Australian Government
Department of Health and Aged Care
Australian Industrial Chemicals Introduction Scheme

Tellurium and its inorganic compounds

Evaluation statement (EVA00151)

16 December 2024

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AICIS evaluation statement (EVA00151)

Subject of the evaluation

Tellurium and its inorganic compounds

Chemicals in this evaluation

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Tellurium oxide (TeO_2)	7446-07-3
Telluric acid (H_2TeO_3), potassium salt (1:2)	7790-58-1
Telluric acid (H_6TeO_6)	7803-68-1
Tellurium chloride ($TeCl_4$), (*T*-4)-	10026-07-0
Telluric acid (H_2TeO_3)	10049-23-7
Tellurium	13494-80-9

Reason for the evaluation

Evaluation Selection Analysis indicated a potential human health risk.

Parameters of evaluation

These chemicals tellurium and its inorganic compounds are listed on the Australian Inventory of Industrial Chemicals (the Inventory). The evaluation is a human health risk assessment for all identified industrial uses of chemicals in this group.

The chemicals are grouped together based on a proposed common metabolic pathway. Hence, this evaluation will focus specifically on systemic toxicity. Conclusions for other hazard endpoints will be made where data is available.

This evaluation does not cover nanoforms of these chemicals.

In this evaluation these chemicals will be referred to as:

- tellurium (CAS no. 13494-80-9)
- tellurium oxide (CAS no. 7446-07-3)
- tellurium tetrachloride (CAS no. 10026-07-0)
- tellurous acid (CAS no. 10049-23-7)
- potassium tellurite (CAS no. 7790-58-1)
- telluric acid (CAS no. 7803-68-1).

Summary of evaluation

Summary of introduction, use and end use

There is currently no specific information about the introduction, use and end use of these chemicals in Australia.

Based on international use information, these chemicals have predominantly site-limited uses. Tellurium and tellurium oxide are used as catalysts and in the manufacture of a range of products including metals, vulcanised rubber, glass and ceramics as well as in a range of electronic products. Tellurium tetrachloride is also used as a catalyst. Limited use information is available for telluric acid, tellurous acid and its salt; however, historically they have been used as antimicrobial agents.

Human health

Summary of health hazards

The identified health hazards are based on available data for tellurium and tellurium oxide. Limited data are available for the other chemicals in this group. The chemicals in this group are considered to share the same metabolic pathway; therefore, data for tellurium and tellurium oxide have been used to support hazard conclusions for systemic endpoints.

Based on the available data these chemicals may cause specific adverse effects on fertility and development warranting classification and may cause effects in offspring via lactation.

In a reproductive screening study with tellurium, a reduction in gestation index and a prolonged gestation period were observed in the absence of severe general toxicity. At the highest dose, effects on fertility and atrophy of female reproductive organs were observed in the presence of pronounced general toxicity. There is no information on possible effects on reproductive organs at doses without severe general toxicity.

Severe developmental effects (hydrocephalus and/or foetal and pup mortality) were consistently seen in the screening study and all available pre-natal developmental toxicity studies with tellurium and tellurium oxide, even at the lowest dose and without any maternal toxicity. Tellurium caused hydrocephalus in 2 animal species (rats and rabbits).

Transfer of tellurium compounds with low water solubility, from dams via lactation to their offspring, has been demonstrated and there is evidence of adverse effects (neuropathies) in offspring due to transfer in milk. Although similar effects could occur with tellurium compounds with higher solubilities in the absence of toxicokinetic data and uncertainty as to the nature of the tellurium species that is transferred by lactation (i.e. parent chemical or metabolite) it is not considered appropriate to read across for effects via lactation from chemicals with low water solubility (tellurium, tellurium oxide and tellurous acid) to other chemicals in this evaluation.

In repeated dose toxicity studies, primary targets for tellurium and tellurium oxide after oral exposure are the nervous system and the liver. Neuropathy was observed after and administration of high oral doses. Reversible liver effects occurred at low doses with no evidence of severe liver dysfunction. Considering these effects occurred only at high doses or were reversible, hazard classification is not warranted. However, the doses at which

systemic toxicity effects may occur may vary between compounds due to differences in the oxidation state and bioavailability.

The limited available data indicate that repeated inhalation of tellurium may cause serious toxic effects however, there is insufficient information to warrant hazard classification.

Conclusions for other hazard endpoints have been drawn where data are available.

Based on the available data, tellurium and tellurium oxide:

- have low acute oral toxicity (based on LD50s)
- are not skin or eye irritants
- are not considered to have genotoxic potential.

Observation in humans suggests that tellurium compounds can cause headache, nausea, vomiting, cyanosis, weight loss, alopecia, and garlic like odour after a single exposure.

Based the limited available LD50 values, telluric acid, tellurous acid and potassium tellurite may have high acute toxicity; however, there is insufficient information to support hazard classification.

Tellurium tetrachloride is expected to be corrosive at the point of contact due to the formation of hydrochloric acid when it comes in contact with water. Tellurous acid, telluric acid and potassium tellurite may all have irritation potential; however, in the absence of experimental data no hazard conclusions can be drawn.

There is insufficient information available to draw any conclusions on skin sensitisation.

For further details of the health hazard information see **Supporting information**.

Hazard classifications relevant for worker health and safety

These chemicals satisfy the criteria for classification to the Globally Harmonised System of Classification and Labelling of Chemicals (GHS) (UNECE 2017) for hazard classes relevant for work health and safety as follows.

The classification for effects via lactation only applies to tellurium (CAS no. 13494-80-9), tellurium oxide (CAS no. 7446-07-3) and tellurous acid (CAS no. 10049-23-7).

This does not consider classification of physical hazards and environmental hazards.

Health hazards	Hazard category	Hazard statement
Reproductive toxicity	Repr. 1B	H360Df: May damage the unborn child. Suspected of damaging fertility.
Reproductive toxicity	Effects on or via lactation	H362: May cause harm to breast-fed children.

Summary of health risk

Public

Based on the available use information it is unlikely that the public will be exposed to these chemicals. Therefore, there are no identified risks to the public that require management.

Workers

During site-limited processes, dermal, inhalation and ocular exposure might occur, particularly where manual or open processes are used. These could include transfer and blending activities, quality control analysis, and cleaning and maintaining equipment. Good hygiene practices to minimise incidental oral exposure are expected to be in place.

Given the critical systemic effects, the chemicals could pose risk to workers. In addition, there is uncertainty regarding local effects, acute systemic effects and effects following inhalation exposure. Control measures to minimise oral, dermal, inhalation and ocular exposure are needed to manage the risk to workers (see **Proposed means for managing risk** section). Controls in place due to reproductive toxicity classification and workplace exposure limits should minimise the potential risks relating to other toxicity endpoints.

Proposed means for managing risk

Workers

Recommendation to Safe Work Australia

It is recommended that Safe Work Australia (SWA) update the Hazardous Chemical Information System (HCIS) to include classifications relevant to work health and safety (**see Summary of Health Hazards Section**).

Information relating to safe introduction and use

The information in this statement including recommended hazard classifications, should be used by a person conducting a business or undertaking at a workplace (such as an employer) to determine the appropriate controls under the relevant jurisdiction Work Health and Safety laws.

Control measures that could be implemented to manage the risk arising from exposure to these chemicals include, but are not limited to:

- using closed systems or isolating operations
- using local exhaust ventilation to prevent these chemicals from entering the breathing zone of any worker
- minimising manual processes and work tasks through automating processes
- adopting work procedures that minimise splashes and spills
- cleaning equipment and work areas regularly
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with these chemicals.

Measures required to eliminate or manage risk arising from storing, handling and using these hazardous chemicals depends on the physical form and how these chemicals are used.

These control measures may need to be supplemented with:

- conducting health monitoring for any worker who is at significant risk of exposure to these chemicals if valid techniques are available to monitor the effect on the worker's health
- conducting air monitoring to ensure control measures in place are working effectively and continue to do so
- personal protective equipment should not solely be relied upon to control risk and should only be used when all other reasonably practicable control measures do not eliminate or sufficiently minimise risk.

Model codes of practice, available from the Safe Work Australia website, provide information on how to manage risks of hazardous chemicals in the workplace, prepare an SDS and label containers of hazardous chemicals. Your Work Health and Safety regulator should be contacted for information on Work Health and Safety laws and relevant Codes of Practice in your jurisdiction.

Conclusions

The Executive Director is satisfied that the identified risks to human health from the introduction and use of the industrial chemicals can be managed.

Note:

1. Obligations to report additional information about hazards under *Section 100* of the *Industrial Chemicals Act* 2019 apply.
2. You should be aware of your obligations under environmental, workplace health and safety and poisons legislation as adopted by the relevant state or territory.

Supporting information

Grouping rationale

The 6 chemicals in this evaluation are tellurium and its inorganic compounds. The group contains:

- elemental tellurium (oxidation state 0)
- tellurium oxide (oxidation state +IV)
- tellurium tetrachloride (oxidation state +IV)
- oxoacids of tellurium
- tellurous acid (H_2TeO_3) and its potassium salt (oxidation state +IV)
- telluric acid (H_6TeO_6) (oxidation state +VI).

All chemicals in this group are expected to metabolise in humans and form common metabolites (see **Toxicokinetics**). Therefore, the types of systemic adverse effects for the group are expected to be similar.

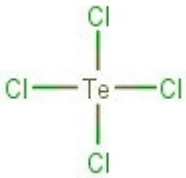
However, the acute and local effects for this group are expected to differ based on differences in acidity, water solubility (see **Relevant Physical and Chemical Properties**) and oxidation state.

Chemical identity

CAS number	13494-80-9
CAS name	Tellurium
Molecular formula	Te
Associated names	Tellurium, metallic Tellurium elemental
Molecular weight (g/mol)	127.60
SMILES (canonical)	[Te]
Structural formula	

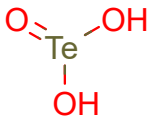
Te

CAS number	7446-07-3
CAS name	Tellurium oxide (TeO_2)
Molecular formula	O_2Te
Associated names	Tellurium dioxide
Molecular weight (g/mol)	159.60
SMILES (canonical)	O=[Te]=O
Structural formula	

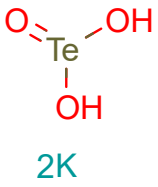
CAS number	10026-07-0
CAS name	Tellurium chloride ($TeCl_4$), (*T*-4)-
Molecular formula	Cl_4Te
Associated names	Tellurium tetrachloride
Molecular weight (g/mol)	269.41
SMILES (canonical)	Cl[Te](Cl)(Cl)Cl
Structural formula	

CAS number	10049-23-7
CAS name	Telluric acid (H_2TeO_3)
Molecular formula	H_2O_3Te
Associated names	Tellurous acid
Molecular weight (g/mol)	177.62
SMILES (canonical)	O=[Te](O)O

Structural formula

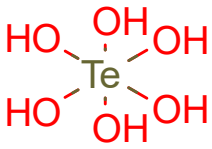


CAS number	7790-58-1
CAS name	Telluric acid (H_2TeO_3), potassium salt (1:2)
Molecular formula*	$H_2O_3Te.2K$
Associated names	Telluric acid (H_2TeO_3), dipotassium salt
	Potassium tellurite
Molecular weight (g/mol)*	255.81
SMILES (canonical)	[K].O=[Te](O)O
Representative formula*	



Additional chemical identity information
* This chemical is a salt and has been represented according to CAS nomenclature/identity convention

CAS number	7803-68-1
CAS name	Telluric acid (H_6TeO_6)
Molecular formula	H_6O_6Te
Associated names	Orthotelluric acid
	Telluric(VI) acid (H_6TeO_6)
	Tellurium hydroxide ($Te(OH)_6$)
Molecular weight (g/mol)	229.65
SMILES (canonical)	O[Te](O)(O)(O)(O)O



Relevant physical and chemical properties

Tellurium is a metalloid that belongs to the chalcogen family (Group 16 on the periodic table).

The following relevant physicochemical properties have been identified (MAK 2006; Merck n.d.):

- Tellurium and tellurium oxide share similar physicochemical properties. They are insoluble in water and have high melting and boiling points.
- Tellurous acid (H_2TeO_3) is reported to be barely soluble in water whereas its potassium salt (K_2TeO_3) is reported to be soluble in water.
- Telluric acid (H_6TeO_6) is reported to be soluble in water (about 33% at 30°C) and is a very weak acid.
- Tellurium tetrachloride is a very hygroscopic solid which decomposes in water forming TeO_2 and HCl.

Introduction and use

Australia

There is currently no specific information about the introduction, use and end use of the chemical in Australia.

International

The following international uses have been identified through the:

- Galleria Chemica (ChemWatch)
- European Union (EU) Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) dossiers
- US Environmental Protection Agency Chemical Data Reporting (CDR) (US EPA 2020)
- European Chemicals Agency (2020) Committee for Risk Assessment (RAC - Annex 1)
- Gad SC & Pham T (2014) Tellurium. Encyclopedia of Toxicology (Third edition) 2014
- United States Environmental Protection Agency (US EPA) CompTox Chemical Dashboard v2.3.0
- Various published literature (Gerhardsson 2015, Vavrova et al. 2021, Zhu et al. 2023).

Tellurium and tellurium oxide have the following site-limited uses as catalysts and in the manufacture of:

- basic metals, including alloys
- in silverware
- in vulcanised rubber
- rewritable CDs and DVDs
- semiconductors
- glass and ceramics as a colouring agent
- storage batteries.

Tellurium tetrachloride is used as a catalyst in metal coatings.

Telluric acid, tellurous acid and potassium tellurite have reported uses as laboratory reagents.

Tellurium and its soluble salts have historic use as antimicrobials (Vavrova et al. 2021).

Existing Australian regulatory controls

AICIS

No specific controls are currently available for these chemicals.

Public

No specific controls are currently available for these chemicals.

Workers

Tellurium and compounds (as Te) are listed on the Hazardous Chemical Information system (HCIS) (SWA) with a workplace exposure standard (WES) of 0.1 mg/m^3 (8-hour time weighted average (TWA)).

From 1 December 2026 and following implementation into the work health and safety laws of the Commonwealth, states and territories, new Workplace Exposure Limits (WEL) for airborne contaminants (WEL list) will be adopted throughout Australia. The WEL for tellurium and compounds (as Te) is the same as the WES (TWA 0.1 mg/m^3).

International regulatory status

Exposure standards

The following Temporary Emergency Exposure Limits (TEELs) have been recommended by the United States Department of Energy (Chemwatch n.d):

- 110 mg/m^3 (TEEL-3)
- 20 mg/m^3 (TEEL-2)
- 1.8 mg/m^3 (TEEL-1)

The majority of international exposure standards for tellurium and its compounds (as Te) (excluding hydrogen telluride and tellurium hexafluoride) have time weighted exposure (TWA; 8-hour) that are consistent with Australian exposure standard of 0.1 mg/m^3 including

in: Argentina, Austria, Belgium, Canada, Denmark, Finland, Greece, Iceland, New Zealand and United States of America (ChemWatch n.d).

Short Term Exposure Limits (STEL; 15 minutes) in the range of 0.01–2.4 mg/m^3 were reported for Belarus, Austria, Denmark, Canada, Finland, Germany (ChemWatch n.d).

Canada

Tellurium and its compounds are listed in the Cosmetic Ingredient Hotlist that are Prohibited for use in Cosmetic Products (Government of Canada 2022).

European Union

As part of ECHA's Substance Regulatory Obligations tellurium and its compounds (CAS no. 13494-80-9) and tellurium oxide (CAS no. 7446-07-3) are listed as prohibited substances in the following:

- Cosmetics Regulation 1223/2009 Annex II - List of substances Prohibited in Cosmetic Products (EU 2023).
- Food Contact Active and Intelligent Materials and Articles Regulation: Carcinogens, Mutagens and Reproductive substances (CMRs) that are not allowed for use in components of active and intelligent materials and articles intended to come into contact with food.

New Zealand

Tellurium and its compounds are listed on the Cosmetic Products Group Standard as Schedule 4 - Components cosmetic products must not contain (NZ EPA 2019).

Asia

Tellurium and its compounds are listed in the ASEAN Cosmetic directive Annex II Part 1 - List of substances which must not form part of the composition of cosmetic products (ASEAN 2019).

Health hazard information

In the absence of toxicological data, and based on an assumed common metabolic pathway, available data for tellurium and tellurium oxide are used to predict systemic toxicity of the chemicals in this evaluation. However, the doses at which systemic toxicity effects may occur may vary between compounds due to differences in the oxidation state and bioavailability.

Toxicokinetics

Based on the available animal and human data, tellurium and its inorganic compounds can be absorbed via oral and inhalation exposure. In healthy male volunteers, oral absorption of tellurate (TeO_4^{2-} / Te^{6+}), tellurite (TeO_3^{2-} / Te^{4+}) and elemental tellurium (Te^0) was 17.5–35.5%, 21.5% and 6–14%, respectively (Kron et a l. 1991). In rats and rabbits, the absorption was 10% and 40% for elemental tellurium and sodium tellurite, respectively (MAK 2006). There are some reports (limited details available) that tellurium can be absorbed through skin (Gad & Pham 2014; MAK 2006).

Tellurium can also enter the organism via the lungs (MAK, 2006). No information on dermal uptake was identified.

Following absorption, the chemicals in this group are expected to be distributed to a variety of organs, including the kidney, bone, liver, brain, heart, lung, and spleen (MAK 2006). Tellurium can freely cross the blood brain barrier and the placenta, as well as be transferred to the offspring via breast milk (ECHA 2020; Jackson et al. 1989).

The proposed metabolism of tellurium and its inorganic compounds is based on metabolism of excessive doses of selenium. Based on this finding tellurium is thought to be reduced in the liver from tellurate → tellurite → telluride (Te^{2-}), and then stepwise methylated to mono-, di-, or trimethyltellurium. Following absorption, TeO_2 is transformed systemically to tellurite (TeO_3^{2-}) and then to telluride (Te^{2-}) (ECHA 2020; Gad & Pham 2014; Ogra 2009).

The methylated species are the most abundant forms of tellerium in the human body. In rats, dimethyltellurium can bind to haemoglobin and accumulate in erythrocytes. In humans, 90% body burden of tellerium was in the bone (femur) (Government of Canada 2022; MAK 2006; Vavrova et al. 2021).

Tellurium is predominantly excreted in the urine after parenteral administration, and in the faeces after oral ingestion in rats. Elimination of tellurium can be slow ($t_{1/2}$ ~ 20 days in kidney to >200 days in bone) and biphasic or multiphasic, dependent on the mode of administration. Tellurite has slower excretion rate and longer retention than tellurate in humans. Small amounts of absorbed tellurium are eliminated as volatile dimethyltellurium ($(CH_3)_2Te$), causing a characteristic garlic like odour of the breath and sweat (ECHA 2020; Government of Canada 2022; Kron et al. 1991; MAK 2006).

The characteristic garlic like odour (indicative of formation of dimethyl tellurium) has been identified after exposure to different forms of tellurium including both soluble (tellurite salts) and insoluble forms (Te and TeO_2) (see **Acute toxicity**). This supports the presence of the proposed common metabolic pathway for chemicals listed in this evaluation.

Acute toxicity

Oral

Based on the available animal data, tellurium and tellurium oxide (insoluble compounds) are expected to have low acute oral toxicity with median lethal doses (LD50s) >2000 mg/kg bw.

In various OECD TG 401 studies in rat and mouse, an LD50 of >5000 mg/kg bw for tellurium and >2000 mg/kg bw for tellurium oxide were reported. The only clinical signs of toxicity were sluggishness (1 hour after dosing) and diarrhoea in males (MAK 2006; REACH n.d.-a; REACH n.d.-b).

Based on the limited available data including reported LD50 values for sodium salts of telluric acid and tellurous acid of 12–56 mg/kg bw in mice and rabbits, telluric acid, tellurous acid and potassium tellurite may have high acute toxicity (MAK 2006); however, there is insufficient information to warrant hazard classification.

Tellurium tetrachloride may cause local corrosive effects at point of contact due to its reaction with water to form TeO_2 and HCl.

Dermal

No data are available.

Inhalation

Based on the limited available information, inhalation of tellurium is not lethal up to concentrations of 2.42 mg/L. Higher concentrations have not been tested in animals. No lethality has been observed in humans exposed to tellurium via the inhalation route.

In an acute inhalation toxicity study (OECD TG 403), Wistar rats (5/sex) were exposed nose only to tellurium powder at 2.42 mg/L/4h (aerosol). The aerodynamic diameter of 72% of the particles was 1.8–4.2 µm. No mortality was reported. Black heads, half closed eyes and hunched postures were observed immediately after exposure. Alopecia was seen in all treated female rats during a 14 day observation period. All animals showed large body weight reduction (the magnitude of bodyweight reduction was not reported) during week one post exposure. Although some weight gains were observed in female rats at the end of the observation period. At autopsy, greyish, brown or black discolouration of the lungs was observed in all animals, and a grey thymus in 4/5 female rats (MAK 2006; REACH n.d.-a).

Observation in humans

Oral

Observations in humans suggests that tellurium compounds can cause unpleasant effects after a single exposure even at low concentrations, including headache, nausea, vomiting, cyanosis, weight loss, alopecia, and garlic like odour (Gad & Pham 2014; MAK 2006).

Tellurium (Te)

Weight loss, fatigue, nausea, vomiting, metallic taste in the mouth, and garlic like odour in the breath, sweat and excrement were reported in a 37 year old woman who tasted a piece of meat with Te 0.8–1.0 mg/kg meat. Biological concentrations were 27.6 µg/L in serum, and 6.7 µg/L in 24h urine. Fever developed on day 2 and resolved on day 5, together with previously occurring nausea and vomiting. Hair loss was observed 2 weeks after exposure. The garlic like odour became milder but lasted for 8 months (Muller et al. 1989; MAK 2006).

Vomiting, black discolouration of the oral mucosa, and garlic like odour in the breath were observed in 2 children who ingested metal-oxidizing solutions containing substantial concentrations of tellurium. Both patients recovered without serious sequelae (Yarema & Curry 2005).

Tellurium oxide (TeO_2)

A garlic like odour in the breath and excrement occurred within 75 minutes and lasted for 30 hours following ingestion of 0.5 µg TeO_2. After intake of 15 mg TeO_2, the odour lasted for 8 months (Reisert 1884; MAK 2006).

Potassium tellurite (K_2TeO_3)

There are 2 reported cases of accidentally swallowing 1% aqueous K_2TeO_3 solution in a bacteriological laboratory. The first patient developed repulsive taste in the mouth and garlic like odour in exhaled air, sweat, urine, and faeces. The black grey discolouration of lips,

teeth, tongue, buccal and laryngeal mucosa subsided after 10 days. These effects reoccurred again at intervals, particularly in the morning, for further 5.5 months. The patient also suffered from severe headaches, nausea, vomiting, spasmic body pains, profuse perspiration, drowsiness, transitory fever, exhaustion, and weight loss. The other patient, despite immediately having spat out the rest of the solution, the patient complained of mild headaches and fatigue for few days, and garlic like odour in the breath for 14 days, plus subsequent gastric irritation (MAK 2006).

A garlic like odour was observed in healthy volunteers (65–76 kg) after each one received a single oral dose of 15 µg of the sodium salt of tellurous acid. No toxic symptoms were observed.

Telluric acid

No data are available for telluric acid. A single oral dose of 57 µg of the sodium salt of telluric acid did not produce toxic symptoms or a garlic like odour in healthy human volunteers (Kron et al. 1991; MAK 2006).

Inhalation

Transitory headaches, pain in the upper abdomen for few days and garlic like odour and taste in the mouth for 14 days were reported in 3 employees after a 10 minute inhalation of tellurium fume (tellurium and tellurium oxide) (MAK 2006).

Corrosion/Irritation

Tellurium tetrachloride ($TeCl_4$) can cause direct local corrosive or irritating effects on the skin, eyes, and respiratory tract due to its reaction with water and formation of hydrochloric acid.

Skin irritation

Based on the available animal data, tellurium and tellurium oxide are not expected to be irritating to the skin. There are no data available for the other chemicals.

In two separate GLP compliant in vitro skin irritation studies in accordance with OECD TG 439 (in vitro reconstructed human epidermis) using EPISKIN model, tellurium and tellurium oxide were considered to be non-irritant. Mean relative viability values were 103% and 76%, respectively after an exposure period of 15 minutes, followed by an observation period of 42 hours (REACH n.d.-a; REACH n.d.-b).

Eye irritation

Based on the available data, tellurium and tellurium oxide are not irritating to the eyes. There are no data available for the other chemicals in this evaluation.

In a GLP compliant in vitro eye irritation study conducted in accordance with the OECD TG 438 (isolated chicken eyes model), tellurium produced a mean corneal swelling score of 0 (ICE class I), a maximum mean opacity score of 0 (ICE class I) and a mean fluorescein retention score at 30 minutes post treatment of 0 (ICE class I). Based on the prediction model criteria, tellurium is not considered to require classification for eye irritation and serious eye damage (REACH n.d.-a).

In a GLP compliant in vitro eye irritation study conducted in accordance with the OECD TG 438 (isolated chicken eyes model), tellurium oxide produced a mean corneal swelling score of 0 (ICE class I), a maximum mean opacity score of 0 (ICE class I) and a mean fluorescein retention score at 30 minutes post treatment of 0.5 (ICE class I). Based on the prediction model criteria, tellurium oxide is not considered to require classification for eye irritation and serious eye damage (REACH n.d.-b).

Respiratory irritation

Inhalation of tellurium tetrachloride ($TeCl_4$) may cause respiratory tract irritation as it decomposes immediately by reacting with air moisture to form tellurium oxide (TeO_2) and hydrochloric acid (HCl) (PubChem 2024).

Sensitisation

Skin sensitisation

Based on the limited available data for tellurium and tellurium oxide, it is not possible to determine whether chemicals in this evaluation are skin sensitisers.

In 2 local lymph node assay (LLNA) conducted in accordance with OECD TG 429, 4 female CBA/J Rj/dose mice received topical applications of either tellurium or tellurium oxide suspended in propylene glycol. Stimulation indices (SI) were 3.8, 3.2, 3.2 for tellurium and 3.9, 2.0, 3.7 for tellurium oxide at concentrations of 25%, 50%, 100%, (REACH n.d.-a; REACH n.d.-b). All SI were close to 3 with no clear dose response. Furthermore, the LLNA assay may be less accurate for metals; therefore, these results can be considered inconclusive (EU 2002).

Repeat dose toxicity

Based on the available data, primary targets for tellurium and tellurium oxide after oral exposure are the nervous system and the liver. Neuropathy was observed after and administration of high oral doses (see **Neurotoxicity section**). Liver effects occurred at low doses; however, the effects were reversible and there was no evidence of severe liver dysfunction. Considering these effects occurred only at high doses or were reversible, hazard classification is not warranted. However, the doses at which systemic toxicity effects may occur could vary between compounds due to differences in the oxidation state and bioavailability.

Only one study assessing repeat dose inhalation toxicity with limited experimental details is available. Mortality was high (50%) in animals exposed to 0.1 mg/L, 2 hours per day for 13–15 weeks. Haematological effects and effects that may be indicative of liver damage were reported. However, the magnitude of these effects was not described and no statistical analyses was reported. While this study indicates that repeated inhalation of tellurium may cause serious effects including paralysis and necrosis, there is insufficient information to warrant hazard classification.

Oral

Tellurium (Te)

In a non-guideline study, adult rats (strain and numbers of rats were not specified) were fed Te 1.25% in the diet (approximately 625 mg/kg bw/day) for 30 days. Mortality was observed

in the study (number of animals and dose not reported). Garlic like odour, blue-grey discolouration of the skin, restricted movement and substantial weight loss (50%) were observed. Myelin changes was also observed in the study (see **Neurotoxicity** section) (MAK 2006).

Tellurium oxide (TeO_2)

In a GLP complaint 13 week study similar to OECD TG 408, Wistar Hannover rats (10-15/sex/dose) were administered tellurium oxide in carboxymethyl cellulose (CWC) via gavage at 0, 10, 30 or 100 mg/kg bw/day daily 13 for consecutive weeks.

There was no treatment related mortality in the study. Clinical signs of toxicity included piloerection and hair loss in a few animals (mainly at the high dose levels). These signs were no longer observed in male animals during the recovery period. No signs of neurotoxicity were observed. Statistically significant decreases in body weight (6–10%) were observed in high and mid dose female rats from day 29 and in high dose male rats from day 85. These body weight decreases continued throughout the recovery periods in both sexes. Statistically significant reductions in absolute body weight gain were more pronounced in females (12–31% at high, mid, and low doses from days 15, 29 and 36, respectively) than in males (12–16% at high dose from day 15) to the end of the study (day 92). Food consumption was slightly reduced in females (-6% to -15%), but not in males. These decreases were no longer observed during the recovery period.

Some fluctuations in haematological parameters were reported; however, these were not dose dependent and were not consistent between males and females. Albumin levels were reduced in all dosed females (8–10%). Dose related fatty liver changes (steatosis) such as micro and macro vesicular vacuolation were seen in both sexes of the rat at ≥10 mg/kg bw/day, with an increased severity in high dose female rats. Given the high dose recovery animals did not show any relevant histological changes when compared with controls. The no observed adverse effect level (NOAEL) was considered to be 100 mg/kg/day in this study (REACH n.d.-b).

In a 28 day study (OECD TG 407; GLP), Wistar rats (5/sex/dose) were administered tellurium oxide (in 1% aqueous methylcellulose) via gavage daily at 0, 25, 120, 600 mg/kg bw/day. At 600 mg/kg bw/day, one female rat showed hunched posture, piloerection, moderately decreased activity and marked body weight loss prior to death on day 24. Similar clinical signs and reductions in body weight, body weight gain (males 66%; females 90%), platelet count, thymus weight; and increases in liver and spleen weights were observed in both sexes, together with black/grey discolouration or foci in various organs, and histological changes in the liver thymus and vagina. At 120 mg/kg bw/day, clinical signs (including transient hind limb weakness in males) and reductions in body weight gain (males 35%; female 14%) were reported, as well as mild lymphoid atrophy, and mild increased apoptosis of lymphocytes in the thymus of one female. At 25 mg/kg bw/day, reductions in body weight and body weight gain (only males 21%) were observed. Therefore, the lowest observed adverse effect level (LOAEL) was 25 mg/kg bw/day for male rats, while the NOAEL was 25 mg/kg bw/day for female rats (REACH n.d.-b).

In a non-guideline 128 day study, rats were fed tellurium oxide in food at doses up to 150 mg/kg bw/day. Mortality was reported after 26 days in high dose animals with necrotic liver and kidney tubules. Other signs of toxicity included a dose dependent reduction in growth, decreased food intake, oedema and inflammation of the toes, paralysis of the hindlimbs, alopecia, and garlic like odour in the breath, urine and inner organs (MAK 2006).

Dermal

No data are available.

Inhalation

In a 13–15 week repeated dose inhalation study, rats were exposed to aerosols containing 0.01 to 0.1 mg/L, elemental tellurium or tellurium oxide for 2 hours per day. Mortality occurred in 50% of animals at 0.1 mg/L. Weight loss, somnolence, respiratory irritation, loss of fur, and strong garlic like odour from exhaled air and all organs were observed. Haemolysis with decreased erythrocytes and haemoglobin content, decreased albumin, increased bilirubin in the urine, and increased serum β and α2-globulins were reported. No other details were available (MAK 2006).

Observation in humans

After 22 months exposure to tellurium fume (tellurium and tellurium oxide), 5/57 workers of an iron foundry with concentrations of 0.1–0.29 mg/m^3 and one with 0.74 mg/m^3 experienced fatigue, loss of appetite, metallic taste, dryness in the mouth, and garlic like odour in the breath, sweat and urine. Two of the 62 employees who worked closely to the tellurium source also described nausea (Gad & Pham 2014; MAK 2006).

Five cases of acute pulmonary oedema associated with inhalation of tellurium were reported under a voluntary scheme for the surveillance of work related and occupational respiratory disease (SWORD) in United Kingdom 1989 (Meredith et al. 1991).

Genotoxicity

No in vivo data are available. Based on the guideline in vitro studies (despite some inconsistencies in reporting cytotoxicity and precipitation), tellurium and tellurium oxide are not expected to have genotoxic potential.

Genotoxicity data available for other chemicals in this group are insufficient for evaluation.

In vitro

Tellurium

For tellurium, cytotoxicity was reported in *Salmonella typhimurium* TA1535, TA98 strains at 5000 µg/plate with metabolic activation, and in TA1535, TA1537, TA100 strains at 50, 158.1 µg/plate without metabolic activation (No information was provided at higher concentrations of 500–5000 µg/plate). Cytotoxicity was also reported in *Escherichia coli* WP2 uvrA at 5000 µg/plate with metabolic activation, and at ≥500 µg/plate without metabolic activation. No information for precipitation was available (ECHA 2020; REACH n.d.-b).

Tellurium oxide

Negative results were reported in bacterial reverse mutation tests (OECD TG 471, GLP) in *S. typhimurium* TA1535, TA1537, TA98, TA100, and *E. coli* WP2 uvrA, with or without metabolic activation.

No clear mutagenic or clastogenic effects were reported in (REACH n.d.-a; REACH n.d.-b):

- Cytotoxicity was reported in all tested strains at ≥500 µg/plate with and without metabolic activation (ECHA 2020; REACH n.d.-b).
- A mammalian cell gene mutation test (OECD TG 476, GLP) in L5178Y TK+/- mouse lymphoma cells, with and without metabolic activation.
  In Assay 1, test concentrations for a 3 hour exposure period were ≤10 µg/mL and ≤20 µg/mL, with and without metabolic activation, respectively. In Assay 2, test concentrations for evaluation were reduced to ≤5 µg/mL for 3 hour exposure with metabolic activation, and ≤8 µg/mL for 24 hour exposure without metabolic activation. Cytotoxicity was observed at ≥15 µg/mL (Assay 1) and ≥7.5 µg/mL (Assay 2). No concentrations were specified for the precipitation at the end of treatment.
- A mammalian chromosome aberration test (OECD TG 473, GLP) in Chinese hamster lung fibroblasts (V79), with and without metabolic activation.
  Cytotoxicity was reported at ≥100 µg/mL and ≥75 µg/mL (Assay 1) and at ≥75 µg/mL and ≥10 µg/mL (Assay 2), with and without metabolic activation, respectively. Precipitation was observed at ≥100 µg/mL.

Potassium tellurite (K_2TeO_3)

No significant genotoxicity was reported for potassium tellurite in 2 human cell lines (HepG2 liver and LS-174T colon cells) at up to 100 µM without metabolic activation, using a non-guideline high throughput screening (γH2AX assay). Cytotoxicity was observed at 100–1000 µM (Kopp et al. 2018; REACH n.d.-a).

Sodium tellurite (Na_2TeO_3) and sodium salts of telluric acid ($Na_2H_4TeO_6$), tellurium tetrachloride ($TeCl_4$)

Positive mutagenic effects (DNA damage) were noted for sodium tellurite (0.01 M), sodium salts of telluric acid (0.01 M), and tellurium tetrachloride (0.001 M) in 2 strains of *Bacillus subtilis* bacteria (H17 rec+ and M45 rec-) in a non-guideline rec assay (Kanematsu et al. 1980).

Telluric acid (H_2TeO_4)

A weak increase of micronuclei frequency was found for telluric acid in human lymphocytes in a non-guideline micronucleus assay (Migliore et al. 1999).

Carcinogenicity

No reliable data are available.

Reproductive and developmental toxicity

Based on the available data the chemicals are expected to cause specific adverse effects on fertility and development warranting classification and may cause effects in offspring via lactation.

In a reproductive screening study with tellurium a reduction in gestation index and a prolonged gestation period were observed at 120 mg/kg bw/day in the absence of severe general toxicity. At the high dose (600 mg/kg bw/day) effects on fertility and atrophy of female reproductive organs was observed in the presence of pronounced general toxicity. Due to the large dose spacing and the omission of histological analysis of reproductive organs of the females in the 120 mg/kg bw/day dose group, there is no information on possible effects on reproductive organs at doses without severe general toxicity.

Severe developmental effects (hydrocephalus and/or foetal and pup mortality) were consistently seen in the screening study and all available pre-natal developmental toxicity studies with tellurium and tellurium oxide, even at the lowest dose and without any maternal toxicity. Tellurium caused hydrocephalus in 2 animal species (rats and rabbits). In addition, according to the ECHA (2020) review, the teratogenic effects (primarily hydrocephalus) of tellurium were particularly observed if the exposure of dams were within the critical window of development (gestation days (GD 9–15)). Dose dependent reductions in body weights were reported in females from GD 7 at the mid and high dose (8 and14%) until the end of the study (18 and 30%). Bodyweight reductions at the low dose were reported to be within the historical control range.

There are no data available for the other tellurium compounds in this group. Based on the proposed metabolic pathway and formation of common metabolites (see **Toxicokinetic section**) read across data from tellurium and TeO_2 for effects on fertility and development is considered warranted. Overall, hazard classification in category 1B with hazard statement H360Df (May damage the unborn child. Suspected of damaging fertility) is considered appropriate.

Transfer from dams via lactation to their offspring has been demonstrated for tellurium compounds with low solubility. Overall, there is evidence of adverse effects (neuropathies) in offspring due to transfer of compounds in milk, warranting classification. Although similar effects could occur with tellurium compounds with higher solubilities, in the absence of toxicokinetic data and uncertainty as to the nature of the tellurium species that is transferred by lactation (i.e. parent chemical or metabolite) it is not considered appropriate to read across this classification from the low solubility chemicals (tellurium, tellurium oxide and tellurous acid) to the other chemicals in this evaluation.

Effects on reproductive organs: (only high dose animals were examined histologically)

- At necropsy, absolute and relative weights of seminal vesicles (without histological findings), and absolute weight of vagina (47% below control, probably relating to ovary atrophy) were lowered in high dose animals.
- Atrophy of ovary, uterus and vagina (in 3/5 dead females), a moderate vacuolation of corpora lutea and pigment deposits in the right ovary (1/5) were noted. The effects on female reproductive organs were not considered secondary to the observed general toxicity.

In a reproductive/developmental toxicity screening study (OECD TG 421, GLP), Wistar rats (12/sex/dose) were treated by gavage daily with tellurium oxide at 0, 25, 120 or 600 mg/kg bw/day. Males were dosed for 28 days (14 days pre-mating and 14 days post-mating), and females were dosed 14 days pre-mating, up to 14 days of mating, throughout gestation and up to day 4 of lactation (ECHA 2020; REACH n.d.-a; REACH n.d.-b).

General toxicity:

- Mortality was high among females receiving 600 mg/kg bw/day (approximately 40%). No mortality was observed at the other doses.
- The terminal body weights in males of MD and HD were about 7% and 14% lower than the control group. Food consumption was reduced in both males and females.
- At 600 mg/kg bw/day, female rats exhibited decreased activity, hunched back, lethargy, piloerection, dark faeces, excretion of red liquid from the mouth vulva and laboured respiration.

- Female animals had hepatocellular vacuolation or liver necrosis. The severity and incidence of macroscopic and microscopical effects in females were dose dependent. Less severe liver effects were observed in mid dose females.
- Males showed accumulation of pigmented macrophages in the mesenteric lymph node (mid and high dose).
- The systemic NOAELs were 25 mg/kg bw/day for both male and female rats.
- In a 28 day study (OECD TG 407; GLP) (see **Repeated Dose Toxicity** section), histological changes such as moderate diffuse epithelial atrophy of the vagina were noted in 2/4 female Wistar rats at 600 mg/kg bw/day (REACH n.d.-b).

Effects on sexual function and fertility:

- At the high dose, mating index was decreased to 73% and fertility index to 63%; oestrus cycle was abnormal; and 4/6 females were non-pregnant with reduced or no corpora lutea or implantation sites.
- At mid and high dose, gestation indices decreased to 67% and 0%, respectively. The gestation period was increased by 0.7 and 1.3 days, respectively.
- The reproductive NOAEL was 25 mg/kg bw/day for female rats, based on reduced gestation index and prolonged gestation at mid dose, and reduced fertility index at high dose.

Developmental toxicity

In a GLP compliant prenatal developmental toxicity study (similar to OECD TG 414), Crl female Sprague Dawley rats (32–33/dose) were fed a diet containing tellurium at 0, 30, 300, 3000 or 15000 ppm (equivalent to 0, 1.9, 18, 173 or 579.4 mg/kg bw/day) on GD 6–15.

All dams survived the study. Maternal body weight and food consumption during gestation were significantly decreased in a concentration dependent manner at dose ≥18 mg/kg bw/day. The oestrous cycle was not affected at any dose. There were no effects on the incidence of pregnancy, average numbers of corpora lutea, implantations or resorptions for caesarean-delivered foetuses at GD 20. The average number of live and dead foetuses or litter size is not affected. However, the number of pups surviving 7 days reduced at highest dose. Skeletal and soft tissue malformations, primarily hydrocephalus were observed in two highest dose groups (173 and 579.4 mg/kg bw/day. Other malformations at this dose included kinked and /or stubbed tails, rotation of a hind limb or foot, a malformed retina, mal-positioned manubrium and clavicles, short radius, ulna and/or femur, wavy ribs, and a thickened or split rib. The developmental NOAEL was 18 mg/kg bw/day, based on skeletal maturational delays and soft tissue malformations (primarily hydrocephalus) at ≥173 mg/kg bw/day, and reduced pup survival on Post Natal Day (PND) 7 at 579.4 mg/kg bw/day (ECHA 2020 abstract; REACH n.d.-a).

In a prenatal developmental toxicity study (similar to OECD TG 414), female Wistar rats (10/dose) were exposed subcutaneously to tellurium oxide at 0, 10, 100, 500, 1000 µmol/kg bw/day (or 0, 1.6, 16, 80, 160 mg/kg bw/day) on GD 15–19. The maternal NOAEL was 16 mg/kg bw/day, based on decreased weight gain, centrilobular fatty liver and 40% mortality at higher doses. The developmental NOAEL was 1.6 mg/kg bw/day, based on 100% hydrocephalus and oedema at ≥16 mg/kg bw/day, together with exophthalmia, ocular haemorrhage, umbilical hernia, undescended testes, small kidneys, and increased foetal mortality (11–81% at ≥80 mg/kg bw/day) (ECHA 2020).

In a prenatal developmental toxicity study (similar to OECD TG 414), New Zealand White rabbits (17/dose) were fed a diet containing tellurium at 0, 17.5, 175, 1750, 5250 ppm (or 0, 0.7, 7, 70, 210 mg/kg bw/day) on GD 6–18. The maternal NOAEL was 7 mg/kg bw/day,

based on decreased food intake and body weight gain at higher doses. The developmental NOAEL was 70 mg/kg bw/day, based on decreased foetal body weight, increased skeletal delays and hydrocephalus at the highest dose (ECHA 2020; Johnson et al. 1988 abstract; REACH n.d.-a).

In a reproduction/developmental toxicity screening study (OECD TG 421, GLP) in Wistar rats described above the following effects on development of the offspring were observed:

- Pup mortality was increased in all dose groups including doses that did not affect dam survival.
- Survival index decreased from 75% on PND 4 (below the normal control) at low dose to 0% on PND 0 (i.e. no live pups) at high dose. Gross necropsy revealed absence of cranium or small brain (4/16), as well as whole body subcutaneous gelatinous material (16/16) in the high dose group.
- More male than female pups died on postnatal day (PND) 0–4 at mid dose.

The developmental LOAEL was 25 mg/kg bw/day, based on increased foetus/pup mortality at all doses.

In a non-guideline study, Long Evans rats (>100 dams) were fed a diet containing tellurium at 500, 1250, 2500 ppm (or 25, 62.5, 125 mg/kg bw/day) during pregnancy. High dose dams received normal diet 3–5 days before delivery. The maternal NOAEL was 125 mg/kg bw/day. The developmental LOAEL was 25 mg/kg bw/day based on small new-borns (none survived after a month) and hydrocephalus, 60%, 60–90%, and 100% in the low, mid, and high dose, respectively. All offspring died within the first month after birth. No further details are available (ECHA 2000; Garro & Pentschew 1964).

Many other prenatal developmental toxicity studies (see ECHA 2020 for review) consistently reported the teratogenic effects (primarily hydrocephalus) of tellurium within the critical window of development (GD 9–15), even with a single sufficiently high dose. However, they were not included in this evaluation as there was lack of information on maternal NOAELs or maternal effects, and they were not conducted according to guidelines.

Effects via lactation

In a non-guideline study, Wistar dams (2/dose) were fed a diet containing tellurium at 0, 1.25% (or 0, 625 mg/kg bw/day) during lactation (assuming PND 0–7). Neonatal rats (5/group/time point) were exposed to tellurium via mother's milk from birth until sacrifice at 7, 14, 21, and 28 days of age. Garlic like odour occurred within 2–3 days and greyish discolouration after 7 days of exposure in both dams and offspring, indicating the transfer of tellurium to the offspring via breast milk. The maternal NOAEL was 625 mg/kg bw/day given there were no other clinical effects. Pups at days 14, 21, and 28 showed lethargy, hind limb paralysis, incontinence, reduced weight gain and body weight. Light and electron microscopy revealed Schwann cell and myelin degeneration in the sciatic nerve (starting from PND 7) and hypomyelination of the optic nerves (from PND 14). Similar changes were also seen in the cervical spinal cords although less severe (ECHA 2020).

Nishimura et al. 2003 demonstrated that 2%, 3.9%, and 5% of a single intravenous dose of radiolabelled tellurous acid ($H_2{}^{123m}TeO_3$) administered to dams were transferred via lactation to their offspring on PND 1, 7, and 14, respectively. This finding provides additional evidence for transfer of tellurium via breast milk.

Neurotoxicity

High doses of tellurium can induce peripheral neuropathy (ECHA 2020; Gad & Pham 2014; MAK 2006; Vavrova et al. 2021).

In a non-guideline study weanling rats (6 litters, number of rats not provided) were exposed 625 mg/kg bw/day on postnatal days 20–27. The rat developed signs of neuropathy including demyelination of peripheral nerves. After 5 days of treatment there was a 25% sciatic nerve demyelination. The effects were reversible, 30 days after the end of treatment there were no signs of neuropathy (ECHA 2020).

In a study in adults, rats were fed a diet containing 1.25% (1250 ppm equivalent to 625 mg/kg bw) tellurium daily for 30 days. At the end of the treatment period the rats moved with difficulty but were not paralysed. Myelin abnormalities included myelin bubbling, segmental demyelination, and remyelination (Said and Ducket 1981 abstract).

In a mechanistic study it was demonstrated that tellurium can inhibit cholesterol synthesis in vivo. The mechanism of toxicity was demonstrated to involve tellurium inhibition of squalene epoxidase, leading to disruption of cholesterol synthesis and accumulation of squalene in the sciatic nerve. These may result in degeneration of myelinating Schwann cells in the peripheral nervous system and hence demyelination of axons. The severity of these effects increases with higher doses and longer exposure durations. Although the active tellurium species in vivo is unknown, data suggests that methylated tellurium species may be responsible (Harry et al. 1989 abstract; Goodrum 1998; Laden & Porter 2001; MAK 2006; Said & Duckett 1981 abstract; Toews et al. 1997; Wagner et al. 1995 abstract).

References

ACGIH (American Conference of Industrial Hygienists) (2018) TLVs and BELs with 7th Edition Documentation, CD-ROM, Single User Version. Copyright 2018.

ASEAN (ASEAN Cosmetic Directive) (2019), *Technical Documents on Tellurium and its compounds*, accessed January 2024.

Ashraf MW, Haider SI, Solangi AR and Memon AF (2022) 'Toxicity of tellurium and its compounds.' *Physical Sciences Reviews (Online)* 2022. doi: 10.1515/9783110735840-006.

Bouldin TW, Earnhardt TS, ND Goines and Goodrum J (1989) 'Temporal relationship of blood-nerve barrier breakdown to the metabolic and morphologic alterations of tellurium neuropathy.' *Neurotoxicology* 10(1):79-89. PMID: 2549475.

Canada Government (2022) *Cosmetic Ingredient Hotlist: Prohibited and Restricted Ingredients*, accessed 18 December 2023.

Chasteen TG, Fuentes DE, Tantalean JC and Vasquez CC (2009) 'Tellurite: history, oxidative stress, and molecular mechanisms of resistance.' *FEMS Microbiology Reviews* 33(4):820–832.

Chemwatch (n.d.) *Galleria Chemica*, Chemwatch website, accessed 5 July 2023.

DeLima Associates (n.d.) *Consumer Product Information Database*, DeLima Associates website, accessed 5 July 2023.

Department of Energy, The Emergency Management Issues Special Interest Group (EMI SIG) (n.d.), *Protective Action Criteria (PAC) database*, accessed 5 July 2023.

ECHA (European Chemicals Agency) (n.d.) *Substance Infocard for tellurium oxide (CAS No. 7446-07-3)*, accessed 5 July 2023.

ECHA (European Chemicals Agency) (2022) *Substance Regulatory Obligations on tellurium (CAS No. 13494-80-9)*, accessed 18 December 2023.

ECHA (European Chemicals Agency) (2020) 'Committee for Risk Assessment (RAC) - Annex 1 - Background document to the Opinion proposing harmonised classification and labelling at EU level of tellurium dioxide (CAS No. 7446-07-3)', accessed January 2024.

EU (European Union) (2000) 'Opinion on the murine local lymph node assay (LLNA) adopted by the SCCNFP during the 12th Plenary Meeting of 3 May 2000'. European Commission. Accessed 11 September 2024.

EU (European Union) (2023) *European Commission rules for CMR substances*. Accessed December 2023.

Garro F and Pentschew A (1964) 'Neonatal hydrocephalus in the offspring of rats fed during pregnancy non-toxic amounts of tellurium.' *European Archives of Psychiatry and Clinical Neuroscience* 206: 272–280. https://doi.org/10.1007/BF00940754

Gad SC and Pham T (2014) Tellurium. Encyclopedia of Toxicology (Third edition) 2014: pages 481-483. https://doi.org/10.1016/B978-0-12-386454-3.00936-2

Gerhardsson L (2015) Tellurium. Handbook of the Toxicology of Metals (Fourth Edition) Vol II: Specific Metals: pages 1217-1228. https://doi.org/10.1016/B978-0-444-59453-2.00054-8

Goodrum JF (1998) 'Role of organotellurium species in tellurium neuropathy'. *Neurochemical Research*, Vol 23, No. 10, pp 1313-1319.

Government of Canada (2022) *Cosmetic Ingredient Hotlist - List of Ingredients that are Restricted for Use in Cosmetic Products*, Government of Canada, accessed 25 November 2023.

Harry GJ, Goodrum JF, Bouldin TW, Wagner-Recio M, Toews AD and Morell P (1989) 'tellurium-induced neuropathy: metabolic alterations associated with demyelination and remyelination in rat sciatic nerve.' *Journal of Neurochemistry* March 52(3):938-45. https://doi.org/10.1111/j.1471-4159.1989.tb02545.x

Jackson KF, Hammang JP, Worth SF and Duncan ID (1989) 'Hypomyelination in the neonatal rat central and peripheral nervous systems following tellurium intoxication.' *Acta Neuropathologica*. 78(3):301-9. https://pubmed.ncbi.nlm.nih.gov/2763802/

Johnson EM, Christian MS, Hoberman AM, DeMarco CJ, Kilpper R and Mermelstein R (1988) 'Developmental toxicology investigation of tellurium.' *Fundamental and Applied Toxicology* Vol 11(4): pages 691-702.

Kanematsu N, Hara M and Kada T (1980) 'Rec assay and mutagenicity studies on metal compounds.' *Mutation Research/Genetic Toxicology.* Vol 77(2): pages 109-116. https://doi.org/10.1016/0165-1218(80)90127-5

Klevay LM (1976) 'Pharmacology and Toxicology of heavy metals: selenium,' *Pharmacology and therapeutics.* A. vol.1, pp. 211-222. https://doi.org/10.1016/0362-5478(76)90008-5

Knockaert G (2011) *Tellurium and Tellurium Compounds Ullmann's Encyclopedia of Industrial Chemistry* Vol. 35.

Kopp B, Zalko D and Audebert M (2018) 'Genotoxicity of 11 heavy metals detected as food contaminants in two human cell lines.' *Environ. Mol. Mutagen* 59(3), 202-210.

Kron T, Hansen Ch and Werner E (1991) 'Renal excretion of tellurium after peroral administration of tellurium in different forms to healthy human volunteers.' J*. Trace Elem. Electrolytes Health Dis.* Vol. 5, 1991: pp 239-244.

Laden BP and Porter TD (2001) 'Inhibition of human squalene monooxygenase by tellurium compounds: evidence of interaction with vicinal sulfhydryls.' *Journal of Lipid Research* vol 42, 2001: pp 235-240.

MAK (2006) The MAK-Collection for Occupational Health and Safety, Part I: MAK Value Documentations, Vol 22, DFG Deutsche Forschungsgemeinscha, Wiley-VCH Verlag GmbH & Co. KGaA, Weinheim.

Meredith SK, Taylor VM and McDonald JCM (1991) 'Occupational respiratory disease in the United Kingdom 1989: a report to the British Thoracic Society and the Society of Occupational Medicine by the SWORD project group.' *Br J Ind Med* 48: 292-298.

Merck & Co., Inc. (n.d.). Tellurium. In *Merck Index Online*. Retrieved 29 August 2024.

Migliore L, Cocchi L, Nesti C and Sabbioni E (1999) 'Micronuclei assay and FISH analysis in human lymphocytes treated with six metal salts.' *Environ Mol Mutagen* 34(4):279-84. doi: 10.1002/(sici)1098-2280(1999)34:4<279: aid-em8>3.0.co;2-7.

Mullner R, Zschiesche W, Steffen HM and Schaller KH (1989) 'Case Reports Tellurium-Intoxication.' *Klin Wochenschr* 67: 1152-1155.

Nishimura Y, Sahoo Sk, Kim H-S, Homma-Takeda S, Wtanabe Y and Inaba J (2003) 'Biokinectics of radiotellurium in rats' *Radiation Protection Dosimetry* Vol. 105, Nos 1–4, pp 285-590. DOI:10.1093/oxfordjournals.rpd.a006241.

NIOSH (National Institute for Occupational Safety and Health) (1994) NIOSH Pocket guide to chemical hazards –*Tellurium compounds (as Te),* CDC website, accessed 14 January 2024.

NZ EPA (New Zealand Environmental Protection Authority) (2019) *Hazardous substances,* NZ EPA website, accessed 16 August 2023.

NLM (National Library of Medicine) (n.d.) *ChemIDplus Advanced Database*, NLM website, accessed 5 July 2023.

Perez-D'Gregorio RE and Miller RK (1988) 'Teratogenicity of tellurium dioxide: prenatal assessment.' *Teratology Apr* 37(4):307-1. doi: 10.1002/tera.1420370404.

Personal Care Products Council (n.d.) *Cosmetic Ingredient Identification Database*, Personal Care Products Council website, accessed 5 July 2023.

REACH (Registration, Evaluation, Authorisation and Restriction of Chemicals) (n.d.-a) *Registered dossier for tellurium, CAS No. 13494-80-9,* European Chemicals Agency website, accessed 15 June 2023.

REACH (Registration, Evaluation, Authorisation and Restriction of Chemicals) (n.d.-b) European Chemicals Agency website, accessed 20 June 2023.

Reisert W (1884) 'The so-called bismuth breath.' *American Journal of Pharmacy* 56:177–180.

Said G and Duckett S (1981) 'Tellurium-induced myelinopathy in adult rats.' *Muscle & Nerve.* Vol 4(4): 319-325.

SPIN (Substances in Preparation in Nordic Countries) (n.d.) *SPIN Database*, SPIN website, accessed 20 June 2023.

SWA (Safe Work Australia) (n.d.) *Hazardous Chemical Information System*, SWA website, accessed 25 June 2023.

Toews AD, Roe EB, Goodrum JF, Bouldin TW, Weaver J, Goines ND and Morell P (1997) 'Tellurium causes dose-dependent coordinate down-regulation of myelin gene expression.' *Brain Res Mol Brain Res October* 3;49(1-2):113-9. https://doi.org/10.1016/S0169-328X(97)00132-0

UNECE (United Nations Economic Commission for Europe) (2017) *Globally Harmonized System of Classification and Labelling of Chemicals (GHS) Revised Edition*, UNECE, accessed 29 June 2023.

US EPA (United States Environmental Protection Agency) (n.d.) *CompTox Chemical Dashboard v2.3.0 Tellurium (CAS No. 13494-80-9),* United States Environmental Protection Agency website, accessed December 2023.

US EPA (United States Environmental Protection Agency) (2016) *Chemview (US CDR data)*, United States Environmental Protection Agency website, accessed 20 June 2023.

Vavrova S, Struharnanska E, Turan J and Stuchlik S (2021) ‘Tellurium: A rare element with influence on Prokaryotic and Eukaryotic Biological Systems.’ *Int. J. Mol. Sci. 22(11), 5924*. https://doi.org/10.3390/ijms22115924

Wagner-Recio M, Toews AD and Morell P (1991) ‘Tellurium blocks cholesterol synthesis by inhibiting squalene metabolism: preferential vulnerability to this metabolic block leads to peripheral nervous system demyelination.’ *J Neurochem* Dec 57(6):1891-901. https://doi.org/10.1111/j.1471-4159.1991.tb06400.x

Wagner M, Toews AD and Morell P (1995) ‘Tellurite specifically affects squalene epoxidase: investigations examining the mechanism of tellurium-induced neuropathy.’ *J. Neurochem* 64: 2169-2176. https://doi.org/10.1046/j.1471-4159.1995.64052169.x

Watanabe C, Suzuki T, Ohba T and Dejima Y (1990) ‘Transient hypothermia and hyperphagia induced by selenium and tellurium compounds in mice.’ *Toxicol Lett 50*: 319–326.

Willner J, Fornalczyk A, Jablonska-Czapla M, Grygoyc K and Rachwal M (2021) ‘Studies on the Content of Selected Technology Critical Elements (Germanium, Tellurium and Thallium) in Electronic waste.’ *Materials 14(13), 3722*. https://doi.org/10.3390/ma14133722

Yarema MC and Curry SC (2005) ‘Acute tellurium toxicity from ingestion of metal-oxidising solutions.’ *Pediatrics* 116(2): e319–21. https://doi.org/10.1542/peds.2005-0172

Zhu H, Fan L, Wang K, Liu H, Zhang J and Yan S (2023) ‘Progress in the synthesis and application of tellurium nanomaterials.’ *Nanomaterials* 13(14), 2057. https://doi.org/10.3390/nano13142057