a neutral atom of a nuclide, usually expressed in terms of "atomic mass units." The "atomic mass unit" is one-twelfth the mass of one neutral atom of carbon-12; equivalent to 1.6604×10^{-24} g.

Atomic Mass Number—See Mass Number.

Atomic Number—The number of protons in the nucleus of an atom. The "effective atomic number" is calculated from the composition and atomic numbers of a compound or mixture. An element of this atomic number would interact with photons in the same way as the compound or mixture. (Symbol: Z).

Atomic Weight—The weighted mean of the masses of the neutral isotopes of an element expressed in atomic mass units.

Attenuation—A process by which a beam from a source of radiation is reduced in intensity by absorption and scattering when passing through some material.

Attenuation Coefficient—The fractional reduction in the intensity of a beam of radiation as it passes through an absorbing medium. It may be expressed as reduction per unit distance, per unit mass thickness, or per atom, and is called the linear, mass, or atomic attenuation coefficient, respectively.

Auger Effect—The emission of an electron from the extranuclear portion of an excited atom when the atom undergoes a transition to a less excited state.

Background Radiation—The amount of radiation to which a member of the general population is exposed from natural sources, such as terrestrial radiation from naturally occurring radionuclides in the soil, cosmic radiation originating from outer space, and naturally occurring radionuclides deposited in the human body.

Becquerel (Bq)—International System of Units unit of activity and equals that quantity of radioactive material in which one transformation (disintegration) occurs per second (see Units).

Terabecquerel (TBq)—One trillion becquerel.
Gigabecquerel (GBq)—One billion becquerel.
Megabecquerel (MBq)—One million becquerel.
Kilobecquerel (kBq))—One thousand becquerel.
Millibecquerel (mBq)—One-thousandth of a becquerel.
Microbecquerel (µBq)—One-millionth of a becquerel.

Beta Particle—An electron that is emitted from the nucleus of an atom during one type of radioactive transformation. A beta particle has a mass and charge equal in magnitude to that of the electron. The charge may be either +1 or -1. Beta particles with +1 charges are called positrons (symbolized β^+), and beta particles with -1 charges are called negatrons (symbolized β^-).

Bioconcentration Factor (BCF)—The quotient of the concentration of a chemical in aquatic organisms at a specific time or during a discrete time period of exposure divided by the concentration in the surrounding water at the same time or during the same period.

Biologic Effectiveness of Radiation—See Relative Biological Effectiveness.

Biological Half-time—The time required for a biological system, such as that of a human, to eliminate by natural process half of the amount of a substance (such as a chemical substance, either stable or radioactive) that has entered it.

Biomagnification—The progressive increase in the concentration of a bioaccumulated chemical in organisms as that chemical is passed from the bottom to the top of the food web.

Biomarkers—Broadly defined as indicators signaling events in biologic systems or samples. They have been classified as markers of exposure, markers of effect, and markers of susceptibility.

Body Burden, Chemical—The total amount of a chemical found in an animal or human body.

Body Burden, Radioactivity—The amount of radioactive material found in an animal or human body.

Bone Seeker—Any compound or ion which migrates in the body and preferentially deposits into bone.

Branching—The occurrence of two or more modes by which a radionuclide can undergo radioactive decay. For example, ^{214}Bi can undergo alpha or beta minus decay, ^{64}Cu can undergo beta minus, beta plus, or electron capture decay. An individual atom of a nuclide exhibiting branching disintegrates by one mode only. The fraction disintegrating by a particular mode is the "branching fraction" for that mode. The "branching ratio" is the ratio of two specified branching fractions (also called multiple disintegration).

Bremsstrahlung—X rays that are produced when a charged particle accelerates (speeds up, slows down, or changes direction) in the strong field of a nucleus.

Buildup Factor—The ratio of the radiation intensity, including both primary and scattered radiation, to the intensity of the primary (unscattered) radiation.

Cancer Effect Level (CEL)—The lowest dose of chemical or radiation in a study, or group of studies, that produces significant increases in the incidence of cancer (or tumors) between the exposed population and its appropriate control.

Capture, Electron—A mode of radioactive decay involving the capture of an orbital electron by its nucleus. Capture from a particular electron shell, e.g., K or L shells, is designated as "K-electron capture" or "L-electron capture."

Capture, K-Electron—Electron capture from the K shell by the nucleus of the atom. Also loosely used to designate any orbital electron capture process.

Carcinogen—A chemical or radiation that is capable of inducing cancer.

Carcinoma—Malignant neoplasm composed of epithelial cells, regardless of their derivation.

Case-Control Study—A type of epidemiological study which examines the relationship between a particular outcome (disease or condition) and a variety of potential causative agents (such as toxic chemicals). In a case-controlled study, a group of people with a specified and well-defined outcome is identified and compared to a similar group of people without outcome.

Case Report—Describes a single individual with a particular disease or exposure. These may suggest some potential topics for scientific research but are not actual research studies.

Cataract—A clouding of the crystalline lens of the eye which obstructs the passage of light.

Ceiling Value—A concentration of a substance that should not be exceeded, even temporarily.

Charged Particle—A nuclear particle, atom, or molecule carrying a positive or negative charge.

Chronic Exposure—A long-term, continuous exposure to a chemical or radioactive material. For example, exposure to a chemical for 365 days or more, as specified in the Toxicological Profiles.

Cohort Study—A type of epidemiological study of a specific group or groups of people who have had a common insult (e.g., exposure to an agent suspected of causing disease or a common disease) and are followed forward from exposure to outcome. At least one exposed group is compared to one unexposed group.

Collective Dose—The sum of the individual doses received in a given period of time by a specified population from exposure to a specified source of radiation. Collective dose is expressed in units such as man-rem and person-sievert.

Compton Effect—An attenuation process observed for x- or gamma radiation in which an incident photon interacts with an orbital electron of an atom to produce a recoil electron and a scattered photon whose energy is less than the incident photon.

Containment—The confinement of a chemical or radioactive substance in such a way that it is prevented from being dispersed from its container or into the environment, or is released only at a specified rate.

Contamination—Deposition of a stable or radioactive substance in any place where it is not desired.

Cosmic Rays—High-energy particulate and electromagnetic radiations that originate outside the earth's atmosphere and interact with the atmosphere to produce a shower of secondary cosmic rays.

Count (Radiation Measurements)—The external indication of a radiation-measuring device designed to enumerate ionizing events. It refers to a single detected event. The term “count rate” refers to the total number registered in a given period of time. The term is sometimes erroneously used to designate a disintegration, ionizing event, or voltage pulse.

Counter, Gas-flow Proportional (GPC)—An instrument for detecting beta particle radiation. Beta particles are detected by ionization of the counter gas which results in an electrical impulse at an anode wire.

Counter, Geiger-Mueller (GM counter)—Highly sensitive, gas-filled radiation-measuring device that detects (counts) individual photons or particulate radiation.

Counter, Scintillation—The combination of a crystal or phosphor, photomultiplier tube, and associated circuits for counting light emissions produced in the phosphors by ionizing radiation. Scintillation counters generally are more sensitive than GM counters for gamma radiation.

Counting, Cerenkov—Relatively energetic β-particles pass through a transparent medium of high refractive index and a highly-directional, bluish-white light ("Cerenkov" light) is emitted. This light is detected using liquid scintillation counting equipment.

Cross-sectional Study—A type of epidemiological study of a group or groups which examines the relationship between exposure and outcome to a chemical or to chemicals at one point in time.

Curie (Ci)—A unit of radioactivity. One curie equals that quantity of radioactive material in which there are 3.7×10^{10} nuclear transformations per second. The activity of 1 gram of radium is approximately 1 Ci.

Attocurie (aCi)—One-thousandth of a femtocurie (3.7×10^{-8} disintegrations per second).
Femtocurie (fCi)—One-billionth of a microcurie (3.7×10^{-5} disintegrations per second).

Megacurie (MCi)—One million curies ($3.7x10^{16}$ disintegrations per second).
Microcurie (µCi)—One-millionth of a curie ($3.7x10^{4}$ disintegrations per second).
Millicurie (mCi)—One-thousandth of a curie ($3.7x10^{7}$ disintegrations per second).
Nanocurie (nCi)—One-billionth of a curie ($3.7x10^{1}$ disintegrations per second).
Picocurie (pCi)—One-millionth of a microcurie ($3.7x10^{-2}$ disintegrations per second).

Daughter Products—See Progeny and Decay Product

Decay Chain or Decay Series—A sequence of radioactive decays (transformations) beginning with one nucleus. The initial nucleus, the parent, decays into a daughter or progeny nucleus that differs from the first by whatever particles were emitted during the decay. If further decays take place, the subsequent nuclei are also usually called daughters or progeny. Sometimes, to distinguish the sequence, the daughter of the first daughter is called the granddaughter, etc.

Decay Constant (λ)—The fraction of the number of atoms of a radioactive nuclide which decay in unit time (see Disintegration Constant).

Decay Product, Daughter Product, Progeny—A new nuclide formed as a result of radioactive decay. A nuclide resulting from the radioactive transformation of a radionuclide, formed either directly or as the result of successive transformations in a radioactive series. A decay product (daughter product or progeny) may be either radioactive or stable.

Decay, Radioactive—Transformation of the nucleus of an unstable nuclide by spontaneous emission of radiation, such as charged particles and/or photons (see Disintegration).

Delta Ray—An electron removed from an atom of a medium that is irradiated, or through which radiation passes, during the process of ionization (also called secondary electron). Delta rays cause a track of ionizations along their path.

Derived Air Concentration (DAC)—The concentration of radioactive material in air that, if breathed by the reference man for a working year of 2000 hours under conditions of light work (at a rate of 1.2 liters of air per hour), would result in an intake of one ALI (see Annual Limit on Intake).
Deterministic Effect—A health effect, the severity of which varies with the dose and for which a threshold is believed to exist (also called a non-stochastic effect).

Developmental Toxicity—The occurrence of adverse effects on the developing organism that may result from exposure to a chemical or radiation prior to conception (either parent), during prenatal development, or postnatally to the time of sexual maturation. Adverse developmental effects may be detected at any point in the life span of the organism.

Disintegration Constant—Synonymous with decay constant. The fraction of the number of atoms of a radioactive material that decays per unit time (see Decay Constant.)

Disintegration, Nuclear—A spontaneous nuclear transformation (radioactivity) characterized by the emission of energy and mass from the nucleus. When large numbers of nuclei are involved, the process is characterized by a definite half-life (see Transformation, Nuclear).

Distribution Coefficient (K_d)—Describes the distribution of a chemical between the solid and aqueous phase at thermodynamic equilibrium, is given as follows:

$$K_d = \frac{[C]_s}{[C]_w}$$, Units = (L solution)/(kg solid),

where $[C]_s$ is the concentration of the chemical associated with the solid phase in units of (mg)/(kg solid), and $[C]_w$ is the concentration of the chemical in the aqueous phase in units of (mg)/(L solution). As the magnitude of K_d decreases, the potential mobility of the chemical to groundwater systems increases and vice versa.

Dose—A general term denoting the quantity of a substance, radiation, or energy absorbed. For special purposes it must be appropriately qualified. If unqualified, it refers to radiation absorbed dose.

> **Absorbed Dose**—The energy imparted to matter by ionizing radiation per unit mass of irradiated material at the place of interest. The unit of absorbed dose is the rad. One rad equals 100 ergs per gram. In SI units, the absorbed dose is the gray which is 1 J/kg (see Rad).
>
> **Cumulative Dose (Radiation)**—The total dose resulting from repeated or continuous exposures to radiation.

Dose Assessment—An estimate of the radiation dose to an individual or a population group usually by means of predictive modeling techniques, sometimes supplemented by the results of measurement.

Dose Equivalent (DE)—A quantity used in radiation safety practice to account for the relative biological effectiveness of the several types of radiation. It expresses all radiations on a common scale for calculating the effective absorbed dose. The NRC defines it as the product of the absorbed dose, the quality factor, and all other modifying factors at the location of interest. ICRP has changed its definition to be the product of the absorbed dose and the radiation weighting factor. (The unit of dose equivalent is the rem. In SI units, the dose equivalent is the sievert, which equals 100 rem.)

Dose, Fractionation—A method of administering therapeutic radiation in which relatively small doses are given daily or at longer intervals.

Dose, Protraction—A method of administering therapeutic radiation by delivering it continuously over a relatively long period at a low dose rate.

Dose, Radiation—The amount of energy imparted to matter by ionizing radiation per unit mass of the matter, usually expressed as the unit rad, or in SI units, the gray. 100 rad=1 gray (Gy) (see Absorbed Dose).

> **Committed Dose Equivalent ($H_{T,50}$)**—The dose equivalent to organs or tissues of reference (T) that will be received from an intake of radioactive material by an individual during the 50 years following the intake.
>
> **Committed Effective Dose Equivalent ($H_{E,50}$)**—The sum of the products of the weighting factors applicable to each of the body organs or tissues that are irradiated and the committed dose equivalent to those organs or tissues.
>
> **Effective Dose** —A dose value that attempts to normalize the detriment to the body (for cancer mortality and morbidity, hereditary effects, and years of life lost) from a non-uniform exposure to

that of a uniform whole body exposure. Effective dose is calculated as the sum of products of the equivalent dose and the tissue weighting factor (w_T) for each tissue exposed. ($E = \sum D_{T,R}\ w_R\ w_{T)}$).

Effective Dose Equivalent (H_E)—This dose type is limited to internal exposures and is the sum of the products of the dose equivalent to the organ or tissue (H_T) and the weighting factors (w_T) applicable to each of the body organs or tissues that are irradiated. ($H_E = \sum w_T\ H_T$).

Equivalent Dose—A dose quantity that places the biological effect of all radiation types on a common scale for calculating tissue damage. Alpha particles, for example, are considered to cause 20 times more damage than gamma rays. Equivalent dose is calculated as the sum of products of the average absorbed dose (in gray) in an organ or tissue ($_{DT,R}$) from each type of radiation and the radiation weighting factor (w_R) for that radiation ($\sum D_{T,R}\ w_R$).

External Dose—That portion of the dose equivalent received from radiation sources outside the body.

Internal Dose—That portion of the dose equivalent received from radioactive material taken into the body.

Limit—A permissible upper bound on the radiation dose.

Maximum Permissible Dose (MPD)—The greatest dose equivalent that a person or specified part thereof shall be allowed to receive in a given period of time.

Median Lethal Dose (MLD)—Dose of radiation required to kill, within a specified period (usually 30 days), 50% of the individuals in a large group of animals or organisms. Also called the LD_{50}, or $LD_{50/30}$ if for 30 days.

Threshold Dose—The minimum absorbed dose that will produce a detectable degree of any given effect.

Tissue Dose—Absorbed dose received by tissue in the region of interest, expressed in rad (see Dose, Gray, and Rad).

Dose Rate—The amount of radiation dose delivered per unit time. Generically, the rate at which radiation dose is delivered to any material or tissue.

Dose-Response Relationship—The quantitative relationship between the amount of exposure to a toxicant and the incidence of the adverse effects.

Dosimetry—Quantification of radiation doses to cells, tissues, organs, individuals or populations resulting from radiation exposures.

Early Effects (of radiation exposure)—Effects that appear within 60 days of an acute exposure.

Electron—A stable elementary particle having an electric charge equal to $\pm 1.60210\text{x}10^{-19}$ C (Coulombs) and a rest mass equal to $9.1091\text{x}10^{-31}$ kg. A positron is a positively charged "electron" (see Positron).

Electron Volt—A unit of energy equivalent to the energy gained by an electron in passing through a potential difference of one volt. Larger multiple units of the electron volt are frequently used: keV for thousand or kilo electron volts; MeV for million or mega electron volts (eV). 1 eV=1.6×10^{-12} erg.

Embryotoxicity and Fetotoxicity—Any toxic effect on the conceptus as a result of prenatal exposure to a chemical; the distinguishing feature between the two terms is the stage of development during which the insult occurred. The terms, as used here, include malformations and variations, altered growth, and *in utero* death.

Energy—Capacity for doing work. Gravitationally, "potential energy" is the energy inherent in a mass because of its spatial relation to other masses. Chemically or radiologically, “potential energy” is the energy released when a chemical reaction or radiological transformation goes to completion. "Kinetic energy" is the energy possessed by a mass because of its motion (SI unit: joules):

> **Binding Energy (Electron)**—The amount of energy that must be expended to remove an electron from an atom.
>
> **Binding Energy (Nuclear)**—The energy represented by the difference in mass between the sum of the component parts and the actual mass of the nucleus. It represents the amount of energy that must be expended to break a nucleus into its component neutrons and protons.
>
> **Excitation Energy**—The energy required to change a system from its ground state to an excited state. Each different excited state has a different excitation energy.
>
> **Ionizing Energy**—The energy required to knock an electron out of an atom. The average energy lost by electrons or beta particles in producing an ion pair in air or in soft tissue is about 34 eV.
>
> **Radiant Energy**—The energy of electromagnetic radiation, such as radio waves, visible light, x and gamma rays.

Enrichment, Isotopic—An isotopic separation process by which the relative abundances of the isotopes of a given element are altered, thus producing a form of the element that has been enriched in one or more isotopes and depleted in others. In uranium enrichment, the percentage of uranium-235 in natural uranium can be increased from 0.7% to >90% in a gaseous diffusion process based on the different thermal velocities of the constituents of natural uranium (^{234}U, ^{235}U, ^{238}U) in the molecular form UF_6.

EPA Health Advisory—An estimate of acceptable drinking water levels for a chemical substance based on health effects information. A health advisory is not a legally enforceable federal standard, but serves as technical guidance to assist federal, state, and local officials.

Epidemiology—Refers to the investigation of factors that determine the frequency and distribution of disease or other health-related conditions within a defined human population during a specified period.

Equilibrium, Radioactive—In a radioactive series, the state which prevails when the ratios between the activities of two or more successive members of the series remains constant.

> **Secular Equilibrium**—If a parent element has a very much longer half-life than the daughters (so there is not appreciable change in its amount in the time interval required for later products to attain equilibrium) then, after equilibrium is reached, equal numbers of atoms of all members of

the series disintegrate in unit time. This condition is never exactly attained, but is essentially established in such a case as ^{226}Ra and its transformation series to stable ^{206}Pb. The half-life of ^{226}Ra is about 1,600 years; of ^{222}Rn, approximately 3.82 days, and of each of the subsequent members, a few minutes. After about a month, essentially the equilibrium amount of radon is present; then (and for a long time) all members of the series disintegrate the same number of atoms per unit time. At this time, the activity of the daughter is equal to the activity of the parent.

Transient Equilibrium—If the half-life of the parent is short enough so the quantity present decreases appreciably during the period under consideration, but is still longer than that of successive members of the series, a stage of equilibrium will be reached after which all members of the series decrease in activity exponentially with the period of the parent. At this time, the ratio of the parent activity to the daughter activity is constant.

Equilibrium, Electron—The condition in a radiation field where the energy of the electrons entering a volume equals the energy of the electrons leaving that volume.

Excitation—The addition of energy to a system, thereby transferring it from its ground state to an excited state. Excitation of a nucleus, an atom, or a molecule can result from absorption of photons or from inelastic collisions with other particles. The excited state of an atom is an unstable or metastable state and will return to ground state by radiation of the excess energy.

Exposure (Chemical)—Contact of an organism with a chemical or physical agent. Exposure is quantified as the amount of the agent available at the exchange boundaries of the organism (e.g., skin, lungs, gut) and available for absorption.

Exposure (Radiation)—Subjection to ionizing radiation or to a radioactive material. For example, exposure in air is a measure of the ionization produced in air by x or gamma radiation; the sum of the electric charges on all ions of one sign produced in air when all electrons liberated by photons in a volume of air are completely stopped in air (dQ), divided by the mass of the air in the volume (dm). The unit of exposure in air is the roentgen, or coulomb per kilogram (SI units). One roentgen is equal to 2.58×10^{-4} coulomb per kilogram (C/kg).

Fission, Nuclear—A nuclear transformation characterized by the splitting of a nucleus into at least two other nuclei with emission of several neutrons, accompanied by the release of a relatively large amount of energy.

Gamma Ray, Penetrating—Short wavelength electromagnetic radiation of nuclear origin.

Genetic Effect of Radiation—Inheritable change, chiefly mutations, produced by the absorption of ionizing radiation by germ cells. Genetic effects have not been observed in any human population exposed at any dose level.

Genotoxicity—A specific adverse effect on the genome of living cells that, upon the duplication of affected cells, can be expressed as a mutagenic, clastogenic or carcinogenic event because of specific alteration of the molecular structure of the genome.

Gray (Gy)—SI unit of absorbed dose, 1 J/kg. One gray equals 100 rad (see Units).

Half-life, Effective—See Half-Time, Effective.

Half-life, Radioactive—Time required for a radioactive substance to lose 50% of its activity by decay. Each radio-nuclide has a unique physical half-life. Known also as physical half-time and symbolized as T_r or T_{rad}.

Half-time, Biological—Time required for an organ, tissue, or the whole body to eliminate one-half of any absorbed substance by regular processes of elimination. This is the same for both stable and radioactive isotopes of a particular element, and is sometimes referred to as half-time, symbolized as t_{biol} or T_b.

Half-time, Effective—Time required for a radioactive element in an organ, tissue, or the whole body to be diminished 50% as a result of the combined action of radioactive decay and biological elimination, symbolized as T_e or T_{eff}.

$$\text{Effective half-time} = \frac{\text{Biological half-time} \times \text{Radioactive half-life}}{\text{Biological half-time} + \text{Radioactive half-life}}$$

Immediately Dangerous to Life or Health (IDLH)—The maximum environmental concentration of a contaminant from which one could escape within 30 minutes without any escape-impairing symptoms or irreversible health effects.

Immunologic Toxicity—The occurrence of adverse effects on the immune system that may result from exposure to environmental agents such as chemicals.

Immunological Effects—Functional changes in the immune response.

In Vitro—Isolated from the living organism and artificially maintained, as in a test tube. Literally, “in glass.”

In Vivo—Occurring within the living organism. Literally, “in life.”

Intensity—Amount of energy per unit time passing through a unit area perpendicular to the line of propagation at the point in question.

Intermediate Exposure—Exposure to a chemical for a duration of 15–364 days, as specified in the Toxicological Profiles.

Internal Conversion—Process in which a gamma ray knocks an electron out of the same atom from which the gamma ray was emitted. The ratio of the number of internal conversion electrons to the number of gamma quanta emitted in the de-excitation of the nucleus is called the "conversion ratio."

Ion—Atomic particle, atom or chemical radical bearing a net electrical charge, either negative or positive.

Ion Pair—Two particles of opposite charge, usually referring to the electron and positive atomic or molecular residue resulting after the interaction of ionizing radiation with the orbital electrons of atoms.

Ionization—The process by which a neutral atom or molecule acquires a positive or negative charge.

> **Primary Ionization**—(1) In collision theory: the ionization produced by the primary particles as contrasted to the "total ionization" which includes the "secondary ionization" produced by delta

rays. (2) In counter tubes: the total ionization produced by incident radiation without gas amplification.

Specific Ionization—Number of ion pairs per unit length of path of ionizing radiation in a medium; e.g., per centimeter of air or per micrometer of tissue.

Total Ionization—The total electric charge of one sign on the ions produced by radiation in the process of losing its kinetic energy. For a given gas, the total ionization is closely proportional to the initial ionization and is nearly independent of the nature of the ionizing radiation. It is frequently used as a measure of absorption of radiation energy.

Ionization Density—Number of ion pairs per unit volume.

Ionization Path (Track)—The trail of ion pairs produced by an ionizing particle in its passage through matter.

Ionizing Radiation—Any radiation capable of knocking electrons out of atoms and producing ions. Examples: alpha, beta, gamma and x rays, and neutrons.

Isobars—Nuclides having the same mass number but different atomic numbers.

Isomers—Nuclides having the same number of neutrons and protons but capable of existing, for a measurable time, in different quantum states with different energies and radioactive properties. Commonly the isomer of higher energy decays to one with lower energy by the process of isomeric transition.

Isotopes—Nuclides having the same number of protons in their nuclei, and hence the same atomic number, but differing in the number of neutrons, and therefore in the mass number. Identical chemical properties exist in isotopes of a particular element. The term should not be used as a synonym for nuclide because isotopes refer specifically to different nuclei of the same element.

Stable Isotope—A nonradioactive isotope of an element.

Joule—The S.I. unit for work and energy. It is equal to the work done by raising a mass of one newton through a distance of one meter (J = Nm), which corresponds to about 0.7 ft-pound.

Kerma (k)—A measure of the kinetic energy transferred from gamma rays or neutrons to a unit mass of absorbing medium in the initial collision between the radiation and the absorber atoms. The SI unit is J/kg. The special name of this unit is the rad (traditional system of units) or Gray (SI).

Labeled Compound—A compound containing one or more radioactive atoms intentionally added to its structure. By observations of radioactivity or isotopic composition, this compound or its fragments may be followed through physical, chemical, or biological processes.

Late Effects (of radiation exposure)—Effects which appear 60 days or more following an acute exposure.

$LD_{50/30}$—The dose of a chemical or radiation expected to cause 50% mortality in those exposed within 30 days. For radiation, this is about 350 rad (3.5 gray) received by humans over a short period of time.

Lethal Concentration$_{(Lo)}$ (LC$_{Lo}$)—The lowest concentration of a chemical in air that has been reported to have caused death in humans or animals.

Lethal Concentration$_{(50)}$ (LC$_{50}$)—A calculated concentration of a chemical in air to which exposure for a specific length of time is expected to cause death in 50% of a defined experimental animal population within a specified time, usually 30 days.

Lethal Dose$_{(Lo)}$ (LD$_{Lo}$)—The lowest dose of a chemical introduced by a route other than inhalation that is expected to have caused death in humans or animals within a specified time, usually 30 days.

Lethal Dose$_{(50)}$ (LD$_{50}$)—The dose of a chemical which has been calculated to cause death in 50% of a defined experimental animal population.

Lethal Time$_{(50)}$ (LT$_{50}$)—A calculated period of time within which a specific concentration of a chemical is expected to cause death in 50% of a defined experimental animal population.

Linear Energy Transfer (LET)—A measure of the energy that a charged particle transfers to a material per unit path length.

> **Average LET**—The energy of a charged particle divided by the length of the path over which it deposits all its energy in a material. This is averaged over a number of particles.
>
> **High-LET**—Energy transfer characteristic of heavy charged particles such as protons and alpha particles where the distance between ionizing events is small on the scale of a cellular nucleus.
>
> **Low-LET**—Energy transfer characteristic of light charged particles such as electrons produced by x and gamma rays where the distance between ionizing events is large on the scale of a cellular nucleus.

Lowest-Observed-Adverse-Effect Level (LOAEL)—The lowest dose of chemical in a study, or group of studies, that produces statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control.

Lung Clearance Class (fast, F; medium, M; slow, S)—A classification scheme for inhaled material according to its rate of clearance from the pulmonary region of the lungs to the blood and the gastrointestinal tract.

Lymphoreticular Effects—Represent morphological effects involving lymphatic tissues such as the lymph nodes, spleen, and thymus.

Malformations—Permanent structural changes that may adversely affect survival, development, or function.

Mass Numbers (A)—The number of nucleons (protons and neutrons) in the nucleus of an atom.

Minimal Risk Level—An estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse noncancerous effects over a specified duration of exposure.

Morbidity—State of being diseased; morbidity rate is the incidence or prevalence of disease in a specific population.

Mutagen—A substance that causes changes (mutations) in the genetic material in a cell. Mutations can lead to birth defects, miscarriages, or cancer.

Necropsy—The gross examination of the organs and tissues of a dead body to determine the cause of death or pathological conditions.

Neurotoxicity—The occurrence of adverse effects on the nervous system following exposure to a substance.

Neutrino (ν)—A neutral particle of infinitesimally small rest mass emitted during beta plus or beta minus decay. This particle accounts for conservation of energy in beta plus and beta minus decays. It plays no role in damage from radiation.

No-Observed-Adverse-Effect Level (NOAEL)—The dose of a substance at which there were no statistically or biologically significant increases in frequency or severity of adverse effects seen between the exposed population and its appropriate control. Effects may be produced at this dose, but they are not considered to be adverse.

Nuclear Reactor—A power plant that heats the medium (typically water) by using the energy released from the nuclear fission of uranium or plutonium isotopes instead of burning coal, oil, or natural gas. All of these sources of energy simply heat water and use the steam which is produced to turn turbines that make electricity or propel a ship.

Nucleon—Common name for a constituent particle of the nucleus. Applied to a proton or neutron.

Nuclide—A species of atom characterized by the constitution of its nucleus. The nuclear constitution is specified by the number of protons (Z), number of neutrons (N), and energy content; or, alternatively, by the atomic number (Z), mass number A(N+Z), and atomic mass. To be regarded as a distinct nuclide, the atom must be capable of existing for a measurable time. Thus, nuclear isomers are separate nuclides, whereas promptly decaying excited nuclear states and unstable intermediates in nuclear reactions are not so considered.

Octanol-Water Partition Coefficient (K_{ow})—The equilibrium ratio of the concentrations of a chemical in n-octanol and water, in dilute solution.

Odds Ratio (OR)—A means of measuring the association between an exposure (such as toxic substances and a disease or condition) which represents the best estimate of relative risk (risk as a ratio of the incidence among subjects exposed to a particular risk factor divided by the incidence among subjects who were not exposed to the risk factor). An odds ratio of greater than 1 is considered to indicate greater risk of disease in the exposed group compared to the unexposed.

Pair Production—An absorption process for x- and gamma radiation in which the incident photon is absorbed in the vicinity of the nucleus of the absorbing atom, with subsequent production of an electron and positron pair (see annihilation). This reaction can only occur for incident photon energies exceeding 1.02 MeV.

Parent—Any radionuclide nuclide which, upon disintegration, yields a new nuclide (termed the progeny or daughter), either directly or as a later member of a radioactive series.

Permissible Exposure Limit (PEL)—A maximum allowable atmospheric level of a substance in workplace air averaged over an 8-hour shift.

Pharmacokinetic Model—A set of equations that can be used to describe the time course of a parent chemical or metabolite in an animal system. There are two types of pharmacokinetic models: data-based and physiologically-based. A data-based model divides the animal system into a series of compartments which, in general, do not represent real, identifiable anatomic regions of the body whereas the physiologically-based model compartments represent real anatomic regions of the body.

Pharmacokinetics—The dynamic behavior of a material in the body, used to predict the fate (disposition) of an exogenous substance in an organism. Utilizing computational techniques, it provides the means of studying the absorption, distribution, metabolism and excretion of chemicals by the body.

Physiologically Based Pharmacodynamic (PBPD) Model—A type of physiologically-based dose-response model which quantitatively describes the relationship between target tissue dose and toxic end points. These models advance the importance of physiologically based models in that they clearly describe the biological effect (response) produced by the system following exposure to an exogenous substance.

Physiologically Based Pharmacokinetic (PBPK) Model—A model comprising a series of compartments representing organs or tissue groups with realistic weights and blood flows. These models require a variety of physiological information: tissue volumes, blood flow rates to tissues, cardiac output, alveolar ventilation rates and, possibly membrane permeabilities. The models also utilize biochemical information such as air/blood partition coefficients, and metabolic parameters. PBPK models are also called biologically based tissue dosimetry models.

Photoelectric Effect—An attenuation process observed for x and gamma radiation in which an incident photon interacts with a tightly bound inner orbital electron of an atom delivering all of its energy to knock the electron out of the atom. The incident photon disappears in the process.

Photon—A quantum of electromagnetic energy (E) whose value is the product of its frequency (ν) in hertz and Planck's constant (h). The equation is: $E = h\nu$.

Population dose—See Collective dose.

Positron—A positively charged electron.

Potential, Ionization—The energy expressed as electron volts (eV) necessary to separate one electron from an atom, resulting in the formation of an ion pair.

Power, Stopping—A measure of the ability of a material to absorb energy from an ionizing particle passing through it; the greater the stopping power, the greater the energy absorbing ability (see Linear Energy Transfer).

Progeny—The decay product or daughter products resulting after a radioactive decay or a series of radioactive decays. The progeny can also be radioactive, and the chain continues until a stable nuclide is formed.

Proton—Elementary nuclear particle with a positive electric charge equal numerically to the charge of the electron and a rest mass of 1.007 mass units.

Quality—A term describing the distribution of the energy deposited by a particle along its track; radiations that produce different densities of ionization per unit intensity are said to have different "qualities."

Quality Factor (Q)—The linear-energy-transfer-dependent factor by which absorbed doses are multiplied to obtain (for radiation protection purposes) a quantity that expresses - on a common scale for all ionizing radiation - the approximate biological effectiveness of the absorbed dose.

Type of radiation	Quality Factor
X, gamma, or beta	1
Alpha particles	20
Neutrons of unknown energy	10
High energy protons	10

Rad—The traditional unit of absorbed dose equal to 100 ergs per gram, or 0.01 joule per kilogram (0.01 Gy) in any medium (see Absorbed Dose).

Radiation—The emission and propagation of energy through space or through a material medium in the form of waves (e.g., the emission and propagation of electromagnetic waves, or of sound and elastic waves). The term radiation or radiant energy, when unqualified, usually refers to electromagnetic radiation. Such radiation commonly is classified according to frequency, as microwaves, infrared, visible (light), ultraviolet, and x and gamma rays (see Photon.) and, by extension, corpuscular emission, such as alpha and beta radiation, neutrons, or rays of mixed or unknown type, as cosmic radiation.

Radiation, Annihilation—Photons produced when an electron and a positron unite and cease to exist. The annihilation of a positron-electron pair results in the production of two photons, each of 0.51 MeV energy.

Radiation, Background—See Background Radiation.

Radiation, Characteristic (Discrete)—Radiation originating from an excited atom after removal of an electron from an atom. The wavelength of the emitted radiation is specific, depending only on the element and particular energy levels involved.

Radiation, External—Radiation from a source outside the body.

Radiation, Internal—Radiation from a source within the body (as a result of deposition of radionuclides in body tissues).

Radiation, Ionizing—Any electromagnetic or particulate radiation capable of producing ions, directly or indirectly, in its passage through matter (see Radiation).

Radiation, Monoenergetic—Radiation of a given type in which all particles or photons originate with and have the same energy.

Radiation, Scattered—Radiation which during its passage through a substance, has been deviated in direction. It may also have been modified by a decrease in energy.

Radiation, Secondary—A particle or ray that is produced when the primary radiation interacts with a material, and which has sufficient energy to produce its own ionization, such as bremsstrahlung or electrons knocked from atomic orbitals with enough energy to then produce ionization (see Delta Rays).

Radiation Weighting Factor (also called Quality Factor)—In radiation protection, a factor (1 for x-rays, gamma rays, beta particles; 20 for alpha particles) weighting the absorbed dose of radiation of a specific type and energy for its effect on tissue.

Radioactive Material—Material containing radioactive atoms.

Radioactivity—Spontaneous nuclear transformations that result in the formation of new elements. These transformations are accomplished by emission of alpha or beta particles from the nucleus or by the capture of an orbital electron. Each of these reactions may or may not be accompanied by a gamma photon.

Radioactivity, Artificial—Man-made radioactivity produced by particle bombardment or nuclear fission, as opposed to naturally occurring radioactivity.

Radioactivity, Induced—Radioactivity produced in a substance after bombardment with neutrons or other particles. The resulting activity is "natural radioactivity" if formed by nuclear reactions occurring in nature and "artificial radioactivity" if the reactions are caused by man.

Radioactivity, Natural—The property of radioactivity exhibited by more than 50 naturally occurring radionuclides.

Radioisotope—An unstable or radioactive isotope of an element that decays or disintegrates spontaneously, emitting radiation.

Radionuclide—Any radioactive isotope of any element. Approximately 5,000 natural and artificial radioisotopes have been identified.

Radiosensitivity—Relative susceptibility of cells, tissues, organs, organisms, or any living substance to the injurious action of radiation. Radiosensitivity and its antonym, radioresistance, are used comparatively, rather than absolutely.

Reference Dose (RfD)—An estimate of the daily exposure of the human population to a potential hazard that is likely to be without risk of deleterious effects during a lifetime. The RfD is operationally derived from the NOAEL (from animal and human studies) by a consistent application of uncertainty factors that reflect various types of data used to estimate RfDs and an additional modifying factor, which is based on a professional judgment of the entire database on the chemical. The RfDs are not applicable to non-threshold effects such as cancer.

Relative Biological Effectiveness (RBE)—The RBE is a factor used to compare the biological effectiveness of absorbed radiation doses (i.e., rad) due to different types of ionizing radiation. More specifically, it is the experimentally determined ratio of an absorbed dose of a radiation in question to the absorbed dose of a reference radiation (typically ^{60}Co gamma rays or 200 kVp x rays) required to produce an identical biological effect in a particular experimental organism or tissue (see Quality Factor).

Rem—The traditional unit of dose equivalent that is used in the regulatory, administrative, and engineering design aspects of radiation safety practice. The dose equivalent in rem is numerically equal to the absorbed dose in rad multiplied by the quality factor (1 rem is equal to 0.01 sievert).

Reportable Quantity (RQ)—The quantity of a hazardous substance that is considered reportable under CERCLA. Reportable quantities are (1) 1 pound or greater or (2) for selected substances, an amount established by regulation either under CERCLA or under Sect. 311 of the Clean Water Act. Quantities are measured over a 24-hour period.

Reproductive Toxicity—The occurrence of adverse effects on the reproductive system that may result from exposure to a chemical. The toxicity may be directed to the reproductive organs and/or the related endocrine system. The manifestation of such toxicity may be noted as alterations in sexual behavior, fertility, pregnancy outcomes, or modifications in other functions that are dependent on the integrity of this system.

Roentgen (R)—A unit of exposure (in air) to ionizing radiation. It is the amount of x or gamma rays required to produce ions carrying 1 electrostatic unit of electrical charge in 1 cubic centimeter of dry air under standard conditions. Named after William Roentgen, a German scientist who discovered x rays in 1895.

Retrospective Study—A type of cohort study based on a group of persons known to have been exposed at some time in the past. Data are collected from routinely recorded events, up to the time the study is undertaken. Retrospective studies are limited to causal factors that can be ascertained from existing records and/or examining survivors of the cohort.

Self-Absorption—Absorption of radiation (emitted by radioactive atoms) by the material in which the atoms are located; in particular, the absorption of radiation within a sample being assayed.

Short-Term Exposure Limit (STEL)—The maximum concentration to which workers can be exposed for up to 15 minutes continually. No more than four excursions are allowed per day, and there must be at least 60 minutes between exposure periods. The daily TLV-TWA may not be exceeded.

SI Units—The International System of Units as defined by the General Conference of Weights and Measures in 1960. These units are generally based on the meter/kilogram/second units, with special quantities for radiation including the becquerel, gray, and sievert.

Sickness, Acute Radiation (Syndrome)—The complex symptoms and signs characterizing the condition resulting from excessive exposure of the whole body (or large part) to ionizing radiation. The earliest of these symptoms are nausea, fatigue, vomiting, and diarrhea, and may be followed by loss of hair (epilation), hemorrhage, inflammation of the mouth and throat, and general loss of energy. In severe cases, where the radiation dose is relatively high (over several hundred rad or several gray), death may occur within two to four weeks. Those who survive six weeks after exposure of a single high dose of radiation may generally be expected to recover.

Sievert (Sv)—The SI unit of any of the quantities expressed as dose equivalent. The dose equivalent in sieverts is equal to the absorbed dose, in gray, multiplied by the quality factor (1 sievert equals 100 rem). The sievert is also the SI unit for effective dose equivalent, which is the sum of the products of the dose equivalent to each organ or tissue and its corresponding tissue weighting factor.

Specific-Activity—Radioactivity per unit mass of a radionuclide, expressed, for example, as Ci/gram or Bq/kilogram.

Specific Energy—The actual energy per unit mass deposited per unit volume in a small target, such as the cell or cell nucleus, as the result of one or more energy-depositing events. This is a stochastic quantity as opposed to the average value over a large number of instance (i.e., the absorbed dose).

Standardized Mortality Ratio (SMR)—A ratio of the observed number of deaths and the expected number of deaths in a specific standard population.

Stochastic Effect—A health effect that occurs randomly and for which the probability of the effect occurring, rather than its severity, is assumed to be a linear function of dose without a threshold (also called a nondeterministic effect).

Stopping Power—The average rate of energy loss of a charged particle per unit thickness of a material or per unit mass of material traversed.

Surface-seeking Radionuclide—A bone-seeking internal emitter that deposits and remains on the bone surface for a long period of time, although it may eventually diffuse into the bone mineral. This contrasts with a volume seeker, which deposits more uniformly throughout the bone volume.

Target Organ Toxicity—This term covers a broad range of adverse effects on target organs or physiological systems (e.g., renal, cardiovascular) extending from those arising through a single limited exposure to those assumed over a lifetime of exposure to a chemical.

Target Theory (Hit Theory)—A theory explaining some biological effects of radiation on the basis that ionization, occurring in a discrete volume (the target) within the cell, directly causes a lesion which subsequently results in a physiological response to the damage at that location. One, two, or more "hits" (ionizing events within the target) may be necessary to elicit the response.

Teratogen—A chemical that causes birth defects.

Threshold Limit Value (TLV®)—The maximum concentration of a substance to which most workers can be exposed without adverse effect. TLV is a term used exclusively by the ACGIH. Other terms used to express similar concepts are the MAC (Maximum Allowable Concentration) and PEL (Permissible Exposure Limits).

Time-Weighted Average (TWA)—An allowable exposure concentration averaged over a normal 8-hour workday or 40-hour workweek.

Tissue Weighting Factor (W_t)—Organ- or tissue-specific factor by which the equivalent dose is multiplied to give the portion of the effective dose for that organ or tissue. Recommended values of tissue weighting factors are:

Tissue/Organ	Tissue Weighting Factor
Gonads	0.70
Bone marrow (red)	0.12
Colon	0.12
Lung	0.12
Stomach	0.12
Bladder	0.05
Breast	0.05
Liver	0.05
Esophagus	0.05
Thyroid	0.05
Skin	0.01
Bone surface	0.01
Remainder (adrenals, brain, upper large intestine, small intestine, pancreas, spleen, thymus, and uterus)	0.05

Toxic Dose (TD_{50})—A calculated dose of a chemical, introduced by a route other than inhalation, which is expected to cause a specific toxic effect in 50% of a defined experimental animal population.

Toxicokinetic—The absorption, distribution and elimination of toxic compounds in the living organism.

Toxicosis—A diseased condition resulting from poisoning.

Transformation, Nuclear—The process of radioactive decay by which a nuclide is transformed into a different nuclide by absorbing or emitting particulate or electromagnetic radiation.

Transition, Isomeric—The process by which a nuclide decays to an isomeric nuclide (i.e., one of the same mass number and atomic number) of lower quantum energy. Isomeric transitions (often abbreviated I.T.) proceed by gamma ray and internal conversion electron emission.

Tritium—The hydrogen isotope with one proton and two neutrons in the nucleus (Symbol: ^3H). It is radioactive and has a physical half-life of 12.3 years.

Unattached Fraction—That fraction of the radon daughters, usually ^{218}Po and ^{214}Po, which has not yet attached to a dust particle or to water vapor. As a free atom, it has a high probability of being exhaled and not retained within the lung. It is the attached fraction which is primarily retained.

Uncertainty Factor (UF)—A factor used in operationally deriving the RfD from experimental data. UFs are intended to account for (1) the variation in sensitivity among the members of the human population, (2) the uncertainty in extrapolating animal data to the case of human, (3) the uncertainty in extrapolating from data obtained in a study that is of less than lifetime exposure, and (4) the uncertainty in using LOAEL data rather than NOAEL data. Usually each of these factors is set equal to 10.

Units, Prefixes—Many units of measure are expressed as submultiples or multiples of the primary unit (e.g., 10^{-3} curie is 1 mCi and 10^{3} becquerel is 1 kBq).

Factor	Prefix	Symbol	Factor	Prefix	Symbol
10^{-18}	atto	A	10^{3}	kilo	k
10^{-15}	femto	F	10^{6}	mega	M
10^{-12}	pico	p	10^{9}	giga	G
10^{-9}	nano	N	10^{12}	tera	T
10^{-6}	micro	M	10^{15}	peta	P
10^{-3}	milli	M	10^{18}	exa	E
10^{-2}	centi	C			

Units, Radiological—

Units	Equivalents
Becquerel* (Bq)	1 disintegration per second = 2.7×10^{-11} Ci
Curie (Ci)	3.7×10^{10} disintegrations per second = 3.7×10^{10} Bq
Gray* (Gy)	1 J/kg = 100 rad
Rad (rad)	100 erg/g = 0.01 Gy
Rem (rem)	0.01 sievert
Sievert* (Sv)	100 rem

*International Units, designated (SI)

Working Level (WL)—Any combination of short-lived radon daughters in 1 liter of air that will result in the ultimate emission of 1.3×10^{5} MeV of potential alpha energy.

Working Level Month (WLM)—A unit of exposure to radon daughters corresponding to the product of the radon daughter concentration in Working Level (WL) and the exposure time in nominal months (1 nominal month = 170 hours). Inhalation of air with a concentration of 1 WL of radon daughters for 170 working hours results in an exposure of 1 WLM.

X rays—Penetrating electromagnetic radiations whose wave lengths are very much shorter than those of visible light. They are usually produced by bombarding a metallic target with fast electrons in a high vacuum. X rays (called characteristic x rays) are also produced when an orbital electron falls from a high energy level to a low energy level.

Zero-Threshold Linear Hypothesis (or No-Threshold Linear Hypothesis)—The assumption that a dose-response curve derived from data in the high dose and high dose-rate ranges may be extrapolated through the low dose and low dose range to zero, implying that, theoretically, any amount of radiation will cause some damage.

APPENDIX A. ATSDR MINIMAL RISK LEVELS AND WORKSHEETS

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) [42 U.S.C. 9601 et seq.], as amended by the Superfund Amendments and Reauthorization Act (SARA) [Pub. L. 99–499], requires that the Agency for Toxic Substances and Disease Registry (ATSDR) develop jointly with the U.S. Environmental Protection Agency (EPA), in order of priority, a list of hazardous substances most commonly found at facilities on the CERCLA National Priorities List (NPL); prepare toxicological profiles for each substance included on the priority list of hazardous substances; and assure the initiation of a research program to fill identified data needs associated with the substances.

The toxicological profiles include an examination, summary, and interpretation of available toxicological information and epidemiologic evaluations of a hazardous substance. During the development of toxicological profiles, Minimal Risk Levels (MRLs) are derived when reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration for a given route of exposure. An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure. MRLs are based on noncancer health effects only and are not based on a consideration of cancer effects. These substance-specific estimates, which are intended to serve as screening levels, are used by ATSDR health assessors to identify contaminants and potential health effects that may be of concern at hazardous waste sites. It is important to note that MRLs are not intended to define clean-up or action levels.

MRLs are derived for hazardous substances using the no-observed-adverse-effect level/uncertainty factor approach. They are below levels that might cause adverse health effects in the people most sensitive to such chemical-induced effects. MRLs are derived for acute (1–14 days), intermediate (15–364 days), and chronic (365 days and longer) durations and for the oral and inhalation routes of exposure. Currently, MRLs for the dermal route of exposure are not derived because ATSDR has not yet identified a method suitable for this route of exposure. MRLs are generally based on the most sensitive chemical-induced end point considered to be of relevance to humans. Serious health effects (such as irreparable damage to the liver or kidneys, or birth defects) are not used as a basis for establishing MRLs. Exposure to a level above the MRL does not mean that adverse health effects will occur.

MRLs are intended only to serve as a screening tool to help public health professionals decide where to look more closely. They may also be viewed as a mechanism to identify those hazardous waste sites that are not expected to cause adverse health effects. Most MRLs contain a degree of uncertainty because of the lack of precise toxicological information on the people who might be most sensitive (e.g., infants, elderly, nutritionally or immunologically compromised) to the effects of hazardous substances. ATSDR uses a conservative (i.e., protective) approach to address this uncertainty consistent with the public health principle of prevention. Although human data are preferred, MRLs often must be based on animal studies because relevant human studies are lacking. In the absence of evidence to the contrary, ATSDR assumes that humans are more sensitive to the effects of hazardous substance than animals and that certain persons may be particularly sensitive. Thus, the resulting MRL may be as much as 100-fold below levels that have been shown to be nontoxic in laboratory animals.

Proposed MRLs undergo a rigorous review process: Health Effects/MRL Workgroup reviews within the Division of Toxicology, expert panel peer reviews, and agency-wide MRL Workgroup reviews, with participation from other federal agencies and comments from the public. They are subject to change as new information becomes available concomitant with updating the toxicological profiles. Thus, MRLs in the most recent toxicological profiles supersede previously published levels. For additional information regarding MRLs, please contact the Division of Toxicology, Agency for Toxic Substances and Disease Registry, 1600 Clifton Road NE, Mailstop F-32, Atlanta, Georgia 30333.

MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical Name: Cobalt
CAS Number: 10026-24-1
Date: March 2004
Profile Status: Final
Route: [x] Inhalation [ ] Oral
Duration: [ ] Acute [ ] Intermediate [x] Chronic
Key to figure: 26
Species: human

Minimal Risk Level: 1×10^{-4} [ ] mg/kg/day [ ] ppm [x] mg/m^3

Reference:

Nemery B, Casier P, Roosels D, et al. 1992. Survey of cobalt exposure and respiratory health in diamond polishers. Am Rev Respir Dis 145:610-616.

Experimental design:

Nemery et al. (1992) conducted a cross-sectional study of cobalt exposure and respiratory effects in diamond polishers. The study group was composed of 194 polishers working in 10 different workshops. In two of these workshops (#1, 2), the workers used cast iron polishing disks almost exclusively, and in the others, they used cobalt-containing disks primarily. The number of subjects from each workshop varied from 6 to 28 and the participation rate varied from 56 to 100%. The low participation in some workshops reflects the fact that only workers who used cobalt disks were initially asked to be in the study, rather than a high refusal rate (only eight refusals were documented). More than a year after the polishing workshops were studied, an additional three workshops with workers engaged in sawing diamonds, cleaving diamonds, or drawing jewelry were studied as an unexposed control group (n=59 workers). Subjects were asked to fill out a questionnaire regarding employment history, working conditions, medical history, respiratory symptoms, and smoking habits, to give a urine sample for cobalt determination, and to undergo a clinical examination and lung function tests. Both area air samples and personal air samples were collected (always on a Thursday). Sampling for area air determinations started 2 hours after work began and continued until 1 hour before the end of the work day. Personal air samples were collected from the breathing zone of a few workers per workshop for four successive 1-hour periods. Air samples were analyzed for cobalt and iron. In addition, personal air samplers were used to sample the air 1 cm above the polishing disks. These samples were analyzed for the entire spectrum of mineral and metallic compounds. Air samples were not obtained at one of the polishing workshops (#4), but this workshop was reported to be almost identical to an adjoining workshop (#3) for which samples were obtained. Urinary cobalt levels were similar between workers in these two workshops, so exposure was considered to be similar as well. It is important to note that the study authors suggested that the available methods used for air sampling may have underestimated the exposure levels.

There was a good correlation (R=0.92) between the results of area and personal air sampling, with area air sampling reporting lower concentrations than personal air samples in all workshops except one (#9) (Nemery et al. 1992). In this workshop, personal air samples appeared to be artificially low in comparison to area air samples and urinary cobalt levels of the workers. When this workshop was excluded, there was a good correlation (R=0.85–0.88) between urinary cobalt and cobalt in the air. Based on urinary cobalt levels, the concentration of cobalt expected in personal air samples from workshop #9 was about 45 μg/m^3 (the mean value actually reported was 6 μg/m^3). The polishing workshops were divided into two groups: those with low exposure to cobalt (#1–5, n=102) and those with high exposure to cobalt (#6–10, n=91). Mean cobalt exposure concentrations were 0.4, 1.6, and 10.2 μg/m^3 by area air sampling and 0.4, 5.3, and 15.1 μg/m^3 by personal air sampling in the control, low-exposure, and high-exposure groups, respectively. The inclusion of the apparently biased personal air samples from workshop #9 means that the reported mean cobalt exposure in the high-exposure group obtained by personal air sampling (15.1 μg/m^3) may be lower than the true value. Air concentrations of iron were highest in the two polishing workshops that used iron disks and the sawing workshop (highest value =62 μg/m^3), and were not correlated with cobalt levels. Analysis of samples taken near the disks showed the presence of cobalt, with occasional traces of copper, zinc, titanium, manganese, chromium, silicates, and silicon dioxide. No tungsten was detected. There is a possibility that some workers had previously been exposed to asbestos, since pastes containing asbestos had been used in the past to glue the diamonds onto holders. However, the degree of asbestos exposure had apparently been insufficient to produce functional impairment. The researchers considered cobalt to be the only relevant exposure. Smoking habits were similar in workers from the high-exposure, low-exposure, and control groups. Duration of exposure was not discussed.

Effects noted in study and corresponding doses:

Workers in the high-exposure group were more likely than those in the other groups to complain about respiratory symptoms; the prevalences of eye, nose, and throat irritation and cough, and the fraction of these symptoms related to work, were significantly increased in the high-exposure group (Nemery et al. 1992). Workers in the high-exposure group also had significantly reduced lung function compared to controls and low-exposure group workers, as assessed by FVC (forced vital capacity), FEV_1 (forced expiratory volume in 1 second), MMEF (forced expiratory flow between 25 and 75% of the FVC), and mean PEF (peak expiratory flow rate), although the prevalence of abnormal values did not differ significantly between exposure categories. Results in the low-exposure group did not differ from controls. Two-way analysis of variance was used to show that the effect on spirometric parameters in the high exposure group was present in both men and women. Women seemed to be affected more than men, but the interaction between exposure and sex was not significant. Smoking was found to exert a strong effect on lung function, but lung function level remained negatively correlated with exposure to cobalt, independently of smoking.

Dose and end point used for MRL derivation:

[x] NOAEL [ ] LOAEL
Nemery et al. (1992) established a NOAEL of 0.0053 mg cobalt/m^3 for effects on pulmonary function (decreased values upon spirometric examination).

Uncertainty Factors used in MRL derivation:

[x] 1 [ ] 3 [ ] 10 (for use of a NOAEL)
[x] 1 [ ] 3 [ ] 10 (for extrapolation from animals to humans)
[ ] 1 [ ] 3 [x] 10 (for human variability)

The chronic inhalation MRL for cobalt is derived as follows:

$\text{MRL} = \text{NOAEL}_{[\text{ADJ}]} \div \text{UF}$
$\text{MRL} = 0.0013 \text{ mg cobalt/m}^3 \div 10$
$\text{MRL} = 1\text{x}10^{-4} \text{ mg cobalt/m}^3$

Was a conversion used from ppm in food or water to a mg/body weight dose? No.

Was a conversion used from intermittent to continuous exposure? If so, explain:

0.0053 mg cobalt/m^3 * (8 hours/24 hours) * (5 days/7 days) = 0.0013 mg cobalt/m^3 continuous exposure.

If an inhalation study in animals, list the conversion factors used in determining human equivalent dose: NA.

Other additional studies or pertinent information which lend support to this MRL:

Necrosis and inflammation of the respiratory tract epithelium (larynx, trachea, bronchioles, nasal turbinates) were reported in rats exposed to 19 mg cobalt/m^3 and mice exposed to 1.9 mg cobalt/m^3 (and above) as cobalt sulfate over 16 days (NTP 1991). Exposure of rats and mice to cobalt as cobalt sulfate for 13 weeks resulted in adverse effects on all parts of the respiratory tract, with the larynx being the most sensitive part (NTP 1991). At concentrations of ≥0.11 mg cobalt/m^3, rats and mice had squamous metaplasia of the larynx. Histiocytic infiltrates in the lung were also reported at similar levels in both the rats and mice. In rats, chronic inflammation of the larynx was found at ≥0.38 mg cobalt/m^3, and more severe effects on the larynx, nose, and lung were reported at higher exposures. In mice, acute inflammation of the nose was found at ≥1.14 mg cobalt/m^3, and more severe effects on the larynx, nose, and lung were reported at higher exposures.

Exposure of rats and mice to aerosols of cobalt (as cobalt sulfate) at concentrations from 0.11 to 1.14 mg cobalt/m^3 for 2 years resulted in a spectrum of inflammatory, fibrotic, and proliferative lesions in the respiratory tract of male and female rats and mice (NTP 1998). Squamous metaplasia of the larynx occurred in rats and mice at exposure concentrations of ≥0.11 mg cobalt/m^3, with severity of the lesion increasing with increased exposure concentration. Hyperplastic lesions of the nasal epithelium occurred in rats at concentrations of ≥0.11 mg cobalt/m^3, and in mice at concentrations of ≥0.38 mg cobalt/m^3. Both sexes of rats had greatly increased incidences (>90% incidence) of alveolar lesions at all exposure levels, including inflammatory changes, fibrosis, and metaplasia. Similar changes were seen in mice at all exposure levels, though the changes in mice were less severe.

Both studies by NTP (1991, 1998) failed to define a NOAEL, with the lowest concentration examined (0.11 mg/m^3) a LOAEL for a variety of respiratory effects. If an MRL were to be calculated based upon these studies, it would be as follows:

Duration adjustment: 0.11 mg cobalt/m^3 * (6 h/24 h) * (5 d/7 d) = 0.020 mg cobalt/m^3 continuous exposure.
Calculation of human equivalent concentration:
If fractional depositions in humans and animals are assumed to be equal, then:
RDDR = V_E(animal)/S_{ET}(animal) ÷ V_E(human)/S_{ET}(human) = 0.24 m^3/day / 15 cm^2 ÷ 20 m^3/day / 200 cm^2
RDDR = 0.16
$\text{LOAEL}_{[\text{HEC}]}$ = LOAEL[ADJ] * RDDR
= 0.020 mg cobalt/m^3 * 0.16 = 0.0032 mg cobalt/m^3

To the $\text{LOAEL}_{[\text{HEC}]}$, an uncertainty factor of 300 (10 for use of a LOAEL, 3 for animal to human extrapolation, and 10 for human variability) to derive an MRL of $1\text{x}10^{-5}$ mg/m^3. This number is an order of magnitude lower than the number derived from the Nemery et al. (1992) data, reflecting the fact that it is derived from animal data, not from a human study, and is based on a LOAEL, not a NOAEL. As the Nemery et al. (1992) study was a well-performed study in humans that defined a NOAEL and LOAEL, it was selected as the basis for derivation of the MRL.

Agency Contact (Chemical Manager): Obaid Faroon D.V.M., Ph.D.

MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical Name:	Cobalt
CAS Number:	10026-24-1
Date:	March 2004
Profile Status:	Final
Route:	[ ] Inhalation [x] Oral
Duration:	[ ] Acute [x] Intermediate [ ] Chronic
Key to figure:	30
Species:	human

Minimal Risk Level: 1×10^{-4} [x] mg/kg/day [ ] ppm [ ] mg/m^3

Reference:

Davis, J.E. and Fields, J.P. 1958. Experimental production of polycythemia in humans by administration of cobalt chloride. Proc Soc Exp Biol Med 99:493-495.

Experimental design:

Six apparently normal men, ages 20–47, were administered a daily dose of cobalt chloride, administered as a 2% solution diluted in either water or milk, for up to 22 days. Five of the six received 150 mg cobalt chloride per day for the entire exposure period, while the sixth was started on 120 mg/day and later increased to 150 mg/day. Blood samples were obtained daily from free-flowing punctures of fingertips at least 2 hours after eating, and at least 15 hours after the last dosage of cobalt. Blood was analyzed for red blood cell counts, hemoglobin percentage, leukocyte counts, reticulocyte percentages, and thrombocyte counts.

Effects noted in study and corresponding doses:

Exposure to cobalt resulted in the development of polycythemia in all six subjects, with increases in red blood cell numbers ranging from 0.5 to 1.19 million (~16–20% increase above pre-treatment levels). Polycythemic erythrocyte counts returned to normal 9–15 days after cessation of cobalt administration. Hemoglobin levels were also increased by cobalt treatment, though to a lesser extent than the erythrocyte values, with increases of 6–11% over pretreatment values. In five of the six subjects, reticulocyte levels were elevated, reaching at least twice the pre-experiment values. Thrombocyte and total leukocyte counts did not deviate significantly from pretreatment values.

Dose end point used for MRL derivation:

[ ] NOAEL [x] LOAEL

Davis and Fields (1958) identified a LOAEL of 150 mg cobalt chloride per day for increased levels of erythrocytes in volunteers. 150 mg cobalt chloride/day corresponds to ~1 mg Co/kg/day, assuming a reference body weight of 70 kg. Available animal studies, presented below, lend support to this LOAEL, having demonstrated LOAEL values within half an order of magnitude of that identified by Davis and Fields (1958).

Uncertainty factors used in MRL derivation:

[ ] 1 [ ] 3 [x] 10 (for use of a LOAEL)

[x] 1 [ ] 3 [ ] 10 (for extrapolation from animals to humans)
[ ] 1 [ ] 3 [x] 10 (for human variability)

The intermediate oral MRL for cobalt is derived as follows:

MRL = LOAEL ÷ UF
MRL = 1 mg cobalt/kg-day ÷ 100
MRL = 1×10^{-2} mg cobalt/kg-day

Was a conversion factor used from ppm in food or water to a mg/body weight dose? No.

Was a conversion used from intermittent to continuous exposure? If so, explain: No.

If an inhalation study in animals, list conversion factors used in determining human equivalent dose: Not applicable.

Other additional studies or pertinent information that lend support to this MRL:

No other studies of the effect of intermediate oral cobalt exposure on erythrocyte levels in healthy human subjects were identified in a search of the literature. Treatment of pregnant women for 90 days with 0.5–0.6 mg cobalt/kg/day as cobalt chloride did not prevent the reduction in hematocrit and hemoglobin levels often found during pregnancy (Holly 1955). However, treatment of anephric patients (with resulting anemia) with 0.16–1.0 mg cobalt/kg/day daily as cobalt chloride for 3–32 weeks resulted in increased levels of circulating erythrocytes and a decreased need for transfusions (Duckham and Lee 1976b; Taylor et al. 1977). While these studies provide additional evidence that exposure to cobalt can increase erythrocyte levels in humans, the fact that the patients were anephric makes definitive interpretation of the results more difficult.

Roche and Layrisse (1956) exposed volunteers to similar levels (150 mg $CoCl_2$/day) of cobalt, and reported a reversible decrease in uptake of ^{131}I by the thyroid. The decreased uptake is believed to result from cobalt blocking the organic binding of iodine (Paley et al. 1958). This observation adds support to the choice of effect level, as a similar exposure resulted in measurable effects in volunteers, though whether the changes in iodine uptake operate through the same mechanisms as the changes in erythrocyte numbers has not been determined.

Stanley et al. (1947) exposed groups (n=4, 6 for controls) of 6 Sprague-Dawley rats to 0, 0.62, 2.5, or 10 mg cobalt/kg/day (0, 2.5, 10, or 40 mg/kg-day of $CoCl_2 \cdot 6H_2O$) in gelatin capsules for 8 weeks. Blood counts and hemoglobin levels were examined at the beginning of the experiment and at 2-week intervals. Rats exposed to 0.62 mg cobalt/kg-day showed no change in erythrocyte number. At 2.5 mg cobalt/kg-day, a progressive increase in erythrocyte number was seen, increasing up to a maximum of 17% above pretreatment values on week 6. At the highest exposure level, a progressive increase in erythrocyte numbers was seen, reaching 29% above pretreatment values at 8 weeks of exposure. Statistical analyses of the group means were not provided, and the study provided only mean values of the measurements, precluding statistical analysis. However, if a 10% change is assumed to be an effect level, exposure to 2.5 mg cobalt/kg-day was the LOAEL for this study, with a NOAEL of 0.62 mg cobalt/kg-day.

Krasovskii and Fridyland (1971) exposed groups of rats to 0, 0.05, 0.5, or 2.5 mg Co/kg/day for up to 7 months. In the 2.5 mg/kg-day group, a persistent increase in erythrocyte levels was seen. The increase was transient in the 0.5 mg/kg/day rats, and was not present in rats exposed to 0.05 mg/kg/day. However, numerical data were not presented and statistical significance was not reported.

A number of other studies in animals have reported increases in erythrocyte levels following intermediate oral administration of cobalt compounds (see the LSE table for further details of these studies). However, the majority of them have considerable methodological limitations, including examination of either very high exposure levels or only one exposure level, limited reporting of results, or limited or no statistical analysis.

Whether or not polycythemia, a condition wherein an excess of erythrocytes is produced, constitutes an adverse effect is open to interpretation. At the levels seen in the available studies, and in particular in the Davis and Fields (1958) study, the subjects would be expected to be asymptomatic. However, data on the long-term effects of elevated erythrocyte levels are not available. As such, this end point was considered an adverse effect as a health-protective assumption, and was utilized as a critical end point for MRL derivation.

Agency Contact (Chemical Manager): Obaid Faroon D.V.M., Ph.D.

MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical name:	Radioactive Cobalt
CAS number:	Multiple
Date:March	2004
Profile status:	Final
Route:	[ ] Inhalation [ ] Oral [x] External
Duration:	[x] Acute [ ] Intermediate [ ] Chronic
Species:	Human

Minimal Risk Level: 4 [ ] mg/kg/day [ ] ppm [ ] mg/m^3 [x] mSv (400 mrem)

References:

Schull WJ, Otake M, Yoshimaru H. 1988. Effect on intelligence test score of prenatal exposure to ionizing radiation in Hiroshima and Nagasaki: A comparison of the T65DR and DS86 dosimetry systems. Radiation Effects Research Foundation (RERF) Technical Report No. 3-88. Hirroshima, Japan. NTIS Report Number: DE89-906462.

Burt C. 1966. The genetic determination of differences in intelligence: A study of monozygotic twins reared together and apart. Brit J Psychol 57(1&2):137-153.

Experimental design:

Schull et al. (1988) study: Schull et al. (1988) evaluated the quantitative effect of exposure to ionizing radiation on the developing fetal and embryonic human brain. The end point measured was changes in intelligence test scores. The effects on individuals exposed *in utero* to the atomic bombing of Hiroshima and Nagasaki were based on the original PE86 samples (n=1,759; data on available intelligence testing) and a clinical sample (n=1,598). The original PE86 sample included virtually all prenatally exposed individuals who received tissue-absorbed doses of 0.50 Gy or more. There were many more individuals in the dose range 0–0.49 Gy in the PE86 sample than in the clinical sample. The clinical sample does not include children prenatally exposed at distances between 2,000 and 2,999 m in Hiroshima and Nagasaki. Children exposed at greater distances or not present in the city were selected as controls. In 1955–1956, Tanaka-B (emphasis on word-sense, arithmetic abilities, and the like, which were associated with the more subtle processing of visual clues than their simple recognition and depended more on connectedness) and the Koga (emphasis on perception of spatial relationships) intelligence tests were conducted in Nagasaki and the Koga test in Hiroshima.

Burt (1966) study: This study determined differences in intelligence in monozygotic twins reared together (n=95) and apart (n=53). All tests conducted in school consisted of (1) a group test of intelligence containing both non-verbal and verbal items, (2) an individual test (the London Revision of the Terman-Binet Scale) used primarily for standardization and for doubtful cases, and (3) a set of performance tests, based on the Pitner-Paterson tests and standardization. The methods and standard remained much the same throughout the study. Some of the reasons for separation of the twins were given as follows: death of the mother (n=9), unable to bring them up properly, mother's poor health (n=12), unmarried (n=6), and economic difficulties. The children were brought up by parents or foster parents (occupation ranged from unskilled to professional). IQ scores in the study group ranged from 66 to 137. The standard deviation of the group of separated monozygotic twins was reported at 15.3 as compared to 15.0 of ordinary siblings. Twins brought up in different environments were compared with those brought up in similar circumstances.

Effects noted in study and corresponding doses:

Schull et al. (1986) study: No evidence of radiation-related effect on intelligence was observed among individuals exposed within 0–7 weeks after fertilization or in the 26th or subsequent weeks. The highest risk of radiation damage to the embryonic and fetal brain occurs 8–15-weeks after fertilization under both dosimetric systems. The regression of intelligence score on estimated DS86 uterine absorbed dose is linear with dose, and the diminution in intelligence score is 21–29 points per Gy for the 8–15-week group and 10–26 points per Gy for the 16–25-week group. The results for 8–15 weeks applies regardless whether or not the mentally retarded individuals were included. The cumulative distribution of test scores suggested a progressive shift downwards in individual scores with increasing exposure. The mean IQ scores decrease significantly and systematically with uterine or fetal tissue dose within the 8–15- and 16–25-week groups.

In summary, analysis of intelligence test scores at 10–11 years of age of individuals exposed prenatally showed that:

- There is no evidence of a radiation-related effect on intelligence scores among those individuals exposed within 0–7 weeks of fertilization or in the 26th week of gestation and beyond;
- The cumulative distribution of test scores suggests a progressive shift downwards in intelligence scores with increasing exposure to ionizing radiation (dose-response relationship).
- The most sensitive group was the 8–15 weeks exposure group. The regression in intelligence scores was found to be linear, with 1 Gy dose resulting in a 21–29 point decline in intelligence scores.
- There was no indication of groups of individuals with differing sensitivities to radiation.

Burt (1966) study: The average intelligence of the twins measured on a conventional IQ scale (SD=15) was 97.8 for the separated monozygotes, 98.1 for monozygotes brought up together, 99.3 for the dizygotes as compared with 100.2 for the siblings, and 100.0 for the population as a whole. The difference of 0.3 IQ point between the separated and unseparated identical twins is considered a NOAEL for this study.

Dose endpoint used for MRL derivation:

[x] NOAEL [ ] LOAEL 0.3 IQ point reduction in twins, between those raised together and those raised apart.

Uncertainty factors (UF) used in MRL derivation:

[x] 1 [ ] 3 [ ] 10 (for use of a NOAEL)
[x] 1 [ ] 3 [ ] 10 (for extrapolation from animals to humans)
[ ] 1 [x] 3 [ ] 10 (for human variability/sensitive population)

Was a conversion factor used from ppm in food or water to a mg/body weight dose? If so, explain: No.

If an inhalation study in animals, list conversion factors used in determining human equivalent dose: NA

Was a conversion used from intermittent to continuous exposure? No.

Other additional studies or pertinent information that lend support to this MRL:

Husen (1959) reported a study involving 269 pairs of Swedish monozygotic (identical) twins where the intrapair IQ difference was 4 IQ points for a combination of twins raised together and apart. This is somewhat lower than the value of 7 IQ points for identical twins raised apart, and just larger than the range of IQ scores for Washington, DC children repetitively tested (Jacobi and Glauberman 1995).

Supporting evidence for the acute MRL is provided by Jacobi and Glauberman (1995). Children in the 1st, 3rd, and 5th grades born in Washington, DC were tested, and average IQ levels of 94.2, 97.6, and 94.6, respectively, were reported. The range of 3.4 IQ points is considered to be a LOAEL for this study, which, if used for MRL derivation, would yield an MRL of 0.004 Sv (3.4 IQ points x 1 Sv/25 IQ points ÷ 30 [10 for use of a LOAEL and 3 for a sensitive population]).

Additional supporting evidence for the acute MRL is provided by Berger et al. 1997, in a case study of accidental radiation injury to the hand. A Mexican engineer suffered an accidental injury to the hand while repairing an x-ray spectrometer. The day after the accident, his symptoms included a tingling sensation and itching in the index and middle fingers. On days 4 and 7, a "pinching" sensation, swelling, and slight erythema were observed. By day 7, the tip of his index fingers was erythematous and a large blister developed with swelling on other fingers. On day 10, examination by a physician showed that the lesions had worsened and the fingers and palms were discolored. On day 10, he was admitted to the hospital where hyperbaric oxygen therapy was administered without success. One month after the accident, the patient entered the hospital again with pain, discoloration, and desquamation of his hand. Clinical examination showed decreased circulation in the entire hand, most notably in the index and middle finger. Total white blood count decreased to 3,000/μL (normal range 4,300–10,800/μL). Cytogenic studies of peripheral blood lymphocytes revealed four dicentrics, two rings, and eight chromosomal fragments in the 300 metaphases studied. The estimated whole body dose was reported to be 0.382 Gy (38.2 rad). This dose is a potential LOAEL for acute ionizing radiation and would yield an MRL of 0.004 Sv (0.38 Sv ÷100 [10 for use of LOAEL and 10 for sensitive human population]).

The USNRC set a radiation exposure limit of 0.5 rem (50 mSv) for pregnant working women over the full gestational period (USNRC 1991). For the critical gestational period of 8–15 weeks, ATSDR believes that the conservative acute MRL of 4 mSv is consistent with the USNRC limit and could be applied to either acute (0–14-day) or intermediate (15–365-day) exposure periods.

Calculations

Given: 0.3 IQ point is a NOAEL. A 1 Sv dose results in a 25 IQ point reduction (range=21–29 points; mean=25) and provides a conversion factor from IQ prediction to radiation dose. Assume that the radiation dose and the subsequent reduction in IQ is a linear relationship.
MRL = NOAEL x CF ÷ UF
MRL = 0.3 x 1/25 ÷ 3
MRL = 0.004 Sv = 4 mSv (400 mrem)

Agency Contact (Chemical Manager): Obaid Faroon D.V.M., Ph.D.

MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical Name:	Radioactive Cobalt
CAS Number:	Multiple
Date:	March 2004
Profile Status:	Final
Route:	[ ] Inhalation [ ] Oral [X] External
Duration:	[ ] Acute [ ] Intermediate [X] Chronic
Species:	Human

Minimal Risk Level: 1 [ ] mg/kg/day [ ] ppm [ ] mg/m^3 [X] mSv/year (100 mrem/year)

Reference: BEIR V. 1990. Health effects of exposure to low levels of ionizing radiation. Committee on the Biological Effects of Ionizing Radiations, National Research Council. National Academy Press. Washington, DC.

Experimental design: Not applicable

Effects noted in study and corresponding doses: No individual studies were identified that could be used to base a chronic-duration external exposure MRL that did not result in a cancer-producing end point. However, two sources of information were identified that did provide doses of ionizing radiation that have not been reported to be associated with detrimental effects (NOAELs). These sources provide estimates of background levels of primarily natural sources of ionizing radiation that have not been implicated in producing cancerous or noncancerous toxicological endpoints. BEIR V states that the average annual effective dose to the U.S. population is 3.6 mSv/year. A total annual effective dose equivalent of 3.6 mSv (360 mrem)/year to members of the U.S. population is obtained mainly by naturally occurring radiation from external sources, medical uses of radiation, and radiation from consumer products. The largest contribution (82%) is from natural sources, two-thirds of which is from naturally occurring radon and its decay products. Specific sources of this radiation are demonstrated in Table A-1.

The annual dose of 3.6 mSv per year has not been associated with adverse health effects or increases in the incidences of any type of cancers in humans or other animals.

Dose and end point used for MRL derivation: 3.6 mSv/year

[X] NOAEL [ ] LOAEL 3.6 mSv/year

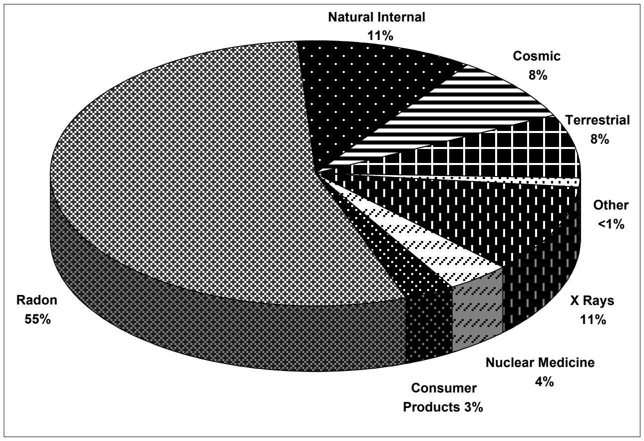
Uncertainty Factors used in MRL derivation:

[X] 1 [ ] 3 [ ] 10 (for use of a NOAEL)
[X] 1 [ ] 3 [ ] 10 (for extrapolation from animals to humans)
[ ] 1 [X] 3 [ ] 10 (for human variability)

Was a conversion used from ppm in food or water to a mg/body weight dose? No.

Table A-1. Average Annual Effective Dose Equivalent from Ionizing Radiation to a Member of the U.S. Population[a]

	Effective dose equivalent	
Source	mSv	Percent of total dose
Natural		
Radon[b]	2.0	55
Cosmic	0.27	8.0
Terrestrial	0.28	8.0
Internal	0.39	11
Total natural	3.0	82
Artificial		
Medical		
X-ray	0.39	11
Nuclear	0.14	4.0
Consumer products	0.10	3.0
Other		
Occupational	<0.01	<0.3
Nuclear fuel cycle	<0.01	<0.03
Fallout	<0.01	<0.03
Miscellaneous[c]	<0.01	<0.03
Total artificial	0.63	18
Total natural and artificial	3.6	100



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[a] Adapted from BEIR V, Table 1-3 , page 18.

[b] Dose equivalent to bronchi from radon daughter products

[c] DOE facilities, smelter, transportation, etc.

If an inhalation study in animals, list the conversion factors used in determining human equivalent dose: Not applicable.

Was a conversion used from intermittent to continuous exposure? No.

Other additional studies or pertinent information which lend support to this MRL: ICRP has developed recommended dose limits for occupational and public exposure to ionizing radiation sources. The ICRP recommends limiting public exposure to 1 mSv/year (100 mrem/year), but does note that values at high altitudes above sea level and in some geological areas can sometimes be twice that value (≥2 mSv). In Annex C of ICRP 60, the commission provides data that suggests increasing the dose from 1 mSv to 5 mSv results in a very small, but detectable, increase in age-specific human mortality rate. ICRP states that the value of 1 mSv/year was chosen over the 5 mSv value because 5 mSv/year (500 mrem/year) causes this increase in age specific mortality rate, and 1 mSv/year (100 mrem/year) is typical of the annual effective dose from background, less radon (ICRP 1991). The 1 mSv estimate may underestimate the annual exposure to external sources of ionizing radiation to the U.S. population, as it does not include radiation from radon. Conversely, the 5 mSv estimate may be high, in that increases in mortality rate been reported. The most useful estimate appears to be the BEIR V estimate of 3.6 mSv, in that it accounts for an annual exposure to radon, is specific to the U.S. population, has not been associated with increases mortality, and it falls short of the 5 mSv value associated with small increases in human mortality.

Calculations:

MRL = $\mathrm{NOAEL}_{(\mathrm{ADJ})} \div \mathrm{UF}$
MRL = 3.6 mSv/year ÷ 3
MRL = 1.20 mSv/year
MRL = 1.0 mSv/year =100 mrem/year above background

Agency Contact (Chemical Manager): Obaid Faroon D.V.M., Ph.D.

APPENDIX B. USER'S GUIDE

Chapter 1

Public Health Statement

This chapter of the profile is a health effects summary written in non-technical language. Its intended audience is the general public, especially people living in the vicinity of a hazardous waste site or chemical release. If the Public Health Statement were removed from the rest of the document, it would still communicate to the lay public essential information about the chemical.

The major headings in the Public Health Statement are useful to find specific topics of concern. The topics are written in a question and answer format. The answer to each question includes a sentence that will direct the reader to chapters in the profile that will provide more information on the given topic.

Chapter 2

Relevance to Public Health

This chapter provides a health effects summary based on evaluations of existing toxicologic, epidemiologic, and toxicokinetic information. This summary is designed to present interpretive, weight-of-evidence discussions for human health end points by addressing the following questions.

1. What effects are known to occur in humans?
2. What effects observed in animals are likely to be of concern to humans?
3. What exposure conditions are likely to be of concern to humans, especially around hazardous waste sites?

The chapter covers end points in the same order that they appear within the Discussion of Health Effects by Route of Exposure section, by route (inhalation, oral, and dermal) and within route by effect. Human data are presented first, then animal data. Both are organized by duration (acute, intermediate, chronic). *In vitro* data and data from parenteral routes (intramuscular, intravenous, subcutaneous, etc.) are also considered in this chapter.

The carcinogenic potential of the profiled substance is qualitatively evaluated, when appropriate, using existing toxicokinetic, genotoxic, and carcinogenic data. ATSDR does not currently assess cancer potency or perform cancer risk assessments. Minimal Risk Levels (MRLs) for noncancer end points (if derived) and the end points from which they were derived are indicated and discussed.

Limitations to existing scientific literature that prevent a satisfactory evaluation of the relevance to public health are identified in the Chapter 3 Data Needs section.

Interpretation of Minimal Risk Levels

Where sufficient toxicologic information is available, ATSDR has derived MRLs for inhalation and oral routes of entry at each duration of exposure (acute, intermediate, and chronic). These MRLs are not meant to support regulatory action, but to acquaint health professionals with exposure levels at which adverse health effects are not expected to occur in humans.

MRLs should help physicians and public health officials determine the safety of a community living near a chemical emission, given the concentration of a contaminant in air or the estimated daily dose in water. MRLs are based largely on toxicological studies in animals and on reports of human occupational exposure.

MRL users should be familiar with the toxicologic information on which the number is based. Chapter 2, "Relevance to Public Health," contains basic information known about the substance. Other sections such as Chapter 3 Section 3.10, "Interactions with Other Substances," and Section 3.11, "Populations that are Unusually Susceptible" provide important supplemental information.

MRL users should also understand the MRL derivation methodology. MRLs are derived using a modified version of the risk assessment methodology that the Environmental Protection Agency (EPA) provides (Barnes and Dourson 1988) to determine reference doses (RfDs) for lifetime exposure.

To derive an MRL, ATSDR generally selects the most sensitive end point which, in its best judgement, represents the most sensitive human health effect for a given exposure route and duration. ATSDR cannot make this judgement or derive an MRL unless information (quantitative or qualitative) is available for all potential systemic, neurological, and developmental effects. If this information and reliable quantitative data on the chosen end point are available, ATSDR derives an MRL using the most sensitive species (when information from multiple species is available) with the highest no-observed-adverse-effect level (NOAEL) that does not exceed any adverse effect levels. When a NOAEL is not available, a lowest-observed-adverse-effect level (LOAEL) can be used to derive an MRL, and an uncertainty factor (UF) of 10 must be employed. Additional uncertainty factors of 10 must be used both for human variability to protect sensitive subpopulations (people who are most susceptible to the health effects caused by the substance) and for interspecies variability (extrapolation from animals to humans). In deriving an MRL, these individual uncertainty factors are multiplied together. The product is then divided into the inhalation concentration or oral dosage selected from the study. Uncertainty factors used in developing a substance-specific MRL are provided in the footnotes of the levels of significant exposure (LSE) Tables.

Chapter 3

Health Effects

Tables and Figures for Levels of Significant Exposure (LSE)

Tables and figures are used to summarize health effects and illustrate graphically levels of exposure associated with those effects. These levels cover health effects observed at increasing dose concentrations and durations, differences in response by species, MRLs to humans for noncancer end points, and EPA's estimated range associated with an upper- bound individual lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. Use the LSE tables and figures for a quick review of the health effects and to locate data for a specific exposure scenario. The LSE tables and figures should always be used in conjunction with the text. All entries in these tables and figures represent studies that provide reliable, quantitative estimates of NOAELs, LOAELs, or Cancer Effect Levels (CELs).

The legends presented below demonstrate the application of these tables and figures. Representative examples of LSE Table 3-1 and Figure 3-1 are shown. The numbers in the left column of the legends correspond to the numbers in the example table and figure.

LEGEND

See Sample LSE Table 3-1 (page B-6)

(1) Route of Exposure. One of the first considerations when reviewing the toxicity of a substance using these tables and figures should be the relevant and appropriate route of exposure. Typically when sufficient data exists, three LSE tables and two LSE figures are presented in the document. The three LSE tables present data on the three principal routes of exposure, i.e., inhalation, oral, and dermal (LSE Table 3-1, 3-2, and 3-3, respectively). LSE figures are limited to the inhalation (LSE Figure 3-1) and oral (LSE Figure 3-2) routes. Not all substances will have data on each route of exposure and will not, therefore, have all five of the tables and figures.

(2) Exposure Period. Three exposure periods—acute (less than 15 days), intermediate (15–364 days), and chronic (365 days or more)—are presented within each relevant route of exposure. In this example, an inhalation study of intermediate exposure duration is reported. For quick reference to health effects occurring from a known length of exposure, locate the applicable exposure period within the LSE table and figure.

(3) Health Effect. The major categories of health effects included in LSE tables and figures are death, systemic, immunological, neurological, developmental, reproductive, and cancer. NOAELs and LOAELs can be reported in the tables and figures for all effects but cancer. Systemic effects are further defined in the "System" column of the LSE table (see key number 18).

(4) Key to Figure. Each key number in the LSE table links study information to one or more data points using the same key number in the corresponding LSE figure. In this example, the study represented by key number 18 has been used to derive a NOAEL and a Less Serious LOAEL (also see the two "18r" data points in sample Figure 3-1).

(5) Species. The test species, whether animal or human, are identified in this column. Chapter 2, "Relevance to Public Health," covers the relevance of animal data to human toxicity and Section 3.5, "Toxicokinetics," contains any available information on comparative toxicokinetics. Although NOAELs and LOAELs are species specific, the levels are extrapolated to equivalent human doses to derive an MRL.

(6) Exposure Frequency/Duration. The duration of the study and the weekly and daily exposure regimen are provided in this column. This permits comparison of NOAELs and LOAELs from different studies. In this case (key number 18), rats were exposed to “Chemical x” via inhalation for 6 hours/day, 5 days/week, for 13 weeks. For a more complete review of the dosing regimen refer to the appropriate sections of the text or the original reference paper (i.e., Nitschke et al. 1981).

(7) System. This column further defines the systemic effects. These systems include respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, and dermal/ocular. "Other" refers to any systemic effect (e.g., a decrease in body weight) not covered in these systems. In the example of key number 18, one systemic effect (respiratory) was investigated.

(8) NOAEL. A NOAEL is the highest exposure level at which no harmful effects were seen in the organ system studied. Key number 18 reports a NOAEL of 3 ppm for the respiratory system, which was used to derive an intermediate exposure, inhalation MRL of 0.005 ppm (see footnote "b").

(9) LOAEL. A LOAEL is the lowest dose used in the study that caused a harmful health effect. LOAELs have been classified into "Less Serious" and "Serious" effects. These distinctions help readers identify the levels of exposure at which adverse health effects first appear and the gradation of effects with increasing dose. A brief description of the specific end point used to quantify the adverse effect accompanies the LOAEL. The respiratory effect reported in key number 18 (hyperplasia) is a Less Serious LOAEL of 10 ppm. MRLs are not derived from Serious LOAELs.

(10) Reference. The complete reference citation is given in Chapter 9 of the profile.

(11) CEL. A CEL is the lowest exposure level associated with the onset of carcinogenesis in experimental or epidemiologic studies. CELs are always considered serious effects. The LSE tables and figures do not contain NOAELs for cancer, but the text may report doses not causing measurable cancer increases.

(12) Footnotes. Explanations of abbreviations or reference notes for data in the LSE tables are found in the footnotes. Footnote "b" indicates that the NOAEL of 3 ppm in key number 18 was used to derive an MRL of 0.005 ppm.

LEGEND

See Sample Figure 3-1 (page B-7)

LSE figures graphically illustrate the data presented in the corresponding LSE tables. Figures help the reader quickly compare health effects according to exposure concentrations for particular exposure periods.

(13) Exposure Period. The same exposure periods appear as in the LSE table. In this example, health effects observed within the acute and intermediate exposure periods are illustrated.

(14) Health Effect. These are the categories of health effects for which reliable quantitative data exists. The same health effects appear in the LSE table.

(15) Levels of Exposure. Concentrations or doses for each health effect in the LSE tables are graphically displayed in the LSE figures. Exposure concentration or dose is measured on the log scale "y" axis. Inhalation exposure is reported in mg/m^3 or ppm and oral exposure is reported in mg/kg/day.

(16) NOAEL. In this example, the open circle designated 18r identifies a NOAEL critical end point in the rat upon which an intermediate inhalation exposure MRL is based. The key number 18 corresponds to the entry in the LSE table. The dashed descending arrow indicates the extrapolation from the exposure level of 3 ppm (see entry 18 in the Table) to the MRL of 0.005 ppm (see footnote "b" in the LSE table).

(17) CEL. Key number 38r is one of three studies for which CELs were derived. The diamond symbol refers to a CEL for the test species-mouse. The number 38 corresponds to the entry in the LSE table.

(18) Estimated Upper-Bound Human Cancer Risk Levels. This is the range associated with the upper-bound for lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. These risk levels are derived from the EPA's Human Health Assessment Group's upper-bound estimates of the slope of the cancer dose response curve at low dose levels (q_1*).

(19) Key to LSE Figure. The Key explains the abbreviations and symbols used in the figure.

SAMPLE

1 → **TABLE 3-1. Levels of Significant Exposure to [Chemical x] – Inhalation**

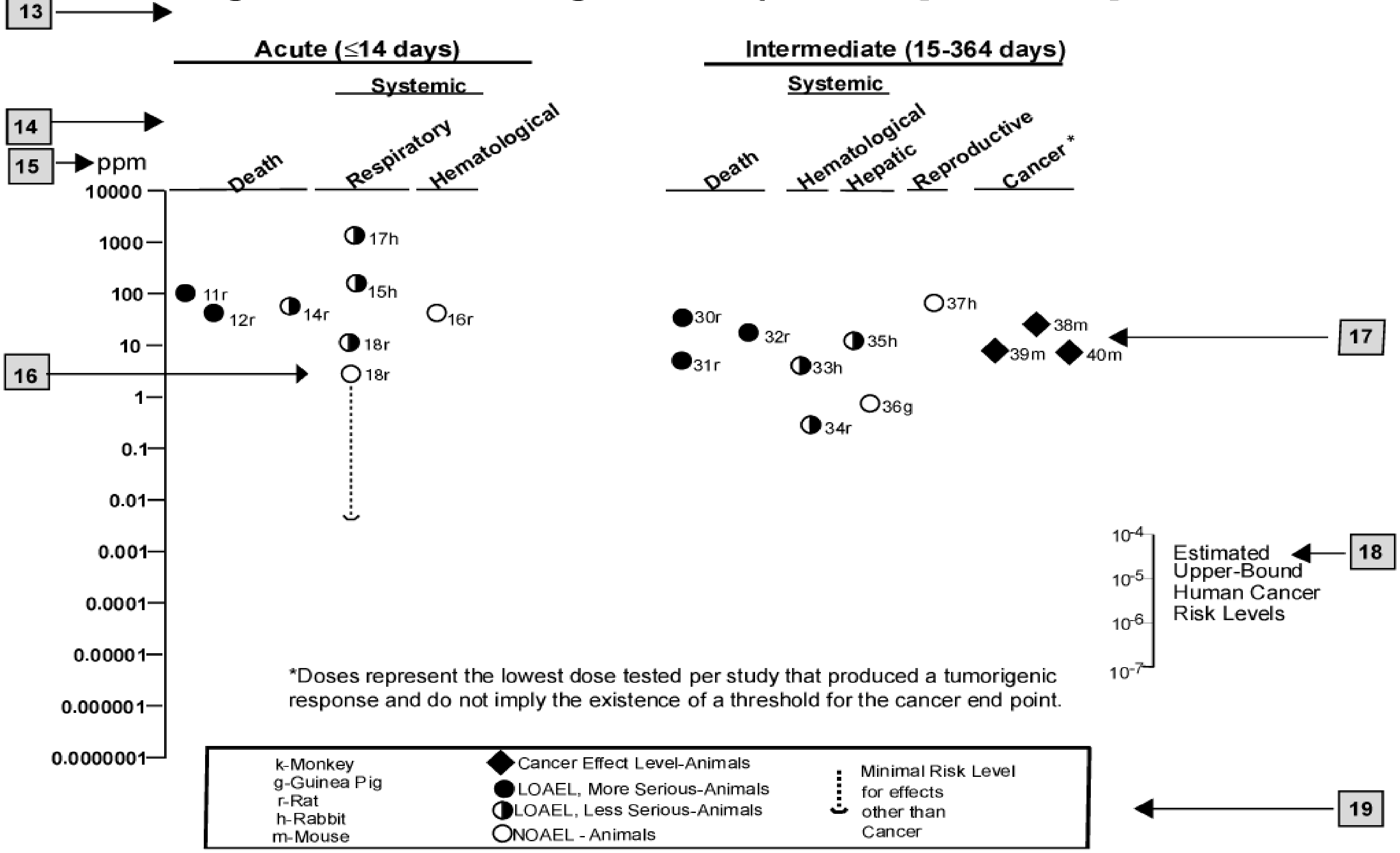
	Key to figure[a]	Species	Exposure frequency/ duration	System	NOAEL (ppm)	LOAEL (effect) Less serious (ppm)	LOAEL (effect) Serious (ppm)	Reference
2 →	INTERMEDIATE EXPOSURE							
		5	6	7	8	9		10
3 →	Systemic	↓	↓	↓	↓	↓		↓
4 →	18	Rat	13 wk 5 d/wk 6 hr/d	Resp	3[b]	10 (hyperplasia)		Nitschke et al. 1981
	CHRONIC EXPOSURE							
	Cancer						11 ↓	
	38	Rat	18 mo 5 d/wk 7 hr/d				20 (CEL, multiple organs)	Wong et al. 1982
	39	Rat	89-104 wk 5 d/wk 6 hr/d				10 (CEL, lung tumors, nasal tumors)	NTP 1982
	40	Mouse	79-103 wk 5 d/wk 6 hr/d				10 (CEL, lung tumors, hemangiosarcomas)	NTP 1982

12 → [a] The number corresponds to entries in Figure 3-1.

[b] Used to derive an intermediate inhalation Minimal Risk Level (MRL) of 5x10-3 ppm; dose adjusted for intermittent exposure and divided by an uncertainty factor of 100 (10 for extrapolation from animal to humans, 10 for human variability).

SAMPLE

Figure 3-1. Levels of Significant Exposure to [Chemical X] - Inhalation



*Doses represent the lowest dose tested per study that produced a tumorigenic response and do not imply the existence of a threshold for the cancer end point.

APPENDIX C. ACRONYMS, ABBREVIATIONS, AND SYMBOLS

Some terms are generic and may not be used in this profile.

ACGIH	American Conference of Governmental Industrial Hygienists
ACOEM	American College of Occupational and Environmental Medicine
ADI	acceptable daily intake
ADME	absorption, distribution, metabolism, and excretion
AED	atomic emission detection
AFID	alkali flame ionization detector
AFOSH	Air Force Office of Safety and Health
ALI	annual limit on intake
ALT	alanine aminotransferase
AML	acute myeloid leukemia
AOAC	Association of Official Analytical Chemists
AOEC	Association of Occupational and Environmental Clinics
AP	alkaline phosphatase
APHA	American Public Health Association
AST	aspartate aminotransferase
atm	atmosphere
ATSDR	Agency for Toxic Substances and Disease Registry
AWQC	Ambient Water Quality Criteria
BAT	best available technology
BCF	bioconcentration factor
BEI	Biological Exposure Index
BMD	benchmark dose
BMR	benchmark response
BSC	Board of Scientific Counselors
C	centigrade
CAA	Clean Air Act
CAG	Cancer Assessment Group of the U.S. Environmental Protection Agency
CAS	Chemical Abstract Services
CDC	Centers for Disease Control and Prevention
CEL	cancer effect level
CELDS	Computer-Environmental Legislative Data System
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFR	Code of Federal Regulations
Ci	curie
CI	confidence interval
CL	ceiling limit value
CLP	Contract Laboratory Program
cm	centimeter
CML	chronic myeloid leukemia
CPSC	Consumer Products Safety Commission
CWA	Clean Water Act
DAC	derived air concentration
DHEW	Department of Health, Education, and Welfare
DHHS	Department of Health and Human Services
DNA	deoxyribonucleic acid
DOD	Department of Defense

DOE	Department of Energy
DOL	Department of Labor
DOT	Department of Transportation
DOT/UN/ NA/IMCO	Department of Transportation/United Nations/ North America/International Maritime Dangerous Goods Code
DWEL	drinking water exposure level
ECD	electron capture detection
ECG/EKG	electrocardiogram
EEG	electroencephalogram
EEGL	Emergency Exposure Guidance Level
EPA	Environmental Protection Agency
F	Fahrenheit
F_1	first-filial generation
FAO	Food and Agricultural Organization of the United Nations
FDA	Food and Drug Administration
FEMA	Federal Emergency Management Agency
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
FPD	flame photometric detection
fpm	feet per minute
FR	Federal Register
FSH	follicle stimulating hormone
g	gram
GC	gas chromatography
gd	gestational day
GLC	gas liquid chromatography
GPC	gel permeation chromatography
HPLC	high-performance liquid chromatography
HRGC	high resolution gas chromatography
HSDB	Hazardous Substance Data Bank
IARC	International Agency for Research on Cancer
IDLH	immediately dangerous to life and health
ILO	International Labor Organization
IRIS	Integrated Risk Information System
Kd	adsorption ratio
kg	kilogram
kkg	metric ton
K_{oc}	organic carbon partition coefficient
K_{ow}	octanol-water partition coefficient
L	liter
LC	liquid chromatography
LC_{50}	lethal concentration, 50% kill
LC_{Lo}	lethal concentration, low
LD_{50}	lethal dose, 50% kill
LD_{Lo}	lethal dose, low
LDH	lactic dehydrogenase
LH	luteinizing hormone
LOAEL	lowest-observed-adverse-effect level
LSE	Levels of Significant Exposure
LT_{50}	lethal time, 50% kill
m	meter
MA	*trans,trans*-muconic acid

MAL	maximum allowable level
mCi	millicurie
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
MFO	mixed function oxidase
mg	milligram
mL	milliliter
mm	millimeter
mmHg	millimeters of mercury
mmol	millimole
mppcf	millions of particles per cubic foot
MRL	Minimal Risk Level
MS	mass spectrometry
NAAQS	National Ambient Air Quality Standard
NAS	National Academy of Science
NATICH	National Air Toxics Information Clearinghouse
NATO	North Atlantic Treaty Organization
NCE	normochromatic erythrocytes
NCEH	National Center for Environmental Health
NCI	National Cancer Institute
ND	not detected
NFPA	National Fire Protection Association
ng	nanogram
NHANES	National Health and Nutrition Examination Survey
NIEHS	National Institute of Environmental Health Sciences
NIOSH	National Institute for Occupational Safety and Health
NIOSHTIC	NIOSH's Computerized Information Retrieval System
NLM	National Library of Medicine
nm	nanometer
nmol	nanomole
NOAEL	no-observed-adverse-effect level
NOES	National Occupational Exposure Survey
NOHS	National Occupational Hazard Survey
NPD	nitrogen phosphorus detection
NPDES	National Pollutant Discharge Elimination System
NPL	National Priorities List
NR	not reported
NRC	National Research Council
NS	not specified
NSPS	New Source Performance Standards
NTIS	National Technical Information Service
NTP	National Toxicology Program
ODW	Office of Drinking Water, EPA
OERR	Office of Emergency and Remedial Response, EPA
OHM/TADS	Oil and Hazardous Materials/Technical Assistance Data System
OPP	Office of Pesticide Programs, EPA
OPPT	Office of Pollution Prevention and Toxics, EPA
OPPTS	Office of Prevention, Pesticides and Toxic Substances, EPA
OR	odds ratio
OSHA	Occupational Safety and Health Administration

OSW	Office of Solid Waste, EPA
OTS	Office of Toxic Substances
OW	Office of Water
OWRS	Office of Water Regulations and Standards, EPA
PAH	polycyclic aromatic hydrocarbon
PBPD	physiologically based pharmacodynamic
PBPK	physiologically based pharmacokinetic
PCE	polychromatic erythrocytes
PEL	permissible exposure limit
pg	picogram
PHS	Public Health Service
PID	photo ionization detector
pmol	picomole
PMR	proportionate mortality ratio
ppb	parts per billion
ppm	parts per million
ppt	parts per trillion
PSNS	pretreatment standards for new sources
RBC	red blood cell
REL	recommended exposure level/limit
RfC	reference concentration
RfD	reference dose
RNA	ribonucleic acid
RQ	reportable quantity
RTECS	Registry of Toxic Effects of Chemical Substances
SARA	Superfund Amendments and Reauthorization Act
SCE	sister chromatid exchange
SGOT	serum glutamic oxaloacetic transaminase
SGPT	serum glutamic pyruvic transaminase
SIC	standard industrial classification
SIM	selected ion monitoring
SMCL	secondary maximum contaminant level
SMR	standardized mortality ratio
SNARL	suggested no adverse response level
SPEGL	Short-Term Public Emergency Guidance Level
STEL	short term exposure limit
STORET	Storage and Retrieval
TD_{50}	toxic dose, 50% specific toxic effect
TLV	threshold limit value
TOC	total organic carbon
TPQ	threshold planning quantity
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
TWA	time-weighted average
UF	uncertainty factor
U.S.	United States
USDA	United States Department of Agriculture
USGS	United States Geological Survey
USNRC	United States Nuclear Regulatory Commission
VOC	volatile organic compound

WBC	white blood cell
WHO	World Health Organization

>	greater than
≥	greater than or equal to
=	equal to
<	less than
≤	less than or equal to
%	percent
α	alpha
β	beta
γ	gamma
δ	delta
µm	micrometer
µg	microgram
q_1^*	cancer slope factor
–	negative
+	positive
(+)	weakly positive result
(–)	weakly negative result

APPENDIX D. OVERVIEW OF BASIC RADIATION PHYSICS, CHEMISTRY, AND BIOLOGY

Understanding the basic concepts in radiation physics, chemistry, and biology is important to the evaluation and interpretation of radiation-induced adverse health effects and to the derivation of radiation protection principles. This appendix presents a brief overview of the areas of radiation physics, chemistry, and biology and is based to a large extent on the reviews of Mettler and Moseley (1985), Hobbs and McClellan (1986), Eichholz (1982), Hendee (1973), Cember (1996), and Early et al. (1979).

D.1 RADIONUCLIDES AND RADIOACTIVITY

The substances we call elements are composed of atoms. Atoms in turn are made up of neutrons, protons and electrons: neutrons and protons in the nucleus and electrons in a cloud of orbits around the nucleus. Nuclide is the general term referring to any nucleus along with its orbital electrons. The nuclide is characterized by the composition of its nucleus and hence by the number of protons and neutrons in the nucleus. All atoms of an element have the same number of protons (this is given by the atomic number) but may have different numbers of neutrons (this is reflected by the atomic mass numbers or atomic weight of the element). Atoms with different atomic mass but the same atomic numbers are referred to as isotopes of an element.

The numerical combination of protons and neutrons in most nuclides is such that the nucleus is quantum mechanically stable and the atom is said to be stable, i.e., not radioactive; however, if there are too few or too many neutrons, the nucleus is unstable and the atom is said to be radioactive. Unstable nuclides undergo radioactive transformation, a process in which a neutron or proton converts into the other and a beta particle is emitted, or else an alpha particle is emitted. Each type of decay is typically accompanied by the emission of gamma rays. These unstable atoms are called radionuclides; their emissions are called ionizing radiation; and the whole property is called radioactivity. Transformation or decay results in the formation of new nuclides some of which may themselves be radionuclides, while others are stable nuclides. This series of transformations is called the decay chain of the radionuclide. The first radionuclide in the chain is called the parent; the subsequent products of the transformation are called progeny, daughters, or decay products.

In general there are two classifications of radioactivity and radionuclides: natural and artificial (man-made). Naturally-occurring radioactive materials (NORMs) exist in nature and no additional energy is necessary to place them in an unstable state. Natural radioactivity is the property of some naturally occurring, usually heavy elements, that are heavier than lead. Radionuclides, such as radium and uranium, primarily emit alpha particles. Some lighter elements such as carbon-14 and tritium (hydrogen-3) primarily emit beta particles as they transform to a more stable atom. Natural radioactive atoms heavier than lead cannot attain a stable nucleus heavier than lead. Everyone is exposed to background radiation from naturally-occurring radionuclides throughout life. This background radiation is the major source of radiation exposure to man and arises from several sources. The natural background exposures are frequently used as a standard of comparison for exposures to various artificial sources of ionizing radiation.

Artificial radioactive atoms are produced either as a by-product of fission of uranium or plutonium atoms in a nuclear reactor or by bombarding stable atoms with particles, such as neutrons or protons, directed at the stable atoms with high velocity. These artificially produced radioactive elements usually decay by emission of particles, such as positive or negative beta particles and one or more high energy photons (gamma rays). Unstable (radioactive) atoms of any element can be produced.

Both naturally occurring and artificial radioisotopes find application in medicine, industrial products, and consumer products. Some specific radioisotopes, called fall-out, are still found in the environment as a result of nuclear weapons use or testing.

D.2 RADIOACTIVE DECAY

D.2.1 Principles of Radioactive Decay

The stability of an atom is the result of the balance of the forces of the various components of the nucleus. An atom that is unstable (radionuclide) will release energy (decay) in various ways and transform to stable atoms or to other radioactive species called daughters, often with the release of ionizing radiation. If there are either too many or too few neutrons for a given number of protons, the resulting nucleus may undergo transformation. For some elements, a chain of daughter decay products may be produced until stable atoms are formed. Radionuclides can be characterized by the type and energy of the radiation emitted, the rate of decay, and the mode of decay. The mode of decay indicates how a parent compound undergoes transformation. Radiations considered here are primarily of nuclear origin, i.e., they arise from nuclear excitation, usually caused by the capture of charged or uncharged nucleons by a nucleus, or by the radioactive decay or transformation of an unstable nuclide. The type of radiation may be categorized as charged or uncharged particles, protons, and fission products) or electromagnetic radiation (gamma rays and x rays). Table D-1 summarizes the basic characteristics of the more common types of radiation encountered.

D.2.2 Half-Life and Activity

For any given radionuclide, the rate of decay is a first-order process that is constant, regardless of the radioactive atoms present and is characteristic for each radionuclide. The process of decay is a series of random events; temperature, pressure, or chemical combinations do not effect the rate of decay. While it may not be possible to predict exactly which atom is going to undergo transformation at any given time, it is possible to predict, on average, the fraction of the radioactive atoms that will transform during any interval of time.

The *activity* is a measure of the quantity of radioactive material. For these radioactive materials it is customary to describe the activity as the number of disintegrations (transformations) per unit time. The unit of activity is the curie (Ci), which was originally related to the activity of one gram of radium, but is now defined as that quantity of radioactive material in which there are:

1 curie (Ci) = 3.7×10^{10} disintegrations (transformations)/second (dps) or 2.22×10^{12} disintegrations (transformations)/minute (dpm).

The SI unit of activity is the becquerel (Bq); 1 Bq = that quantity of radioactive material in which there is 1 transformation/second. Since activity is proportional to the number of atoms of the radioactive material, the quantity of any radioactive material is usually expressed in curies, regardless of its purity or concentration. The transformation of radioactive nuclei is a random process, and the number of transformations is directly proportional to the number of radioactive atoms present. For any pure radioactive substance, the rate of decay is usually described by its radiological half-life, T_R, i.e., the time it takes for a specified source material to decay to half its initial activity. The specific activity is the activity of a radionuclide per mass of that radionuclide. If properly qualified, it can refer to activity per unit mass of related materials, such as the element itself or a chemical compound labeled with the radionuclide. The higher the specific activity of a radioisotope, the faster it is decaying.

The activity of a radionuclide at time t may be calculated by:

$$A = A_o e^{-0.693t/T_{rad}}$$

where A is the activity in dps or curies or becquerels, A_o is the activity at time zero, t is the time at which measured, and T_{rad} is the radiological half-life of the radionuclide (T_{rad} and t must be in the same units of time). The time when the activity of a sample of radioactivity becomes one-half its original value is the radioactive half-life and is expressed in any suitable unit of time.

Table D-1. Characteristics of Nuclear Radiations

Radiation	Rest mass[a]	Charge	Typical energy range	Path length[b] Air	Path length[b] Solid	Comments
Alpha (α)	4.00 amu	+2	4–10 MeV	5–10 cm	25–80 µm	Identical to ionized He nucleus
Negatron (β^-)	5.48×10^{-4} amu; 0.51 MeV	–1	0–4 MeV	0–10 m	0–1 cm	Identical to electron
Positron (β^+)	5.48×10^{-4} amu; 0.51 MeV	+1	0-4 MeV	0–10 m	0–1 cm	Identical to electron except for sign of charge
Neutron	1.0086 amu; 939.55 MeV	0	0–15 MeV	b	b	Free half-life: 16 min
X ray (e.m. photon)	–	0	5 keV–100 keV	b	b	Photon from transition of an electron between atomic orbits
Gamma (γ) (e.m. photon)	–	0	10 keV–3 MeV	b	b	Photon from nuclear transformation

[a] The rest mass (in amu) has an energy equivalent in MeV that is obtained using the equation $E=mc^2$, where 1 amu = 932 MeV.
[b] Path lengths are not applicable to x- and gamma rays since their intensities decrease exponentially; path lengths in solid tissue are variable, depending on particle energy, electron density of material, and other factors.

amu = atomic mass unit; e.m. = electromagnetic; MeV = Megaelectron Volts

The specific activity is a measure of activity, and is defined as the activity of a radionuclide per mass of that radionuclide. This activity is usually expressed in curies per gram and may be calculated by

$$\text{curies/gram} = 1.3\text{x}10^8 / (T_{rad}) (\text{atomic weight}) \quad \text{or}$$

$$[3.577 \text{ x } 10^5 \text{ x mass(g)}] / [T_{rad} \text{ x atomic weight}]$$

where T_{rad} is the radiological half-life in days.

In the case of radioactive materials contained in living organisms, an additional consideration is made for the reduction in observed activity due to regular processes of elimination of the respective chemical or biochemical substance from the organism. This introduces a rate constant called the biological half-life (T_{biol}) which is the time required for biological processes to eliminate one-half of the activity. This time is virtually the same for both stable and radioactive isotopes of any given element.

Under such conditions the time required for a radioactive element to be halved as a result of the combined action of radioactive decay and biological elimination is the effective clearance half-time:

$$T_{eff} = (T_{biol} \times T_{rad}) / (T_{biol} + T_{rad}).$$

Table D-2 presents representative effective half-lives of particular interest.

Table D-2. Half-Lives of Some Radionuclides in Adult Body Organs

		Half-life[a]		
Radionuclide	Critical organ	Physical	Biological	Effective
Uranium 238	Kidney	4,460,000,000 y	4 d	4 d
Hydrogen 3[b] (Tritium)	Whole body	12.3 y	10 d	10 d
Iodine 131	Thyroid	8 d	80 d	7.3 d
Strontium 90	Bone	28 y	50 y	18 y
Plutonium 239	Bone surface	24,400 y	50 y	50 y
	Lung	24,400 y	500 d	474 d
Cobalt 60	Whole body	5.3 y	99.5 d	95 d
Iron 55	Spleen	2.7 y	600 d	388 d
Iron 59	Spleen	45.1 d	600 d	42 d
Manganese 54	Liver	303 d	25 d	23 d
Cesium 137	Whole body	30 y	70 d	70 d

[a] d = days, y = years
[b] Mixed in body water as tritiated water

D.2.3 Interaction of Radiation with Matter

Both ionizing and nonionizing radiation will interact with materials; that is, radiation will lose kinetic energy to any solid, liquid or gas through which it passes by a variety of mechanisms. The transfer of energy to a medium by either electromagnetic or particulate radiation may be sufficient to cause formation of ions. This process is called ionization. Compared to other types of radiation that may be absorbed, such as ultraviolet radiation, ionizing radiation deposits a relatively large amount of energy into a small volume.

The method by which incident radiation interacts with the medium to cause ionization may be direct or indirect. Electromagnetic radiations (x rays and gamma photons) are indirectly ionizing; that is, they give up their energy in various interactions with cellular molecules, and the energy is then utilized to produce a fast-moving charged particle such as an electron. It is the electron that then may react with a target molecule. This particle is called a “primary ionizing particle. Charged particles, in contrast, strike the tissue or medium and directly react with target molecules, such as oxygen or water. These particulate radiations are directly ionizing radiations. Examples of directly ionizing particles include alpha and beta particles. Indirectly ionizing radiations are always more penetrating than directly ionizing particulate radiations.

Mass, charge, and velocity of a particle, as well as the electron density of the material with which it interacts, all affect the rate at which ionization occurs. The higher the charge of the particle and the lower the velocity, the greater the propensity to cause ionization. Heavy, highly charged particles, such as alpha particles, lose energy rapidly with distance and, therefore, do not penetrate deeply. The result of these interaction processes is a gradual slowing down of any incident particle until it is brought to rest or "stopped" at the end of its range.

D.2.4 Characteristics of Emitted Radiation

D.2.4.1 Alpha Emission. In alpha emission, an alpha particle consisting of two protons and two neutrons is emitted with a resulting decrease in the atomic mass number by four and reduction of the atomic number of two, thereby changing the parent to a different element. The alpha particle is identical to a helium nucleus consisting of two neutrons and two protons. It results from the radioactive decay of some heavy elements such as uranium, plutonium, radium, thorium, and radon. The alpha particles emitted by a given radionuclide have the same energy and intensity combination. Most of the alpha particles that are likely to be found have energies in the range of about 4 to 8 MeV, depending on the isotope from which they came.

The alpha particle has an electrical charge of +2. Because of this double positive charge and their size, alpha particles have great ionizing power and, thus, lose their kinetic energy quickly. This results in very little penetrating power. In fact, an alpha particle cannot penetrate a sheet of paper. The range of an alpha particle (the distance the charged particle travels from the point of origin to its resting point) is about 4 cm in air, which decreases considerably to a few micrometers in tissue. These properties cause alpha emitters to be hazardous only if there is internal contamination (i.e., if the radionuclide is inside the body).

D.2.4.2 Beta Emission. A beta particle (β) is a high-velocity electron ejected from a disintegrating nucleus. The particle may be either a negatively charged electron, termed a negatron (β-) or a positively charged electron, termed a positron (β+). Although the precise definition of "beta emission" refers to both β- and β+, common usage of the term generally applies only to the negative particle, as distinguished from the positron emission, which refers to the β+ particle.

D.2.4.2.1 Beta Negative Emission. Beta particle (β-) emission is another process by which a radionuclide, with a neutron excess achieves stability. Beta particle emission decreases the number of neutrons by one and increases the number of protons by one, while the atomic mass number remains unchanged.[1] This transformation results in the formation of a different element. The energy spectrum of beta particle emission ranges from a certain maximum down to zero with the mean energy of the spectrum being about one-third of the maximum. The range of betas is much less in tissue than in air. Beta negative emitting radionuclides can cause injury to the skin and superficial body tissues, but mostly present an internal contamination hazard.

D.2.4.2.2 Positron Emission. In cases in which there are too many protons in the nucleus, positron emission may occur. In this case a proton may be thought of as being converted into a neutron, and a positron (β+) is emitted.1 This increases the number of neutrons by one, decreases the number of protons by one, and again leaves the atomic mass number unchanged. The gamma radiation resulting from the annihilation (see glossary) of the positron makes all positron emitting isotopes more of an external radiation hazard than pure β emitters of equal energy.

D.2.4.2.3 Gamma Emission. Radioactive decay by alpha, beta, or positron emission, or electron capture often leaves some of the energy resulting from these changes in the nucleus. As a result, the nucleus is raised to an excited level. None of these excited nuclei can remain in this high-energy state. Nuclei release this energy returning to ground state or to the lowest possible stable energy level. The energy released is in the form of gamma radiation (high energy photons) and has an energy equal to the change in the energy state of the nucleus. Gamma and x rays behave similarly but differ in their origin;

---

[1] Neutrinos also accompany negative beta particles and positron emissions

gamma emissions originate in the nucleus while x rays originate in the orbital electron structure or from rapidly changing the velocity of an electron (e.g., as occurs when shielding high energy beta particles or stopping the electron beam in an x ray tube).

D.3 ESTIMATION OF ENERGY DEPOSITION IN HUMAN TISSUES

Two forms of potential radiation exposures can result: internal and external. The term exposure denotes physical interaction of the radiation emitted from the radioactive material with cells and tissues of the human body. An exposure can be "acute" or "chronic" depending on how long an individual or organ is exposed to the radiation. Internal exposures occur when radionuclides, which have entered the body (e.g., through the inhalation, ingestion, or dermal pathways), undergo radioactive decay resulting in the deposition of energy to internal organs. External exposures occur when radiation enters the body directly from sources located outside the body, such as radiation emitters from radionuclides on ground surfaces, dissolved in water, or dispersed in the air. In general, external exposures are from material emitting gamma radiation, which readily penetrate the skin and internal organs. Beta and alpha radiation from external sources are far less penetrating and deposit their energy primarily on the skin's outer layer. Consequently, their contribution to the absorbed dose of the total body dose, compared to that deposited by gamma rays, may be negligible.

Characterizing the radiation dose to persons as a result of exposure to radiation is a complex issue. It is difficult to: (1) measure internally the amount of energy actually transferred to an organic material and to correlate any observed effects with this energy deposition; and (2) account for and predict secondary processes, such as collision effects or biologically triggered effects, that are an indirect consequence of the primary interaction event.

D.3.1 Dose/Exposure Units

D.3.1.1 Roentgen. The roentgen (R) is a unit of x or gamma-ray exposure and is a measured by the amount of ionization caused in air by gamma or x radiation. One roentgen produces 2.58×10^{-4} coulomb per kilogram of air. In the case of gamma radiation, over the commonly encountered range of photon energy, the energy deposition in tissue for a dose of 1 R is about 0.0096 joules (J) /kg of tissue.

D.3.1.2 Absorbed Dose and Absorbed Dose Rate. The absorbed dose is defined as the energy imparted by radiation to a unit mass of the tissue or organ. The unit of absorbed dose is the rad; 1 rad = 100 erg/gram = 0.01 J/kg in any medium. An exposure of 1 R results in a dose to soft tissue of approximately 0.01 J/kg. The SI unit is the gray which is equivalent to 100 rad or 1 J/kg. Internal and external exposures from radiation sources are not usually instantaneous but are distributed over extended periods of time. The resulting rate of change of the absorbed dose to a small volume of mass is referred to as the absorbed dose rate in units of rad/unit time.

D.3.1.3 Working Levels and Working Level Months. Working level (WL) is a measure of the atmospheric concentration of radon and its short-lived progeny. One WL is defined as any combination of short-lived radon daughters (through polonium-214), per liter of air, that will result in the emission of 1.3×10^5 MeV of alpha energy. An activity concentration of 100 pCi radon-222/L of air, in equilibrium with its daughters, corresponds approximately to a potential alpha-energy concentration of 1 WL. The WL unit can also be used for thoron daughters. In this case, 1.3×10^5 MeV of alpha energy (1 WL) is released by the thoron daughters in equilibrium with 7.5 pCi thoron/L. The potential alpha energy exposure of miners is commonly expressed in the unit Working Level Month (WLM). One WLM corresponds to exposure to a concentration of 1 WL for the reference period of 170 hours, or more generally

WLM = concentration (WL) x exposure time (months) (one “month” = 170 working hours).

D.3.2 Dosimetry Models

Dosimetry models are used to estimate the dose from internally deposited to radioactive substances. The models for internal dosimetry consider the amount of radionuclides entering the body, the factors affecting their movement or transport through the body, distribution and retention of radionuclides in the body, and the energy deposited in organs and tissues from the radiation that is emitted during spontaneous decay processes. The dose pattern for radioactive materials in the body may be strongly influenced by the route of entry of the material. For industrial workers, inhalation of radioactive particles with pulmonary deposition and puncture wounds with subcutaneous deposition have been the most frequent. The general population has been exposed via ingestion and inhalation of low levels of naturally occurring radionuclides as well as radionuclides from nuclear weapons testing.

The models for external dosimetry consider only the photon doses (and neutron doses, where applicable) to organs of individuals who are immersed in air or are exposed to a contaminated object.

D.3.2.1 Ingestion. Ingestion of radioactive materials is most likely to occur from contaminated foodstuffs or water or eventual ingestion of inhaled compounds initially deposited in the lung. Ingestion of radioactive material may result in toxic effects as a result of either absorption of the radionuclide or irradiation of the gastrointestinal tract during passage through the tract, or a combination of both. The fraction of a radioactive material absorbed from the gastrointestinal tract is variable, depending on the specific element, the physical and chemical form of the material ingested, and the diet, as well as some other metabolic and physiological factors. The absorption of some elements is influenced by age, usually with higher absorption in the very young.

D.3.2.2 Inhalation. The inhalation route of exposure has long been recognized as being a major portal of entry for both nonradioactive and radioactive materials. The deposition of particles within the lung is largely dependent upon the size of the particles being inhaled. After the particle is deposited, the retention will depend upon the physical and chemical properties of the dust and the physiological status of the lung. The retention of the particle in the lung depends on the location of deposition, in addition to the physical and chemical properties of the particles. The converse of pulmonary retention is pulmonary clearance. There are three distinct mechanisms of clearance which operate simultaneously. Ciliary clearance acts only in the upper respiratory tract. The second and third mechanisms act mainly in the deep respiratory tract. These are phagocytosis and absorption. Phagocytosis is the engulfing of foreign bodies by alveolar macrophages and their subsequent removal either up the ciliary "escalator" or by entrance into the lymphatic system. Some inhaled soluble particles are absorbed into the blood and translocated to other organs and tissues.

D.3.3 Internal Emitters

An internal emitter is a radionuclide that is inside the body. The absorbed dose from internally deposited radionuclide depends on the energy absorbed per unit mass by the irradiated tissue. For a radionuclide distributed uniformly throughout an infinitely large medium, the concentration of absorbed energy must be equal to the concentration of energy emitted by the radionuclide. An infinitely large medium may be approximated by a tissue mass whose dimensions exceed the range of the particle. All alpha and most beta radiation will be absorbed in the organ (or tissue) of reference. Gamma-emitting radionuclide emissions are penetrating radiation, and a substantial fraction of gamma energy may be absorbed in tissue. The dose to an organ or tissue is a function of the effective retention half-time, the energy released in the tissue, the amount of radioactivity initially introduced, and the mass of the organ or tissue.

D.4 BIOLOGICAL EFFECTS OF RADIATION

When biological material is exposed to ionizing radiation, a chain of cellular events occurs as the ionizing particle passes through the biological material. A number of theories have been proposed to describe the interaction of radiation with biologically important molecules in cells and to explain the resulting damage to biological systems from those interactions. Many factors may modify the response of a living organism to a given dose of radiation. Factors related to the exposure include the dose rate, the energy of the radiation, and the temporal pattern of the exposure. Biological considerations include factors such as species, age, sex, and the portion of the body exposed. Several excellent reviews of the biological effects of radiation have been published, and the reader is referred to these for a more in-depth discussion (Brodsky 1996; Hobbs and McClellan 1986; ICRP 1984; Mettler and Moseley 1985; Rubin and Casarett 1968).

D.4.1 Radiation Effects at the Cellular Level

According to Mettler and Moseley (1985), at acute doses up to 10 rad (100 mGy), single strand breaks in DNA may be produced. These single strand breaks may be repaired rapidly. With doses in the range of 50–500 rad (0.5–5 Gy), irreparable double-stranded DNA breaks are likely, resulting in cellular reproductive death after one or more divisions of the irradiated parent cell. At large doses of radiation, usually greater than 500 rad (5 Gy), direct cell death before division (interphase death) may occur from the direct interaction of free-radicals with essential cellular macromolecules. Morphological changes at the cellular level, the severity of which are dose-dependent, may also be observed.

The sensitivity of various cell types varies. According to the Bergonie-Tribondeau law, the sensitivity of cell lines is directly proportional to their mitotic rate and inversely proportional to the degree of differentiation (Mettler and Moseley 1985). Rubin and Casarett (1968) devised a classification system that categorized cells according to type, function, and mitotic activity. The categories range from the most sensitive type, "vegetative intermitotic cells", found in the stem cells of the bone marrow and the gastrointestinal tract, to the least sensitive cell type, "fixed postmitotic cells," found in striated muscles or long-lived neural tissues.

Cellular changes may result in cell death, which if extensive, may produce irreversible damage to an organ or tissue or may result in the death of the individual. If the cell recovers, altered metabolism and function may still occur, which may be repaired or may result in the manifestation of clinical symptoms. These changes may also be expressed at a later time as tumors or cellular mutations, which may result in abnormal tissue.

D.4.2 Radiation Effects at the Organ Level

In most organs and tissues the injury and the underlying mechanism for that injury are complex and may involve a combination of events. The extent and severity of this tissue injury are dependent upon the radiosensitivity of the various cell types in that organ system. Rubin and Casarett (1968) describe and schematically display the events following radiation in several organ system types. These include: a rapid renewal system, such as the gastrointestinal mucosa; a slow renewal system, such as the pulmonary epithelium; and a nonrenewal system, such as neural or muscle tissue. In the rapid renewal system, organ injury results from the direct destruction of highly radiosensitive cells, such as the stem cells in the bone marrow. Injury may also result from constriction of the microcirculation and from edema and inflammation of the basement membrane, designated as the histohematic barrier, which may progress to fibrosis. In slow renewal and nonrenewal systems, the radiation may have little effect on the parenchymal cells, but ultimate parenchymal atrophy and death over several months result from fibrosis and occlusion of the microcirculation.

D.4.3 Low Level Radiation Effects

Cancer is the major latent harmful effect produced by ionizing radiation and the one that most people exposed to radiation are concerned about. The ability of alpha, beta, and gamma radiation to produce cancer in virtually every tissue and organ in laboratory animals has been well-demonstrated. The development of cancer is not an immediate effect. Radiation-induced leukemia has the shortest latent period at about 2 years, while other radiation induced cancers, such as osteosarcoma, have latent periods greater than 20 years. The mechanism by which cancer is induced in living cells is complex and is a topic of intense study. Exposure to ionizing radiation can produce cancer at any site within the body; however, some sites appear to be more common than others, such as the breast, lung, stomach, and thyroid.

DNA is the major target molecule during exposure to ionizing radiation. Other macromolecules, such as lipids and proteins, are also at risk of damage when exposed to ionizing radiation. The genotoxicity of ionizing radiation is an area of intense study, as damage to the DNA is ultimately responsible for many of the adverse toxicological effects ascribed to ionizing radiation, including cancer. Damage to genetic material is basic to developmental or teratogenic effects, as well. However, for effects other than cancer, there is little evidence of human effects at low levels of exposure.

D.5 UNITS IN RADIATION PROTECTION AND REGULATION

D.5.1 Dose Equivalent (or Equivalent Dose)

Dose equivalent (as measured in rem or sievert) is a special radiation protection quantity that is used for administrative and radiation safety purposes to express the absorbed dose in a manner which considers the difference in biological effectiveness of various kinds of ionizing radiation. ICRP (1990) changed this term to equivalent dose, but it has not yet been adopted by the USNRC or DOE.

The USNRC defines the dose equivalent, H, as the product of the absorbed dose, D, and the quality factor, Q, at the point of interest in biological tissue. This relationship is expressed as $H = D \times Q$. The dose equivalent concept is applicable only to doses that are not great enough to produce biomedical effects.

The quality factor or radiation weighting factor is a dimensionless quantity that depends in part on the stopping power for charged particles, and it accounts for the differences in biological effectiveness found among the types of radiation. Originally relative biological effectiveness (RBE) was used rather than Q to define the quantity, rem, which was of use in risk assessment. The generally accepted values for quality factors and radiation weighting factors for various radiation types are provided in Table D-3. The dose equivalent rate is the time rate of change of the dose equivalent to organs and tissues and is expressed as rem/unit time or sievert/unit time.

Table D-3. Quality Factors (Q) and Absorbed Dose Equivalencies

Type of radiation	Quality factor (Q)	Radiation weighting factor (w_r)*
X, gamma, or beta radiation	1	1
Alpha particles, multiple-charged particles, fission fragments and heavy particles of unknown charge	20	0.05
Neutrons (other than thermal >> 100 keV to 2 MeV), protons, alpha particles, charged particles of unknown energy	10	20
Neutrons of unknown energy	10	
High-energy protons	10	0.1
Thermal neutrons		5

*Absorbed dose in rad equal to 1 rem or the absorbed dose in gray equal to 1 sievert.

Source: USNRC. 2004. Standards for the protection against radiation, table 1004(b).1. 10 CFR 20.1004. U.S. Nuclear Regulatory Commission, Washington, D.C. NCRP 1993

D.5.2 Relative Biological Effectiveness

RBE is used to denote the experimentally determined ratio of the absorbed dose from one radiation type to the absorbed dose of a reference radiation required to produce an identical biologic effect under the same conditions. Gamma rays from cobalt-60 and 200–250 kVp x-rays have been used as reference standards. The term RBE has been widely used in experimental radiobiology, and the term quality factor (or radiation weighting factor) used in calculations of dose equivalents for radiation safety purposes (ICRP 1977; NCRP 1971; UNSCEAR 1982). Any RBE value applies only to a specific biological end point, in a specific exposure, under specific conditions to a specific species. There are no generally applicable values of RBE since RBEs are specific to a given exposure scenario.

D.5.3 Effective Dose Equivalent (or Effective Dose)

The absorbed dose is usually defined as the mean energy imparted per unit mass to an organ or tissue. This represents a simplification of the actual problem. Normally when an individual ingests or inhales a radionuclide or is exposed to external radiation that enters the body (gamma), the dose is not uniform throughout the whole body. The simplifying assumption is that the detriment will be the same whether the body is uniformly or non-uniformly irradiated. In an attempt to compare detriment from absorbed dose of a limited portion of the body with the detriment from total body dose, the ICRP (1977) has derived a concept of effective dose equivalent. ICRP (1990) changed this term to effective dose, but it has not yet been adopted by the USNRC or DOE.

The effective dose equivalent, H_E, is

$$H_E = \text{(the sum of) } W_t \, H_t$$

where H_t is the dose equivalent (or equivalent dose) in the tissue t, W_t is the tissue weighting factor in that tissue, which represents the estimated proportion of the stochastic risk resulting from tissue, t, to the stochastic risk when the whole body is uniformly irradiated for occupational exposures under certain conditions (ICRP 1977). Tissue weighting factors for selected tissues are listed in Table D-4.

D.5.4 SI Units

The ICRU (1980), ICRP (1984), and NCRP (1985) now recommend that the rad, roentgen, curie, and rem be replaced by the SI units: gray (Gy), Coulomb per kilogram (C/kg), Becquerel (Bq), and sievert (Sv), respectively. The relationship between the customary units and the international system of units (SI) for radiological quantities is shown in Table D-5.

Table D-4. Tissue Weighting Factors for Calculating Effective Dose Equivalent and Effective Dose for Selected Tissues

	Tissue weighting factor	
Tissue	NCRP115/ ICRP60	USNRC/ICRP26
Bladder	0.05	–
Bone marrow	0.12	0.12
Bone surface	0.01	0.03
Breast	0.05	0.15
Colon	0.12	–
Esophagus	0.05	–
Gonads	0.20	0.25
Liver	0.05	–
Lung	0.12	0.12
Skin	0.01	–
Stomach	0.12	–
Thyroid	0.05	0.03
Remainder	0.05	0.30
Total	1.00	1.00

ICRP60 = International Commission on Radiological Protection, 1990 Recommendations of the ICRP
NCRP115 = National Council on Radiation Protection and Measurements. 1993. Risk Estimates for Radiation Protection, Report 115. Bethesda, Maryland
USNRC = Nuclear Regulatory Commission, Title 10, Code of Federal Regulations, Part 20

Table D-5. Comparison of Common and SI Units for Radiation Quantities

Quantity	Customary units	Definition	SI units	Definition
Activity (A)	curie (Ci)	3.7x10^{10} transformations s^{-1}	becquerel (Bq)	s^{-1}
Absorbed dose (D)	rad	10^{-2} Jkg^{-1}	gray (Gy)	Jkg^{-1}
Absorbed dose rate (Ď)	rad per second (rad s^{-1})	10^{-2} $Jkg^{-1}s^{-1}$	gray per second (Gy s^{-1})	Jkg^{-1} s^{-1}
Dose equivalent (H)	rem	10^{-2} Jkg^{-1}	sievert (Sv)	Jkg^{-1}
Dose equivalent rate (Ĥ)	rem per second (rem s^{-1})	10^{-2} $Jkg^{-1}s^{-1}$	sievert per second (Sv s^{-1})	Jkg^{-1} s^{-1}
Effective dose	rem	10^{-2} Jkg^{-1}	Sievert (Sv)	Jkg^{-1}
Equivalent dose (H)	rem	10^{-2} Jkg^{-1}	Sievert (Sv)	Jkg^{-1}
Linear energy transfer (LET)	kiloelectron volts per micrometer (keV μm^{-1})	1.602x10^{-10} Jm^{-1}	kiloelectron volts per micrometer (keV μm^{-1})	1.602x10^{-10} Jm^{-1}

Jkg^{-1} = Joules per kilogram; $Jkg^{-1}s^{-1}$ = Joules per kilogram per second; Jm^{-1} = Joules per meter; s^{-1} = per second

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APPENDIX E. INDEX

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