

Thallium (I), soluble salts; CASRN Various

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 Thallium (I), soluble salts

File First On-Line 01/31/1987

Category (section)	Assessment Available?	Last Revised
Oral RfD (I.A.)	qualitative discussion	09/30/2009
Inhalation RfC (I.B.)	qualitative discussion	09/30/2009
Carcinogenicity Assessment (II.)	yes	09/30/2009

I. HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE (RfD) FOR CHRONIC ORAL EXPOSURE

Substance Name — Thallium (I), soluble salts

CASRN — Various

Section I.A. Last Revised — 09/30/2009

The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfD is intended for use in risk assessments for health effects known or assumed to be produced through a nonlinear (presumed threshold) mode of action. It is expressed in units of mg/kg-day. Please refer to the guidance documents at <http://www.epa.gov/iris/backgrd.html> for an elaboration of these concepts. Because RfDs can be derived for the noncarcinogenic health effects of

substances that are also carcinogens, it is essential to refer to other sources of information concerning the carcinogenicity of this chemical 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.

Previously, the IRIS database contained separate IRIS summaries for each of the five soluble thallium salts (thallium (I) acetate, thallium (I) carbonate, thallium (I) chloride, thallium (I) nitrate, and thallium (I) sulfate) that were posted in 1988. The previous RfD values for these soluble salts, ranging from 8×10^{-5} to 9×10^{-5} mg/kg-day, were based on the same principal study considered in the current assessment (Midwest Research Institute [MRI] [1988], previously cited as U.S. EPA [1986]). In the previous assessment, the high-dose group in the principal study (0.25 mg/kg-day thallium sulfate or 0.20 mg/kg-day Tl) was identified as the no-observed-adverse-effect level (NOAEL) and a NOAEL-LOAEL approach was used to identify the point of departure (POD). The previous RfD was derived by using a composite uncertainty factor (UF) of 3,000 (10 to extrapolate from subchronic to chronic data, 10 for intraspecies extrapolation, 10 to account for interspecies variability, and 3 to account for lack of reproductive and chronic toxicity data).

I.A.1. CHRONIC ORAL RfD SUMMARY

The 90-day oral toxicity study of thallium (I) sulfate in Sprague-Dawley rats (MRI, 1988) was identified as a potential principal study. PODs for two alternative endpoints from the MRI study (1988) were selected for dose-response analysis: (1) hair follicle atrophy in female rats with alopecia and (2) clinical observations including those related to animal coat (rough coat, piloerection, shedding, and alopecia), eyes (including lacrimation, exophthalmos, and miosis), and behavior. Based on hair follicle atrophy in female rats, the high dose (0.2 mg/kg-day Tl) was identified as a lowest-observed-adverse-effect-level (LOAEL); the mid dose (0.04 mg/kg-day Tl) was considered a NOAEL and was used as a candidate POD. Benchmark dose (BMD) modeling methods were used to analyze clinical observation data (U.S. EPA, 2000). Based on incidence of alopecia in female rats as representative of clinical observation data, the average 95% lower bound on the dose corresponding to a 10% excess risk ($BMDL_{10}$) was 0.01 mg/kg-day Tl and was also considered a candidate POD.

A total uncertainty factor of 3,000 (10 for interspecies extrapolation, 10 for intraspecies extrapolation, 3 for extrapolation from a subchronic to chronic study, and 10 for database deficiencies) was applied to the PODs to yield candidate RfD values for thallium (in the form of soluble thallium salts) of 1×10^{-5} mg/kg-day Tl (for hair follicle atrophy) or 3×10^{-6} mg/kg-day Tl (for clinical observations).

The available toxicity database for thallium contains studies that are generally of poor quality. The MRI (1988) study that was selected as a candidate principal study suffers from certain critical limitations (e.g., high background incidence of alopecia, lack of histopathological examination of skin tissue in low- and mid-dose groups, and inadequate examination of objective measures of neurotoxicity), and there are particular difficulties in the selection of appropriate endpoints. Therefore, even though an RfD would generally be derived with a combined uncertainty factor of 3000, and RfD for soluble thallium salts is not derived in this specific case. For further discussion, see the *Toxicological Review of Thallium and Compounds* (U.S. EPA, 2009).

I.A.2. PRINCIPAL AND SUPPORTING STUDIES

Not applicable

I.A.3. UNCERTAINTY FACTORS

Not applicable

I.A.4. ADDITIONAL STUDIES/COMMENTS

Symptoms of thallium toxicity are diverse in both humans and animals. The nervous system as a target organ of thallium is supported by observations from human case reports and animal studies. Relatively high doses of thallium caused neurological symptoms in humans (e.g., paresthesia of the hands and feet, weakness, tremors, coma, and convulsions). Some of these neurological symptoms (e.g., paresthesia and weakness) were reversible, although recovery was slow. Other effects, including mental and/or psychological problems, were more persistent. Neurological symptoms also were associated with chronic exposure to thallium in humans. These symptoms included sleep disorders, tiredness, weakness, nervousness, headache, other psychic alterations, and neurological and muscular problems. In experimental animal studies, thallium exposure was associated with biochemical changes, lipid peroxidation, and histopathologic changes in the brain and functional and histopathologic changes in peripheral nerves. The areas affected in the brain differed with the age of the treated animal; nevertheless, all measured endpoints (symptoms, biochemical measurements, and histopathology) indicated that high doses (close to lethal doses) of thallium induced significant degradation of the nervous system. Results from in vitro studies further confirmed these observations. See the *Toxicological Review of Thallium and Compounds* (U.S. EPA, 2009) for a more detailed summary and specific references.

In a 90-day gavage study (MRI, 1988), rats exposed to thallium sulfate showed consistently increased incidences of clinical observations related to the coat (rough coat, piloerection,

shedding, and alopecia), eyes (lacrimation, exophthalmos, and miosis), and behavioral signs compared with untreated or vehicle controls. The underlying mode of action for these clinical observations is not known. Collectively, however, these observations suggest a treatment-related effect of thallium on the rat and possibly an indirect measure of stress or other effects on the nervous system. For example, it has been suggested that barbering (or overgrooming) in rodents may represent a stress-evoked behavioral response or other nervous system dysfunction (Welch et al., 2007; Kalueff et al., 2006; Kalueff and Tuohimaa, 2005; Greer and Capecchi, 2002).

In humans, it is generally alopecia that leads to a diagnosis of thallium poisoning. Alopecia occurs about 2 weeks after exposure and is reversible after exposure to thallium is discontinued. Alopecia has also been repeatedly observed in experimental animals exposed to thallium compounds. See the *Toxicological Review of Thallium and Compounds* (U.S. EPA, 2009) for a more detailed summary and specific references.

Thallium exposure in humans has been associated with respiratory effects and gastrointestinal effects, including diarrhea and vomiting. Other toxic effects associated with oral thallium exposure in humans and animals are changes in blood pressure (high, low, and fluctuating values have all been noted) and liver and kidney damage (kidney damage is age dependent and occurs only in mature kidneys), all of which appear to be reversible with the removal of thallium exposure. Doses that do not affect survival have been shown to affect clinical chemistry parameters, such as ALT, AST, BUN, blood glucose, and blood sodium levels, indicating liver and kidney damage with subchronic exposures (Leung and Ooi, 2000; Appenroth et al., 1996, 1995; Fleck and Appenroth, 1996; El-Garawany et al., 1990; Mourelle et al., 1988).

Thallium compounds have been shown to affect reproductive function. A dose as low as 0.7 mg/kg-day TI (10 ppm of thallium (I) sulfate) resulted in testicular damage and reduced sperm motility in male Wistar rats within 60 days (Formigli et al., 1986). Wei (1987) reported that doses to male Kunming mice as low as 0.001 mg/L in the drinking water for 6 months reduced sperm motility (rapid speed only), and 0.01 mg/L reduced overall sperm motility and sperm counts and caused a reduction of live offspring. Confidence in this study, however, is low (see the *Toxicological Review of Thallium and Compounds* [U.S. EPA, 2009] for a more detailed discussion of the limitations of this study).

Limited data in humans and experimental animals suggest that thallium may produce developmental toxicity. A review of case studies of women exposed orally to high levels (approximately 120 to 1,100 mg) of thallium during pregnancy suggested a trend toward premature and low-birth-weight infants, especially if exposure took place in the last trimester; no other developmental abnormalities were identified (Hoffman, 2000). Dolgner et al. (1983)

examined birth defects in a German population living near a cement plant emitting thallium dusts during the mid-1970s and found a higher incidence of congenital malformations than the incidence documented in the government birth records from the area. The association between the number of birth defects and thallium exposure was weak, however, because two of the malformations were considered hereditary and the incidence of birth defects, although greater than that determined from civil records, was consistent with that reported in the literature. Confidence in this study was limited by lack of exposure data during pregnancy and possible underreporting in controls. In vivo data in rats support an association between intraperitoneal thallium exposure and low birth weight, although such an association has not been reported with orally administered thallium. In vitro data demonstrated an increase in bone malformations in both rat and chick embryos. See the *Toxicological Review of Thallium and Compounds* (U.S. EPA, 2009) for a more detailed summary and specific references.

For more detail on Susceptible Populations, exit to [the toxicological review, Section 4.8 \(PDF\)](#).

I.A.5. CONFIDENCE IN THE CHRONIC ORAL RfD

Not applicable

For more detail on Characterization of Hazard and Dose Response, exit to [the toxicological review, Section 6 \(PDF\)](#).

I.A.6. EPA DOCUMENTATION AND REVIEW OF THE CHRONIC ORAL RfD

Source Document — U.S. EPA, 2009

This document has been reviewed by EPA scientists, interagency reviewers from other federal agencies and White House offices, and the public, and peer reviewed by independent scientists external to EPA. A summary and EPA's disposition of the comments received from the independent external peer reviewers and from the public is included in Appendix A of the *Toxicological Review of Thallium and Compounds* (U.S. EPA, 2009). [To review this appendix, exit to the toxicological review, Appendix A, Summary Of External Peer Review And Public Comments And Disposition \(PDF\)](#).

I.A.7. EPA CONTACTS

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 (email address).

I.B. REFERENCE CONCENTRATION (RfC) FOR CHRONIC INHALATION EXPOSURE

Substance Name — Thallium (I), soluble salts

CASRN — Various

Section I.B. Last Revised — 09/30/2009

The RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfC considers toxic effects for both the respiratory system (portal of entry) and for effects peripheral to the respiratory system (extrarespiratory effects). The inhalation RfC (generally expressed in units of mg/m^3) is analogous to the oral RfD and is similarly intended for use in risk assessments for health effects known or assumed to be produced through a nonlinear (presumed threshold) mode of action.

Inhalation RfCs are derived according to *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry* (U.S. EPA, 1994). Because RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens, it is essential to refer to other sources of information concerning the carcinogenicity of this chemical 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.

An inhalation RfC for soluble thallium salts was not previously available on the IRIS database.

I.B.1. CHRONIC INHALATION RfC SUMMARY

An inhalation RfC for soluble thallium salts is not available at this time. There are no comprehensive inhalation toxicity studies involving exposure to any thallium compound.

I.B.2. PRINCIPAL AND SUPPORTING STUDIES

Not applicable.

I.B.3. UNCERTAINTY FACTORS

Not applicable.

I.B.4. ADDITIONAL STUDIES/COMMENTS

Not applicable.

For more detail on Susceptible Populations, exit to [the toxicological review, Section 4.8 \(PDF\)](#).

I.B.5. CONFIDENCE IN THE CHRONIC INHALATION RfC

Not applicable.

For more detail on Characterization of Hazard and Dose Response, exit to [the toxicological review, Section 6 \(PDF\)](#).

I.B.6. EPA DOCUMENTATION AND REVIEW OF THE CHRONIC INHALATION RfC

Source Document — U.S. EPA, 2009

This document has been reviewed by EPA scientists, interagency reviewers from other federal agencies and White House offices, and the public, and peer reviewed by independent scientists external to EPA. A summary and EPA's disposition of the comments received from the independent external peer reviewers and from the public is included in Appendix A of the *Toxicological Review of Thallium and Compounds* (U.S. EPA, 2009). [To review this appendix, exit to the toxicological review, Appendix A, Summary Of External Peer Review And Public Comments And Disposition \(PDF\)](#).

I.B.7. EPA CONTACTS

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 (email address).

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name — Thallium (I), soluble salts

CASRN — Various

Section II. Last Revised — 09/30/2009

This section 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 and inhalation exposure. Users are referred to Section I of this file for information on long-term toxic effects other than carcinogenicity.

The rationale and methods used to develop the carcinogenicity information in IRIS are described in the *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005a) and the *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* (U.S. EPA, 2005b). The quantitative risk estimates are derived from the application of a low-dose extrapolation procedure, and are presented in two ways to better facilitate their use. First, route-specific risk values are presented. The "oral slope factor" is a plausible upper bound on the estimate of risk per mg/kg-day of oral exposure. Similarly, a "unit risk" is a plausible upper bound on the estimate of risk per unit of concentration, either per µg/L drinking water (see Section II.B.1.) or per µg/m³ air breathed (see Section II.C.1.). Second, the estimated concentration of the chemical substance in drinking water or air when associated with cancer risks of 1 in 10,000, 1 in 100,000, or 1 in 1,000,000 is also provided.

The previous IRIS assessments for soluble thallium salts (thallium (I) acetate, thallium (I) carbonate, thallium (I) chloride, thallium (I) nitrate, and thallium (I) sulfate) (posted to the IRIS database in 1990) assigned these compounds a weight-of-evidence classification of D, "not classifiable as to human carcinogenicity."

II.A. EVIDENCE FOR HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CHARACTERIZATION

Under the *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005a), the database for thallium and compounds provides "inadequate information to assess carcinogenic potential." There are presently no studies that evaluate the carcinogenic potential of thallium in animals and no adequate studies of humans chronically exposed to thallium.

For more detail on Characterization of Hazard and Dose Response, exit to [the toxicological review, Section 6 \(PDF\)](#).

For more detail on Susceptible Populations, exit to [the toxicological review, Section 4.8 \(PDF\)](#).

II.A.2. HUMAN CARCINOGENICITY DATA

The data available for human carcinogenicity are inadequate. Medical records for 86 workers (sex and length of employment not reported) occupationally exposed to thallium at a magnesium seawater battery factory and 79 unexposed workers matched for age, length of employment, shift pattern, and type of work were examined (Marcus, 1985). No increase in incidence of benign neoplasms (site not specified) was observed. This study is limited by the examination of medical records only, lack of exposure quantitation, small cohort size, and unknown length of observation.

In another study, the health effects associated with exposure to thallium in 128 men (age 16-62 years) exposed for 1-42 years (average = 19.5 years) in three cement manufacturing plants were reported (Schaller et al., 1980). Analyses of roasted pyrites and electro-filter dust confirmed the presence of thallium in various production areas in the plant. Urinary thallium was elevated in the workers. The health evaluation, consisting of a medical history and a physical exam, did not show any indication of thallium poisoning. However, this health evaluation was not adequate to detect any oncogenic response.

II.A.3. ANIMAL CARCINOGENICITY DATA

There are no animal carcinogenicity studies of thallium. Several subchronic animal studies on thallium and compounds are available; however, they were not designed to examine carcinogenic endpoints.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Thallium (I) compounds were not mutagenic in reverse mutation assays using *Salmonella typhimurium* strains TA98, TA100, TA1535, and TA1538 and *Escherichia coli* strains B/r WP2 try and WP2 hcr try; data were not available regarding the presence of metabolic activation (Kanematsu et al., 1980). Positive results were obtained in the Rec assay using 0.001 M thallium nitrate with *Bacillus subtilis* strains H17 and M45; presence of metabolic activation was not reported (Kada et al., 1980; Kanematsu et al., 1980). Thallium nitrate was negative in a screening assay for mitotic gene conversion and reverse mutations in *Saccharomyces cerevisiae* (Singh, 1983). Thallium nitrate produced negative effects on cell division in *S. cerevisiae* and *E. coli* (Loveless et al., 1954). Cytotoxic levels (1,000 µg/mL) of thallium acetate depressed DNA synthesis in Chinese hamster ovary cells (Garrett and Lewtas, 1983). Single-strand DNA breaks occurred in mouse and rat embryo fibroblasts exposed to

thallium carbonate at 10^{-6} to 10^{-4} M (Zasukhina et al., 1983). Thallium carbonate (0.005-0.5 $\mu\text{g}/\text{kg}\text{-day}$) was positive in a dominant lethal test in male white rats (Zasukhina et al., 1983); however confidence in this study is low.

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

Not applicable.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

Not applicable.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

II.D.1. EPA DOCUMENTATION

Source Document — U.S. EPA, 2009

This document has been reviewed by EPA scientists, interagency reviewers from other federal agencies and White House offices, and the public, and peer reviewed by independent scientists external to EPA. A summary and EPA's disposition of the comments received from the independent external peer reviewers and from the public is included in Appendix A of the *Toxicological Review of Thallium and Compounds* (U.S. EPA, 2009). [To review this appendix, exit to the toxicological review, Appendix A, Summary Of External Peer Review And Public Comments And Disposition \(PDF\).](#)

II.D.2. EPA REVIEW

II.D.3. EPA CONTACTS

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 (email address).

III. [reserved]

IV. [reserved]

V. [reserved]

VI. BIBLIOGRAPHY

Substance Name — Thallium (I), soluble salts

CASRN — Various

VI.A. ORAL RfD REFERENCES

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VI.B. INHALATION RfC REFERENCES

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VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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VII. REVISION HISTORY

Substance Name — Thallium (I), soluble salts
CASRN — Various
File First On-Line — 01/31/1987

The following entries apply to all soluble thallium salts except where indicated with a specific chemical name.

Date	Section	Description
03/01/1988	I.A.	RfD changed—new study (thallium sulfate)
06/30/1988	I.A.	RfD withdrawn
09/07/1988	I.A.	Revised oral RfD summary added
09/01/1990	II.	Carcinogen assessment online

Date	Section	Description
12/03/2002	I.A.6, II.D.2.	Screening-level literature review findings message has been added (thallium sulfate, carbonate, chloride, nitrate).
10/28/2003	I.A.6, II.D.2.	Screening-level literature review findings message has been added (thallium acetate).
09/30/2009	All	IRIS Summaries for five thallium soluble salts combined into a single Summary. RfD, RfC, and cancer sections updated. RfD removed.

VIII. SYNONYMS

Substance Name — Thallium (I), soluble salts

CASRN — Various

Last Revised — 09/30/2009

Thallium acetate

- CASRN — 563-68-8
- $\text{TlC}_2\text{H}_3\text{O}_2$
- RCRA Waste Number u214
- Thallium (1+) acetate
- Thallium acetate
- Thallium (I) acetate
- Thallium monoacetate
- Thallous acetate
- Acetic acid, thallium (1+) salt

Thallium carbonate

- CASRN — 6533-73-9
- Tl_2CO_3
- Carbonic acid, dithallium (1+) salt
- Dithallium carbonate
- Thallium carbonate

- Thallium (I) carbonate
- Thallous carbonate

Thallium chloride

- CASRN — 7791-12-0
- TlCl
- Thallium (1+) chloride
- Thallium chloride
- Thallium (I) chloride
- Thallium monochloride
- Thallous chloride

Thallium nitrate

- CASRN — 10102-45-1
- TlNO₃
- RCRA Waste Number U217
- Thallium (1+) salt nitric acid
- Thallium mononitrate
- Thallium nitrate
- Thallous nitrate
- UN 2727
- Thallium (I) nitrate

Thallium sulfate

- CASRN — 7446-18-6
- Tl₂SO₄
- C.F.S.
- Dithallium sulfate
- Sulfuric acid, dithallium (1+) salt
- Sulfuric acid, thallium (1+) salt
- Thallium (I) sulfate
- Thallium sulfate
- Thallous sulfate