Selenium and Compounds; CASRN 7782-49-2

Human health assessment information on a chemical substance is included in the IRIS database only after a comprehensive review of toxicity data, as outlined in the IRIS assessment development process. Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the conclusions that were reached during the assessment development process. Supporting information and explanations of the methods used to derive the values given in IRIS are provided in the guidance documents located on the IRIS website.

STATUS OF DATA FOR Selenium and Compounds

File First On-Line 03/01/1991

Category (section)	Assessment Available?	Last Revised
Oral RfD (I.A.)	yes	06/01/1991
Inhalation RfC (I.B.)	not evaluated	
Carcinogenicity Assessment (II.)	yes	03/01/1991

I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — Selenium and Compounds
CASRN — 7782-49-2
Last Revised — 06/01/1991

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of

information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. Oral RfD Summary

Critical Effect	Experimental Doses*	UF	MF	RfD
Clinical selenosis **Human Epidemiological Study** **Yang et al., 1989b**	NOAEL: 0.015 mg/kg/day LOAEL: 0.023 mg/kg/day	3	1	5E-3 mg/kg/day

* Conversion Factors: NOAEL (0.853 mg/day) and LOAEL (1.261 mg/day) calculated from regression analysis (log Y = 0.767log X - 2.248, where Y = blood selenium and X = selenium intake) as detailed in Yang et al. (1989a) based upon the correlation (r = 0.962) between dietary selenium intake and blood selenium level for data showing incidence of clinical selenosis in adults based on an average adult body weight of 55 kg (Yang et al., 1989b).

I.A.2. Principal and Supporting Studies (Oral RfD)

Yang, G., S. Yin, R. Zhou, et al. 1989b. Studies of safe maximal daily dietary Se-intake in a seleniferous area in China. II. Relation between Se- intake and the manifestation of clinical signs and certain biochemical alterations in blood and urine. J. Trace Elem. Electrolytes Health Dis. 3(2): 123-130.

Yang et al. (1989b), in a follow-up to an earlier study (Yang et al., 1983), studied a population of approximately 400 individuals living in an area of China with unusually high environmental concentrations of selenium (Se). The subjects were evaluated for clinical and biochemical signs of Se intoxication. Three geographical areas with low, medium and high selenium levels in the soil and food supply were chosen for comparison in the studies. The earlier Yang et al. (1983) study was conducted in response to endemic selenium intoxication in two separate areas with sample sizes of only 6 and 3. Comparisons were then made to a selenium-adequate area (n=8) and low- selenium area (n=13). The Yang et al. (1989a,b) studies provide a much larger sample size and include additional analysis of tissue selenium levels. This allows a more accurate estimation of the dose-response relationship observed for selenium toxicity. Selenium levels in

soil and approximately 30 typical food types commonly eaten by the exposed population showed a positive correlation with blood and tissue Se levels. The daily average Se intakes, based on lifetime exposure, 70, 195 and 1438 ug for adult males and 62, 198 and 1238 ug for adult females in the low-, medium- and high-selenium areas, respectively. Significant correlations demonstrated between Se concentrations of various tissues were used to estimate the minimal daily Se intake values that elicited various alterations in biochemical parameters indicative of possible Se- induced liver dysfunction (i.e., prolongation of clotting time and serum glutathione titer) and clinical signs of selenosis (i.e., hair or nail loss, morphological changes of the nails, etc.). In this manner, a marginal safe level of daily Se intake was estimated.

Analysis of the results indicated that persistent clinical signs of selenosis were observed only in 5/349 adults, a potentially sensitive subpopulation. The blood selenium concentration in this group ranged from 1.054 to 1.854 mg/L with a mean of 1.346 mg/L. Clinical signs observed included the characteristic "garlic odor" of excess selenium excretion in the breath and urine, thickened and brittle nails, hair and nail loss, lowered hemoglobin levels, mottled teeth, skin lesions and CNS abnormalities (peripheral anesthesia, acroparesthesia and pain in the extremities). Alterations in the measured biochemical parameters occurred at dietary intake levels of 750-850 ug/day. These alterations were described as a delay in prothrombin time, i.e., increase in blood coagulation time and reduction in blood glutathione concentration. However, these indicators were poorly characterized and are not typically used as an index for clinical selenosis resulting from chronic exposure to selenium (NAS, 1989). Based upon the blood selenium levels shown to reflect clinical signs of selenium intoxication, a whole blood selenium concentration of 1.35 mg/L corresponding to 1.261 mg of daily selenium intake is indicative of the lowest correlative selenium intake causing overt signs of selenosis. The next lowest whole blood selenium concentration of 1.0 mg/L, corresponding to 0.853 mg selenium/day, produces no clinical signs of selenosis. The NOAEL for this study is 0.85 mg Se/day and the LOAEL is 1.26 mg Se/day.

A group of 142 volunteers in South Dakota and Wyoming were recruited by Longnecker et al. (1991) at random from households listed in a telephone directory or from ranches with suspected high selenium intake based on previous cases of livestock selenosis. The geographical areas were chosen because of known seleniferous topsoil and high concentrations of selenium in plants and food. The subjects were followed for 1 year and completed health questionnaires, underwent physical examinations, provided blood samples for clinical assessment, and provided blood, urine, toenails and duplicate-plate food collections for selenium analysis. The average selenium intake was 239 ug/day, approximately 2-3 times higher than the national average. The concentration of selenium in whole blood, serum, urine and toenails and the amount in diet were highly correlated. Blood selenium concentration was highly correlated with selenium intake. The correlation was very similar to that reported by Yang et al. (1989a). Liver function (prothrombin time and alanine aminotransferase, aspartate aminotransferase, gamma glutamyltransferase and

alkaline phosphatase), hematologic function (leukocyte count, hemoglobin and hematocrit) and clinical chemistry (sodium, potassium and chloride concentration) were not found to be altered as a result of selenium intake. High regression coefficient predictor variables for selenium toxicity (muscle twitching, paresthesia, nail loss, nail lines, hair loss and garlic breath) were not found in increased frequency for this population. No signs of selenium toxicity were found in this population, including individuals whose selenium intake was as high as 724 ug/day. This report corroborates that of Yang et al. (1989b), which showed that a selenium intake of up to 853 ug/day is not associated with characteristic nail or hair loss typical of selenium intoxication.

I.A.3. Uncertainty and Modifying Factors (Oral RfD)

UF — An uncertainty factor of 3 was applied to the NOAEL to account for sensitive individuals. A full factor of 10 was not deemed necessary since similar NOAELs were identified in two moderately-sized human populations exposed to selenium levels in excess of the RDA throughout a lifetime without apparent clinical signs of selenosis.

MF — None

I.A.4. Additional Studies/Comments (Oral RfD)

The essentiality for selenium has been well-documented in livestock based upon the alleviation of specific deficiency conditions by selenium supplementation of the diet (Combs and Combs, 1986). Selenium has been clearly demonstrated to be a cofactor of glutathione peroxidase, a hydrogen and lipid peroxide reducing enzyme and is therefore essential (Rotruck et al., 1973). Human requirements for selenium were not conclusively established until 1979 when an association was made between low selenium status and cardiomyopathy (Keshan disease) in China for young children and women of child-bearing age (Keshan Disease Research Group, 1979a,b). More recently, iatrogenic episodes of selenium deficiency have been reported in patients receiving intravenous total parenteral administration of feeding solutions devoid of selenium. Symptoms included low glutathione peroxidase activity and low selenium levels in erythrocytes (Levander and Burk, 1986), muscular weakness and discomfort (van Rij et al., 1979) and cardiomyopathy (Johnson et al., 1981). It is important to note that glutathione peroxidase activity is a valid indicator of human selenium status only in populations with relatively low selenium intakes, since the enzyme activity plateaus at adequate selenium intake levels (Whanger et al., 1988), thereby precluding the use of this biochemical indicator under excessive selenium intake situations.

The NAS (1989) has determined the recommended dietary allowance for selenium to be 0.87 ug/kg, or approximately 70 and 55 ug/day for the reference adult North American male and female, respectively. Requirements for selenium increase during pregnancy to 65 ug/day and for

lactation to 75 ug/day. Selenium requirements for infants and children vary according to age. However, based on the reference weights of NHANES II, these populations demonstrate an increased requirement per unit weight relative to adults. For infants, the selenium requirement is 1.67 ug/kg and for children the requirement ranges from 1.07-1.53 ug/kg. It should be noted that the most recent RDA for selenium did not consider the 1989 results of Yang et al. (1989a,b) discussed above, but an earlier preliminary report by the same authors (Yang et al., 1983).

Yang et al. (1983) reported clinical signs of selenosis (i.e., loss of hair and nails) in approximately 50% of a population of 248 inhabitants living in Enshi County, Hubei Province of the People's Republic of China. Selenosis was reported in the highest selenium contaminated area where the average daily Se intake was 5.0 mg/day (range 3.2-6.7), but no selenosis occurred when the average intake was 0.750 mg/day (range 0.240-1.51). These estimates, however, were based upon estimates of intake from only 6 and 3 inhabitants in the high and low contaminated areas, respectively. Yang et al. (1989b) reported prolonged clotting time and serum glutathione and these biochemical changes were indicated as adverse effects of selenium exposure. Glutathione is a strong nucleophile that reacts well with soft electrophiles and is an important conjugate-forming compound for the detoxification and excretion of electrophilic metabolites and metabolically produced oxidizing agents. If glutathione is depleted or markedly reduced in the liver, the hepatotoxicity of these compounds would likewise be expected to be enhanced (Ketterer et al., 1983). However, the significance of decreased serum glutathione is not well characterized and should not be used in this context as a biochemical marker of selenium toxicity. Likewise, there is no indication that prothrombin activity is affected by excess selenium administration (Longnecker et al., 1991). Furthermore, the description of this effect in Yang et al. (1989b) was based on a population for which there is insufficient documentation of normal clotting times in the general Chinese population.

Selenium toxicity has been clinically described according to three types: acute selenosis, subacute selenosis and chronic selenosis. The acute condition is caused by consuming relatively high amounts of selenium over a short period of time. After the onset of this condition, walking becomes unsteady, cyanosis of the mucous membranes occurs and labored breathing is usually seen sometimes resulting in death. Pathological findings include congestion of the liver, endocarditis and myocarditis, degeneration of the sooth musculature of the gastrointestinal tract, gallbladder and bladder, and erosion of the long bones (Francke and Moxon, 1936).

Subacute selenosis occurs from exposure to large doses of Se over a longer period of time resulting in neurological dysfunction (impaired vision, ataxia, disorientation) and respiratory distress. It is typically seen most frequently in grazing livestock feeding upon Se-accumulating plants and has been referred to as "blind staggers" (Rosenfeld and Beath, 1964).

Prolonged exposure to more moderate levels of selenium result in skin lesions involving alopecia, hoof necrosis and loss, emaciation and increased serum transaminases and alkaline phosphatase in animals. In man, the condition is characterized by chronic dermatitis, fatigue, anorexia, gastroenteritis, hepatic degeneration, enlarged spleen and increased concentrations of Se in the hair and nails (Harr and Muth, 1972).

Selenium exists naturally in a number of oxidation states, thereby accounting for the different forms of selenium important to living organisms by oral ingestion. In the -2 oxidation state, selenium can be found as hydrogen selenide (H2Se), sodium selenide (Na2Se), di-[(CH3)2Se] and trimethyl selenium [(CH3)3Se] and various selenoamino acids such as selenomethionine, selenocysteine, Se-methyl selenocysteine, selenocystathionine and selenotaurine. Elemental selenium and the dipeptide selenodiglutathione have an oxidation state of 0. In the +4 oxidation state, selenium can exist as selenium dioxide (SeO2), selenious acid (H2SeO3) or as sodium selenite (Na2SeO3). Finally, in its most oxidized state (+6), selenium can be found as selenic acid (H2SeO4) or as sodium selenate (Na2SeO4).

The toxicity of selenium has been consistently well documented. However, some early studies reported that selenium may be a carcinogen. Nelson et al. (1943) showed that rats fed diets containing Se as seleniferous wheat developed hepatic tumors and low-grade carcinomas in 11/53 animals. This work has subsequently been criticized due to low-protein content and relatively high levels of Se in the diet (5, 7 or 10 ppm Se), a poorly characterized source of selenium, and in general poor experimental design. The authors reported no encapsulation or metastases and in fact noted their own difficulty in determining the difference between hyperplasia and tumor. Another early investigation by Seifter et al. (1946) reported several thyroid tumors and adenomatous hyperplasia in livers of rats fed 0.05% bis-4-acetylaminophenyl selenium dihydroxide for 105 days. This organic selenium compound was suspected of having goitrogenic properties but its carcinogenic effect has not been further confirmed to be attributable to the selenium in the molecule.

The first animal experiment which demonstrated anticarcinogenic effects of selenium was performed by Clayton and Baumann (1949). An approximate 50% reduction in dimethylaminoazobenzene-induced tumor incidence occurred in rats fed a diet supplemented with 5 ppm Se as selenite. Additional evidence subsequently reported, further illustrated the inhibitory effect of selenium on transplantable tumors in rats (Weisberger and Suhrland, 1956a) and leukemia in humans (Weisberger and Suhrland, 1956b). The National Cancer Institute sponsored an extensive study on selenium toxicity in rats in order to resolve the issue of selenium carcinogenicity. Diets containing up to 8 ppm selenium did not increase tumor incidence (Tinsley et al., 1967; Harr et al., 1967). Since 1970, there has been an increased interest in characterizing the anti- carcinogenic and anti-tumorigenic properties of selenium. The number of reports characterizing these properties are too numerous to discuss in detail here. The

reader is referred to a review by Milner and Fico (1987) for a more comprehensive treatment of the database.

The essentiality and toxicity of selenium varies according to the valence state of selenium when incorporated into biomolecules and the form in which selenium is fed or administered. This is especially true when comparing the LD50 value as an index of toxicity for the various selenium compounds. Although it is difficult to make an assessment for several selenium compounds by a similar mode of administration in a common species, there is general agreement that sodium selenite, sodium selenate, selenomethionine and selenoglutathione are among the more toxic species (Combs and Combs, 1986). The relative potency of systemic toxicity for selenium compounds is also similar in experiments examining potency of anti-tumorigenic activity. In vitro examination of potency of effect of selenium compounds on incubated Ehrlich ascites tumor cells (EATC) showed that sodium selenite is more efficacious in significantly reducing EATC viability than an equivalent concentration of sodium selenate. Although selenium dioxide, selenomethionine and selenocystine ultimately decreased viability of the EATC, nearly 50% more incubation time was required for the same effect (Poirier and Milner, 1979). The same authors investigated the relative potency of various selenium compounds administered intraperitoneally on EATC growth in vivo. Sodium selenite and selenodiglutathione (an intermediate of selenium metabolism) were the most effective forms of selenium in preventing EATC propagation. Sodium selenide, dimethyl selenide and selenocystine were not effective in inhibiting EATC growth (Poirier and Milner, 1983). Similar relative potency results have been reported in in vitro systems for canine mammary cells (Fico et al., 1986) and human mammary cells (Watrach et al., 1984).

Since selenium has been reported to cause growth retardation, decreased fertility, embryotoxicity, fetotoxicity and teratogenic effects in animals, Yang et al. (1989b) made the following observations: Malformation in chickens hatched from locally produced eggs did occur; however, teratogenic effects in human infants were never seen in this area although Se has been reported to be transmitted through the placenta to the fetus in animals. These findings confirm those reported by Yang et al. (1983) in which chicken eggs from this same area were reported to have very low hatchability and some deformed embryos in those that did hatch.

The developmental toxicity of selenomethionine was investigated by Tarantal et al. (1991) in non-human primates. Forty pregnant long-tailed macaques were dosed daily by nasogastric intubation with 0, 0.025, 0.150 or 0.3 mg selenium/kg as selenomethionine through gestational days 20-50. Dams were examined clinically and the pregnancies of two to three dams within each test group were followed to term (gestational day 165). All other dams were hysterectomized on gestational day 100. Neonates delivered at term were examined for morphometric, neurologic, behavioral and ophthalmologic effects on days 1, 8, 15, 22 and 30. Pregnancy loss among treated animals was not significantly different from concurrent or

historical controls. No statistically significant treatment-related effects were observed at necropsy on gestational day 100. There were no significant maternal or fetal developmental effects or teratogenesis found up to 0.3 mg/kg selenium, the highest dose tested.

Halverson et al. (1966) fed 60-70 g male, post-weanling Sprague-Dawley rats selenium as selenite or seleniferous wheat ad libitum at 1.6, 3.2, 4.8, 6.4, 8.0, 9.6 or 11.2 ppm of selenium (13, 27, 40, 67, 81 or 94 ug/kg/day, respectively). Levels of selenium up to 4.8 ppm showed no effect. At 8.0 ppm selenium as seleniferous wheat, there was an observed decrease in liver weight, increase in spleen weight, and decrease in hemoglobin. Mortality was observed in the groups fed 8.0, 9.6 and 11.2 ppm selenium as seleniferous wheat at incidences of 1/8, 5/8 and 8/8, respectively. The incidences of mortality reported for groups fed 8.0 and 9.6 ppm selenium as selenite were 1/8 and 1/10, respectively. A significant growth reduction was reported for both selenium sources at 6.4 ppm and higher, although feed utilization was not decreased. No other effects were reported for the rats fed sodium selenite.

Schroeder and Mitchener (1971) administered 3 ppm selenium as selenate (390 ug/kg/day) to CD mice through four generations. Maternal effects were not observed. There was a significant increase in young deaths in the F1 generation and an increase in numbers of runts in generations F1 through F3. By the F3 generation there was also a decrease in breeding events.

Rosenfeld and Beath (1954) administered selenium as potassium selenate to sires and pregnant rats through five breeding cycles at 1.5, 2.5 or 7.5 ppm selenium (75, 125 or 375 ug/kg/day). No effect was observed on reproduction, the number of young reared or on the reproduction of two successive generations of dams and sires in groups receiving 1.5 ppm selenium. In the group receiving 2.5 ppm selenium, there was a 50% reduction in the number of young reared. At 7.5 ppm there was a decrease in fertility of the females but not males, a decrease in the number of survivors and a reduction in the rate of growth in the young.

Nobunaga et al. (1979) administered 3 or 6 ppm selenium (390 or 780 ug/kg/day, respectively) as selenite to IVCS mice for 30 days prior to mating and throughout gestation. On day 18 of gestation, maternal mice were sacrificed and the embryos removed. Number of litters, total implants, total implants per dam, dead fetuses, dead embryos, resorptions, surviving fetuses (% to total implants), litter size, gross malformations and skeletal anomalies were not significantly different for either selenium-treated or control mice. The only significant effect noted was a decrease in the body weight of surviving fetuses in mice given 6 ppm selenium.

I.A.5. Confidence in the Oral RfD

Study — Medium
Database — High
RfD — High

Confidence in the chosen principal study is medium. Although this is a human epidemiological study in which a sizable population with sensitive subpopulations was studied, there are still several possible interactions that were not fully accounted for, e.g., fluoride intake and protein status. Also, except for clinical signs of selenosis there are no other reliable indicators, biochemical or clinical, of selenium toxicity. Confidence in the database is high because many animal studies and epidemiologic studies (reviewed by Combs and Combs, 1986) support the principal study. An additional human study with a freestanding NOAEL (Longnecker et al., 1991) strongly corroborates the NOAEL identified in the principal study. Therefore, high confidence in the RfD is selected based upon support of the critical study and the high level of confidence in the database.

I.A.6. EPA Documentation and Review of the Oral RfD

Source Document — This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation — U.S. EPA, 1985

Agency Work Group Review — 01/20/1988, 03/22/1989, 09/21/1989, 11/14/1990, 03/27/1991

Verification Date — 03/27/1991

Screening-Level Literature Review Findings — A screening-level review conducted by an EPA contractor of the more recent toxicology literature pertinent to the RfD for Selenium and Compounds conducted in September 2002 identified one or more significant new studies. IRIS users may request the references for those studies from the IRIS Hotline at hotline.iris@epa.gov or (202)566-1676.

I.A.7. EPA Contacts (Oral RfD)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — Selenium and Compounds
CASRN — 7782-49-2

Not available at this time.

II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — Selenium and Compounds
CASRN — 7782-49-2
Last Revised — 03/01/1991

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

NOTE: This assessment is for the following compounds: Selenium (CASRN 7782- 49-2); sodium selenate (CASRN 13410-01-0); sodium selenite (CASRN 10102-18-8); selenious acid (CASRN 7783-00-8); selenic acid (CASRN 7783-08-6); sodium selenide (CASRN 1313-85-5).

II.A. Evidence for Human Carcinogenicity

II.A.1. Weight-of-Evidence Characterization

Classification — D; not classifiable as to carcinogenicity in humans

Basis — Based on inadequate human data and inadequate evidence of carcinogenicity in animals. The evidence for various selenium compounds in animal and mutagenicity studies is conflicting and difficult to interpret; however, evidence for selenium sulfide is sufficient for a B2 (probable human carcinogen) classification.

II.A.2. Human Carcinogenicity Data

Inadequate. Data on the potential carcinogenicity of selenium and various selenium compounds in humans are inadequate. Epidemiological studies have evaluated selenium in blood and cancer death rates in areas of high vs. low naturally-occurring selenium. However, these studies have limited value because they do not assess specific selenium compounds or correlate exposure with cancer risk.

Several investigators have studied the association between serum selenium and the risk of cancer through prospective, case-control and nested case- control studies. Analysis of blood serum levels indicated that patients with cancer, particularly gastrointestinal cancer, prostatic cancer, or Hodgkin's lymphoma, had significantly lower blood selenium levels in blood than healthy patients (Shamberger et al., 1973; Salonen et al., 1984; Kok et al., 1987; Willet et al., 1983; Willet and Stampfer, 1986). The risk of cancer for men (Kok et al., 1987) or for all subjects (Willet et al., 1983) in the lowest quintile of serum selenium was twice that of subjects with higher levels.

Geographic correlational studies have compared cancer mortality in areas of high vs. low levels of naturally-occurring selenium. In an ecological study Shamberger and Frost (1969) reported that an inverse relationship existed between cancer death rates and the selenium concentrations in foliage plants of several Canadian provinces. The human cancer death rate in provinces with selenium-containing plants was 122.2 +/- 7.8 (presumably per 100,000 population although this was not specified), while in the provinces devoid of these plants, the human death rate was 139.9 +/- 4.0.

In an ecological study Shamberger and Willis (1971) reported that there was a correlation between decreased cancer death rates in humans and an increase in the selenium in the forage crops in California. In high-selenium areas (selenium 0.11 ppm of forage crops) the cancer death rate per 100,000 was 141.2. In the medium-selenium areas (0.05-0.10 ppm) the cancer death rate was 190.1. In low-selenium areas (0.02-0.05 ppm) the cancer death rate was 233.0. Shamberger and Willis (1971) also investigated the ratio of observed to expected cancer death rates by anatomic site for men in 17 paired cities including high- and low-selenium areas. The anatomic sites that would come into contact with dietary selenium, such as pharynx, esophagus, stomach, bladder and intestine, showed a substantially lower rate ratio in the high- selenium cities than in the low-selenium cities. Other ecological and prospective studies have correlated an increased

incidence of colon, breast and other forms of cancer in humans in geographic areas where selenium is deficient and a lowered cancer incidence with higher selenium concentrations (Schrauzer and Ishmael, 1974; Shamberger, 1976; Schrauzer et al., 1976; Jansson et al., 1978; Yang et al., 1983).

In a study of approximately 300 employees exposed to selenium (form not specified) in a rectifier (electronics) process over a 26-year period, only 17 deaths occurred, 6 of which were because of cancer (Glover, 1970). This number, however, is not statistically different from the 5.1 deaths expected based on national mortality rates. The source of the mortality rates was not specified. Several toxic effects including pulmonary irritation, epigastric pain and dermal irritation and dermatitis were associated with selenium exposure in men, but no carcinogenic effect was reported.

II.A.3. Animal Carcinogenicity Data

Inadequate. The carcinogenicity of selenium compounds has been evaluated in several animal studies. However, the data are conflicting and difficult to interpret because of apparent anticarcinogenic activity and high toxicity of some selenium salts. In addition, comparison of the available data is difficult because several different salts with varying degrees of bioavailability were used in the assays.

In a 2-year dietary study reported by Nelson et al. (1943), Osborne-Mendel rats (sex not specified) were fed selenium in the form of seleniferous corn or wheat or ammonium potassium selenide at 5-10 ppm. Survival was lower in the treated rats; 53/126 (42%) rats fed selenium survived 18 months or longer compared with 14/18 (78%) control rats. Of the 53 surviving selenium-treated rats, 43 (81%) developed liver cirrhosis and 11 (21%) developed hepatocellular adenoma or carcinoma. All 11 animals with tumors also had liver cirrhosis. None of the 14 control animals surviving 2 years developed liver tumors. Only pooled group data were reported and no statistical analysis was reported.

No tumors developed in a total of 1437 Wistar rats fed sodium selenite or sodium selenate in the diet at levels of 0.5-16 ppm for their lifetime (Harr et al., 1967; Tinsley et al., 1967). Nonneoplastic liver effects such as hyperemia, cellular degeneration, binucleation, and mild proliferation of hepatocytes were observed at concentrations of 4 ppm and higher.

Long-Evans rats (approximately 50/sex/group at study initiation) received 2 ppm (as selenium) sodium selenate or sodium selenite in drinking water for 1 year, then 3 ppm for the remainder of the study (Schroeder and Mitchener, 1971). The treatment of the control group was not discussed. The animals were observed for the duration of their natural lifespan, approximately 36 months, although one selenate-treated female lived for 5 years. Selenite produced 50% mortality

in males by 58 days. At this time, 2 ppm selenate was substituted for selenite in the male group. The concentration of selenium was raised to 3 ppm in this group when the animals were 1 year old; however, the high mortality rendered the group size too small for further statistical analysis. Selenite produced 50% mortality in females by 348 days; selenite- treated females were sacrificed at 23 months due to high mortality. Selenate produced 50% mortality in females by 1014 days and in males by 962 days. In the control groups 50% mortality was achieved by 872 and 853 days in females and males, respectively. Survival of rats receiving selenate was comparable to controls and median lifespan was increased by >100 days. Body weights of treated males were comparable to controls throughout the study. Body weights of females fed selenate were significantly greater than controls at 24 and 36 months; body weights of females fed selenite were significantly less than controls at all times but 18 months.

Incidence of all tumors and of malignant tumors was significantly increased in the selenate-treated rats compared with the controls. Incidence of all tumors in controls, selenate- and selenite-treated rats was 20/65 (30.8%), 30/48 (62.5%) and 4/32 (12.5%), respectively. Incidence of malignant tumors in the same groups was 11/65 (16.9%), 20/48 (41.7%) and 4/32 (12.5%), respectively. The earliest tumor occurred on day 833 in the control males, on day 633 in the control females, on day 344 in selenate males and on day 633 in selenate females. The shortened survival time of the selenite groups was thought to be responsible for the small number of tumors. This study is considered inadequate because only the heart, lung, liver, kidney and spleen tissues from animals necropsied were examined histologically, and an increase in longevity was observed in selenate-treated female rats.

Schroeder and Mitchener (1972) administered 3 ppm sodium selenate or sodium selenite in drinking water to Swiss mice (50/sex/group). Body weights of selenate-treated animals were comparable to controls. Body weights of males fed selenite were significantly increased compared with controls, but body weights of females fed selenite were significantly decreased compared with controls. Longevity in males fed selenate was increased compared with controls. Longevity in females fed selenate increased, but longevity in females fed selenite decreased compared with controls. When compared to controls, there was no significant increase in total tumor incidence or malignant tumor incidence observed in selenium- (form not specified) treated mice. In the control group 23/119 (19%) had tumors (10/119 (8%) malignant tumors). Selenium-fed mice showed 13/88 (15%) tumors (all were malignant). In selenium-treated group 8/13 malignancies were lymphoma or leukemia, 4/13 were papillary or alveologenic adenocarcinoma and 1/13 an osteosarcoma. In the control group there were two incidences of lymphoma or leukemia, 7 of lung carcinoma and 1 carcinoma of unknown origin. The 13 benign tumors included breast and ovary tumors.

II.A.4. Supporting Data for Carcinogenicity

Selenium is an essential micronutrient for several species, including humans, and is part of several enzymes such as glutathione peroxidase, an enzyme involved in cellular defense against oxidative damage, and heme oxidase. While low doses of selenium are essential, high doses of selenium or a deficiency of dietary selenium may cause a toxic response. Additionally, selenium may be protective against tumor development. The greatest daily exposure to selenium is via food. Bioavailability of selenium is dependent on numerous factors, including the intake levels, chemical form and nutritional status. Organic forms of selenium are more bioavailable than inorganic forms; selenates and selenites are the inorganic forms more readily absorbed. Sodium selenate and selenite are soluble in water, but the extent to which they are absorbed dermally or through the gastrointestinal tract has not been fully elucidated (U.S. EPA, 1989).

Shamberger (1985) reported that the oral administration of 0.1-6 ppm or dermal application of 0.005% of selenium reduced incidences of skin, liver, tracheal, intestinal and lung tumors induced by several carcinogens in rats, mice and hamsters. Shamberger theorized that selenium may reduce cellular damage caused by peroxidation of fat. In another study, natural killer (NK) cell activity was significantly increased in female rats administered 0.5 or 2.0 ppm selenium (sodium selenate) in the drinking water for 10 weeks (Koller et al., 1986), suggesting to the authors that NK-sensitive tumors may be prevented by using selenium therapy.

Data on the mutagenicity of selenium and its compounds are equivocal. Selenate and selenite (12 uM) were mutagenic in a reverse mutation assay using Salmonella typhimurium strains TA98, TA100 and TA1537 (Noda et al., 1979) in the absence of rat hepatic homogenates. In a second assay, sodium selenate, but not sodium selenite, was mutagenic; the S. typhimurium strains used were not reported (Lofroth and Ames, 1978). Selenite (selenious acid and sodium selenite) produced DNA damage in Bacillus subtilis strains 17A and 45T; however, selenate (selenic acid and sodium selenate) was negative in the Rec assay (Nakamuro et al., 1976).

Sodium selenide, sodium selenite, and sodium selenate (in order of decreasing activity) caused an increase in unscheduled DNA synthesis in the presence or absence of glutathione in Chinese hamster ovary cells at concentrations of 1.0E-4 M (Whiting et al., 1980). Increased chromosomal aberrations were also produced by sodium selenite at E-5 M in rat lymphocytes (Newton and Lilly, 1986) and by sodium selenite, selenious acid, selenic acid, and selenium oxide at 2.6E-6 M in human lymphocytes (Nakamuro et al., 1976). Sodium selenite produced an increase in chromosomal aberrations in the bone marrow of rats administered a total of 10-12 mg/kg intravenously (near-lethal doses) (Newton and Lilly, 1986). Selenium (elemental), selenium dioxide, sodium selenide, and sodium selenite (in order of decreasing activity) induced an increase in SCEs in human whole-blood cultures; sodium selenate was not mutagenic in this assay (Ray and Altenburg, 1980).

II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure

None

II.C. Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

None

II.D. EPA Documentation, Review, and Contacts (Carcinogenicity Assessment)

II.D.1. EPA Documentation

Source Document — U.S. EPA, 1980, 1984, 1989

The 1989 Health and Environmental Effects Document on Selenium and Compounds has received OHEA review.

II.D.2. EPA Review (Carcinogenicity Assessment)

Agency Work Group Review — 11/09/1989, 03/07/1990

Verification Date — 03/07/1990

Screening-Level Literature Review Findings — A screening-level review conducted by an EPA contractor of the more recent toxicology literature pertinent to the cancer assessment for Selenium and Compounds conducted in September 2002 did not identify any critical new studies. IRIS users who know of important new studies may provide that information to the IRIS Hotline at hotline.iris@epa.gov or (202)566-1676.

II.D.3. EPA Contacts (Carcinogenicity Assessment)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

III. [reserved]
IV. [reserved]
V. [reserved]

VI. Bibliography

Substance Name — Selenium and Compounds
CASRN — 7782-49-2

VI.A. Oral RfD References

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VI.B. Inhalation RfC References

None

VI.C. Carcinogenicity Assessment References

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Yang, G., S. Wang, R. Zhou and S. Sun. 1983. Endemic selenium intoxication of humans in China. Am. J. Clin. Nutr. 37: 872-881.

VII. Revision History

Substance Name — Selenium and Compounds
CASRN — 7782-49-2

Date	Section	Description
03/01/1991	II.	Carcinogenicity assessment on-line
06/01/1991	I.A.	Oral RfD summary on-line
12/03/2002	I.A.6., II.D.2.	Screening-Level Literature Review Findings message has been added.

VIII. Synonyms

Substance Name — Selenium and Compounds
CASRN — 7782-49-2
Last Revised — 03/01/1991

- 7782-49-2
- Selenium
- C.I. 77805
- Caswell No. 732
- ELEMENTAL SELENIUM
- EPA Pesticide Chemical Code 072001
- HSDB 4493
- SELEN [Polish]
- Selenio [Spanish]

- Selenium
- SELENIUM ALLOY
- SELENIUM BASE
- SELENIUM DUST
- SELENIUM ELEMENTAL
- SELENIUM HOMOPOLYMER
- UN 2658
- 13410-01-0
- Selenic acid, disodium salt
- Caswell No. 791
- Disodium selenate
- EPA Pesticide Chemical Code 072002
- Natriumseleniat [German]
- NSC 378348
- Selenic acid, disodium salt
- Sodium selenate
- 10102-18-8
- Selenious acid, disodium salt
- DISODIUM SELENITE
- DISODIUM SELENIUM TRIOXIDE
- HSDB 768
- Natriumselenit [German]
- SELENIOUS ACID, DISODIUM SALT
- SODIUM SELENITE
- UN 2630
- 7783-00-8
- Selenious acid
- HSDB 6065
- MONOHYDRATED SELENIUM DIOXIDE
- Selenious Acid
- 7783-08-6
- Selenic acid
- Acide selenique [French]
- Acido selenico [Spanish]
- HSDB 675
- Selenic acid
- UN 1905
- 1313-85-5
- Sodium selenide [Na2Se]
- Disodium monoselenide
- Sodium selenide