Thiourea: Human health tier II assessment

01 September 2015

CAS Number: 62-56-6

Preface

This assessment was carried out by staff of the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) using the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework.

The IMAP framework addresses the human health and environmental impacts of previously unassessed industrial chemicals listed on the Australian Inventory of Chemical Substances (the Inventory).

The framework was developed with significant input from stakeholders and provides a more rapid, flexible and transparent approach for the assessment of chemicals listed on the Inventory.

Stage One of the implementation of this framework, which lasted four years from 1 July 2012, examined 3000 chemicals meeting characteristics identified by stakeholders as needing priority assessment. This included chemicals for which NICNAS already held exposure information, chemicals identified as a concern or for which regulatory action had been taken overseas, and chemicals detected in international studies analysing chemicals present in babies' umbilical cord blood.

Stage Two of IMAP began in July 2016. We are continuing to assess chemicals on the Inventory, including chemicals identified as a concern for which action has been taken overseas and chemicals that can be rapidly identified and assessed by using Stage One information. We are also continuing to publish information for chemicals on the Inventory that pose a low risk to human health or the environment or both. This work provides efficiencies and enables us to identify higher risk chemicals requiring assessment.

The IMAP framework is a science and risk-based model designed to align the assessment effort with the human health and environmental impacts of chemicals. It has three tiers of assessment, with the assessment effort increasing with each tier. The Tier I assessment is a high throughput approach using tabulated electronic data. The Tier II assessment is an evaluation of risk on a substance-by-substance or chemical category-by-category basis. Tier III assessments are conducted to address specific concerns that could not be resolved during the Tier II assessment.

These assessments are carried out by staff employed by the Australian Government Department of Health and the Australian Government Department of the Environment and Energy. The human health and environment risk assessments are conducted and published separately, using information available at the time, and may be undertaken at different tiers.

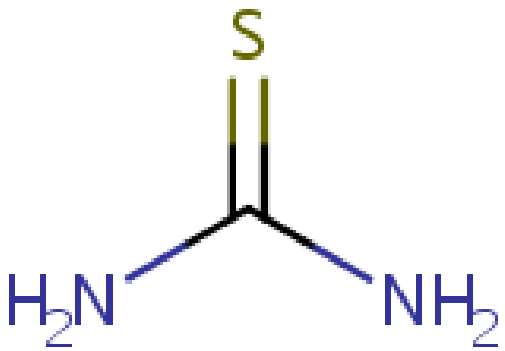
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

For more detail on this program please visit:www.nicnas.gov.au

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	thiocarbamide isothiourea 2-thiourea sulfourea
Structural Formula	
Molecular Formula	CH4N2S
Molecular Weight (g/mol)	76.12
Appearance and Odour (where available)	White, crystalline solid.
SMILES	C(N)(N)=S

Import, Manufacture and Use

Australian

The chemical has reported Australian industrial site-limited uses, including in explosives, as reported under previous mandatory and/or voluntary calls for information.

The total volume introduced into Australia, reported under previous mandatory and/or voluntary calls for information, was <1000 tonnes per annum.

International

The following international industrial uses have been identified through the European Union (EU) Registration, Evaluation, Authorization and Restriction of Chemicals (REACH) dossiers; Galleria Chemica; the Substances and Preparations in Nordic countries (SPIN) database; the United States (US) National Library of Medicine's (NLM) Hazardous Substances Data Bank (HSDB); the US NLM Household Products Database; and various international assessments (Environment Canada and Health Canada, 2008; WHO, 2003; IARC, 2001).

The chemical has reported domestic use, including in metal cleaning and silver jewellery cleaning products (also referred to as tarnish removers).

The chemical has reported commercial use, including:

- in metal engraving processes;
- in industrial cleaning agents;
- as a viscosity stabiliser for polymer solutions, such as in drilling muds;
- in animal hide adhesives;
- in reprography (as an agent in diazo paper); and
- as a corrosion inhibitor.

The chemical has reported site-limited use, including:

- as an accelerant in the production of rubber;
- as a mobility buffer in petroleum extraction processes;
- as an additive for slurry explosives;
- in the production of flame retardent resins; and
- as a catalyst in the synthesis of other chemicals.

Additionally, the chemical has historical non-industrial use, as a therapeutic agent (MAK, 2012).

Restrictions

Australian

This chemical is listed in the *Poisons standard—the Standard for the Uniform Scheduling of Medicines and Poisons* (SUSMP) in Schedule 6 (SUSMP, 2015).

Schedule 6:

'Thiourea and alkyl thioureas except:

(a) when separately specified in these Schedules; or

(b) for therapeutic use.'

Schedule 6 chemicals are described as 'Substances with a moderate potential for causing harm, the extent of which can be reduced through the use of distinctive packaging with strong warnings and safety directions on the label'. Schedule 6 chemicals are labelled with 'Poison' (SUSMP, 2015).

International

The chemical is listed on the following (Galleria Chemica, CosIng):

- EU Cosmetics Regulation 1223/2009 Annex II—List of substances prohibited in cosmetic products;
- New Zealand Cosmetic Products Group Standard—Schedule 4: Components cosmetic products must not contain;
- China List of banned substances in cosmetics;
- Association of Southeast Asian Nations (ASEAN) Cosmetic Directive Annex II—Part 1: List of substances which must not form part of the composition of cosmetic products; and
- Health Canada List of prohibited and restricted cosmetic ingredients (The Cosmetic Ingredient ‘Hotlist’).

Existing Work Health and Safety Controls

Hazard Classification

The chemical is classified as hazardous, with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia):

- Xn; R22 (acute toxicity)
- Xn; Carc. Cat 3; R40 (carcinogenicity)
- Xn; Repr. Cat 3; R63 (reproductive toxicity)

Exposure Standards

Australian

No specific exposure standards are available.

International

Available data indicate an exposure standard of 0.3 mg/m^3 exists in countries including Russia, Latvia and Bulgaria. However, the documentation is not available in English to allow for confirmation (Galleria Chemica).

Health Hazard Information

Toxicokinetics

In toxicokinetics studies in humans, the chemical has been shown to be absorbed rapidly following ingestion (of 28.57 or 100 mg/kg bw single oral dose), with blood levels peaking 30 minutes after exposure, and it is mostly eliminated from the blood 24 hours after exposure (WHO, 2003; IARC, 2001). The chemical is metabolised in the intestine (15 %) and other tissues and body fluids (30-50 %), with 30% being excreted unchanged as thiourea in urine. Complete elimination of the chemical from the body occurs within 48 hours after exposure.

Following dermal exposure of rabbits at 2000 mg/kg bw of the chemical, limited absorption was reported, with 0.1 and 4 % of the applied dose detected in urine, when applied as a solid or in aqueous solution, respectively (WHO, 2003). The chemical is reported to cross the placental barrier in rats and mice. High levels of the chemical are most commonly detected in the thyroid (IARC, 2001).

Acute Toxicity

Oral

The chemical is classified as hazardous with the risk phrase ‘Harmful if swallowed’ (Xn; R22) in the HSIS (Safe Work Australia). The available data (a median lethal dose—LD50—range of 125–1930 mg/kg bw in rats) support this classification (WHO, 2003).

Dermal

The chemical has low acute toxicity based on results from animal tests following dermal exposure. The dermal LD50 in rabbits is >2800 mg/kg bw (WHO, 2003).

Inhalation

The chemical is not expected to be acutely toxic based on animal tests following inhalation exposure, although available data are not conclusive. The median lethal concentration (LC50) was reported to be >195 mg/m^3 in rats, following four-hour inhalation exposure to a 10 % solution of the chemical (Environment Canada and Health Canada, 2008; WHO, 2003).

Corrosion / Irritation

Skin Irritation

The chemical is reported to slightly irritate skin in animal studies.

Twelve New Zealand White rabbits were exposed to 0.5 mg of the chemical for 24 hours under occlusive conditions, on abraded or intact skin (REACH). Slight to moderate erythema and oedema were observed in several animals 24 hours after the initial exposure. However, these effects were reported as fully reversible by the end of the study observation period (72 hours). While one rabbit showed signs of scaliness at the end of the 72 hour observation period, the effect observed in one animal is not sufficient to warrant hazard classification.

In another study, 0.5 mg of the chemical did not produce any skin irritation effects in rabbits after a four hour exposure period (WHO, 2003). No further study details are provided.

Eye Irritation

The chemical may cause slight eye irritation based on studies in animals.

In an eye irritation study in Himalayan rabbits, 100 mg of the chemical was instilled into the conjunctival sac of the left eye of 6 male animals. The right eye served as a control. The eyelids were kept closed for one second after application and eyes were not rinsed during the 72 hour observation period. Observations were made at one, 24 and 72 hours after exposure. Limited irritation scoring details are reported. However, it is noted that while the chemical is not considered to be irritating to the eye, a mean irritation score for the conjunctivae of 1-2 was reported for swelling and redness (REACH).

In another study, a single application of a 10 % (w/w) aqueous solution of the chemical to the eye was tolerated without reaction; no further details are provided (WHO, 2003).

Sensitisation

Skin Sensitisation

The chemical was not found to induce dermal sensitisation in a test using 20 male Pirbright white guinea pigs (REACH; WHO, 2003). Animals were exposed to the chemical intradermally, at concentrations ranging from

0.5–1.0 % during the induction phase of the study, then challenged percutaneously with a 20 % solution of the chemical, seven days after the initial induction phase. No reactions were reported in any of the animals tested.

Observation in humans

Cases of contact dermatitis have been reported in individuals following exposure to the chemical during formulation processes, or to products containing the chemical (diazo copy paper and silver polish; details of other ingredients contained in the products are not provided) (Environment Canada and Health Canada, 2008; WHO, 2003). Allergic reactions were observed in 1.2 % (5/423) of patients in an allergic patch test (WHO, 2003).

Repeated Dose Toxicity

Oral

Repeated oral dose toxicity studies using the chemical have been conducted in rats, mice and lambs. Duration of exposure to the chemical across the studies ranged from 10 days to three years, with dose levels of the chemical ranging from 0.0028 to 1000 mg/kg bw/day (REACH; Environment Canada and Health Canada, 2008; WHO, 2003; IARC, 2001). Regardless of the route of exposure (in drinking water, in the diet, or by oral gavage), adverse effects relating to the thyroid, specifically enlargement or hyperplasia of the thyroid, were consistently reported across the studies. Other reported effects included reduced body weight, enlargement and increased weight of the pituitary, and reduced levels of the thyroid hormone, thyroxine (T4). The occurrence of tumours (neoplastic effects), as reported in some studies, is discussed in the following sections (see **Carcinogenicity**).

In regards to non-neoplastic effects, a lowest observed adverse effect level (LOAEL) of 27.5 mg/kg bw/day (based on reduced body weight and enlargement of the thyroid) was reported for rats that were exposed to the chemical in drinking water, daily for up to three years (WHO, 2003). A no observed adverse effect level (NOAEL) of

6.88 mg/kg bw/day was reported for this study. In another study, a lowest observed effect level (LOEL) of

70 mg/kg bw/day was reported for rats exposed to the chemical daily, for 10 days based on reduced levels of iodine in the thyroid (Environment Canada and Health Canada, 2008).

Dermal

No data are available.

Inhalation

No data are available.

Observation in humans

The chemical is reported to have historical use as a therapeutic drug, for the treatment of hyperthyroidism (excessive thyroid activity) (WHO, 2003; MAK, 2012). Treatment with the chemical inhibits thyroid function, as measured by reduced levels of T4 and T3 (triiodothyronine) thyroid hormones. This effect has been observed in animals exposed to the chemical, as discussed in the previous section. The thyroid responds to reduced levels of T3 and T4, by promoting production of thyroid-stimulating hormone (TSH), secreted by the pituitary.

In a study of 12 patients with hyperthyroidism, treatment with the chemical (together with an iodine solution) at <0.2 mg/kg bw/day (based on a 70 kg adult), daily for 10 to 12 days, did not lead to measureable depression of thyroid activity, while a dose of 1.0 mg/kg bw/day was reported to result in a remission of hyperthyroidism (WHO, 2003; IARC, 2001).

A few cases of occupational exposure to the chemical have been reported (WHO, 2003; IARC, 2001; MAK, 2012). In a factory manufacturing the chemical, reported concentrations of the chemical in air were 0.6 to 12 mg/m^3. Of the 45 workers who were examined, 54.5 % were over 40 years of age, and average duration of exposure to the chemcial was 9.5 ± 1.1 years, with 73% being exposed for at least five years. Reduced thyroid function (significantly lower levels of T3 and T4) was reported in these workers, as compared to a control group of 20 people, with thyroid hyperplasia observed in 17/45 workers.

Genotoxicity

While there are many in vitro genotoxicity studies for this chemical, there are very limited data from in vivo studies. The chemical did not induce point mutations in bacteria, but did induce in vitro chromosomal recombination in yeast cells, with mixed results in mammalian cells (Environment Canada and Health Canada, 2008; WHO, 2003; IARC, 2001). In other in vitro assays, the chemical did induce cell transformation, DNA synthesis inhibition and micronucleus formation in various mammalian cell studies. Results in *Drosophila melanogaster* (fruit fly) were mixed. However, based on negative results seen in the only mammalian in vivo study available (micronucleus test in rats and its lack of potential to cause point mutations), the chemical is considered unlikely to be a genotoxic carcinogen.

In vitro

The chemical did not induce gene mutations in the bacteria *Escherichia coli* or *Salmonella typhimuruim* (strains TA97, TA98, TA100, TA1535, TA1537 and TA1538), with or without metabolic activation (Environment Canada and Health Canada, 2008; IARC, 2001). In yeast cell studies in *Saccharomyces cerevisiae*, intrachromosomal recombination (both with or without metabolic activation) and petite mutations (metabolic activation not tested) were induced following exposure to the chemical.

In in vitro mammalian cell tests, the chemical induced DNA single-strand breaks in primary rat hepatocytes at test concentrations of 2280 µg/mL (WHO, 2003; IARC, 2001). The chemical did not induce unscheduled DNA synthesis in rat hepatocytes at test concentrations of up to 10,000 µg/mL. Mixed (mostly negative) results were obtained in gene mutation tests using Chinese hamster ovary (CHO) cells and mouse lymphoma cells (Environment Canada and Health Canada, 2008; IARC, 2001).

In two separate studies in CHO cells, the chemical did not induce recombination or sister chromatid exchanges at concentrations up to 25 and 7,600 µg/mL, respectively (IARC, 2001). The chemical induced micronucleus formation in Syrian hamster embryo (SHE) cells and CHO cells. It also induced cell transformation in SHE cells, modified rat embryo cells and modified mouse embryo cells. In one study using human fibroblast cells, the chemical was reported to inhibit DNA synthesis at concentrations of 60 mmol/L (Environment Canada and Health Canada, 2008; WHO, 2003).

In vivo

Somatic mutation and recombination tests in *Drosophila melanogaster* gave mixed (positive, negative and inconclusive) results (IARC, 2001); positive somatic gene mutations were observed using the zeste-white assay, while equivocal results were observed using the white-ivory (somatic gene mutation) assay at the same concentrations (WHO, 2003).

Oral exposure to the chemical did not induce micronucleus formation in rats following ingestion of two successive doses of 350 mg/kg bw, 24 hours apart. No clinical signs of toxicity or cytotoxic effects were observed (WHO, 2003).

Carcinogenicity

The chemical is classified as hazardous (Category 3 carcinogenic substance) with the risk phrase ‘Limited evidence of carcinogenic effect’ (Xn; R40) in the HSIS (Safe Work Australia). The available data support this classification.

The International Agency for Research on Cancer (IARC) has concluded that the chemical is not classifiable as to its carcinogenicity to humans (Group 3), based on inadequate evidence for carcinogenicity in humans (IARC, 2001). However, IARC has stated that there is limited evidence for carcinogenicity in animal testing, which is consistent with the existing HSIS classification for this chemical.

Similarly to repeated dose toxicity studies (refer to **Repeat Dose Toxicity** section), thyroid hyperplasia was frequently reported in animal (rat and mouse) carcinogenicity studies. While this effect did not consistently correlate with the incidence of thyroid tumours across the studies, in one study of rats (10 male and 10 female albino *Rattus norvegicus* and 10 male Wistar), thyroid adenomas (benign tumours; in 88 % of treated animals), and carcinomas (in four albino males and three albino females) were observed following daily exposure to 0.25 % of the chemical (equivalent to 350 mg/kg bw/day) in drinking water for up to two years (REACH; Environment Canada and Health Canada, 2008; WHO, 2003; IARC, 2001). In two of these cases, the thyroid carcinomas were also reported to have metastasised to the lung. It was noted that control group animals were not reported in this study.

In other studies in rats, formation of squamous cell carcinomas of the ear (external auditory duct) and eyelid (meibomian glands) were reported in 17/19 rats exposed to the chemical at a concentration of 0.2 %, daily for up to 26 months (WHO, 2003; IARC, 2001). No carcinomas were reported in the 12 control group animals. Similar neoplastic effects (carcinomas of the ear and eye region) were reported in 10/16 rats exposed to a 10 % solution of the chemical by intraperitoneal (i.p.) injection three times per week, for six months, followed by exposure to

0.2 % of the chemical in drinking water for up to 22 months (IARC, 2001). While squamous-cell carcinomas of the ear (Zymbal gland) have also been observed in rats exposed to the chemical through drinking water, IARC (2001) reported that the animals from that particular study were survivors from unsuccessful tumour transplantation attempts.

In a dietary exposure study, male and female albino rats (18 animals/sex/group) were administered the chemical at 0.01, 0.025, 0.05, 0.1, 0.25, 0.5 or 1 % (equivalent to 0, 5, 12, 25, 50, 125, 250, 500 mg/kg bw/day, respectively), daily, through their food, for two years. Thyroid hyperplasia was observed in animals at

≥125 mg/kg bw/day (REACH; Environment Canada and Health Canada, 2008; WHO, 2003). However, all animals at these doses died within the first 17 months of exposure to the chemical. Of the 29 animals that survived the two year study duration, an increased incidence of liver tumours was reported at ≥5 mg/kg bw/day. No liver tumours were observed in control group animals.

Although thyroid hyperplasia was frequently observed in mice, no thyroid tumours were reported in studies in mice exposed to the chemical through the diet or drinking water (doses ranging from 140 to 1000 mg/kg bw/day; exposure periods ranging from four to 21 months) (Environment Canada and Health Canada, 2008; WHO, 2003; IARC, 2001). In one study in female mice, an

increased incidence of mammary tumours (up to 54 %, compared to 28 % in control group animals) was observed following exposure to the chemical at 0.1–0.2 % (equivalent to 140–280 mg/kg bw/day) in drinking water, daily, for up to six months (REACH; WHO, 2003).

Reproductive and Developmental Toxicity

Reproductive Toxicity

While adverse reproductive effects have been linked to inhibited thyroid function caused by exposure to the chemical (WHO, 2003), only limited reproductive toxicity data are available.

In a study in male rats exposed to the chemical in the diet for two years, at 5 to 500 mg/kg bw/day, reduction or cessation of spermatogenesis was observed at >35 mg/kg bw/day (WHO, 2003; IARC, 2001). Effects on the thyroid (not specified) were also reported.

In studies in juvenile lambs (6-8 month old females and 3-3.5 month old males), the chemical was administered orally, at 50 mg/kg bw/day, daily, for 3.5 months in males and 80 days in females. Reduced size and weight of the reproductive tract in females compared to controls (reported as not statistically significant), in addition to shorter endometrial cells, observed at histological examination, were reported (WHO, 2003). In males, hydrocoele (accumulation of fluid around the testes) and small testes with significantly reduced weight compared to controls, were reported. Within the testes, the seminiferous tubules were empty and reported to be ill-developed and small, and the Sertoli cells were non-functional. Testosterone levels were non-detectable (WHO, 2003).

Developmental Toxicity

The chemical is classified as hazardous (Category 3 substance toxic to reproduction) with the risk phrase ‘Possible risk of harm to the unborn child’ (T; R63) in the HSIS (Safe Work Australia). While there are limited data available, the known effect (including in humans) of reduction in T4 is strongly associated with developmental effects.

In a study in pregnant rats administered the chemical in drinking water at 0.2 % (reported as equivalent to 160–200 mg/kg bw/day) on gestational days (GD) one to 14, growth retardation and malformations of the skeleton, nervous system and eyes (no further details provided) were reported in foetuses examined on GD 20 (REACH; WHO, 2003; IARC, 2001; MAK, 2012). No details on the number of treated animals, or specific details of effects observed, were provided. In another study, thyroid hyperplasia was observed in foetuses and newborn pups, following administration of the chemical at 0.2 % in drinking water to pregnant rats (MAK, 2012).

No maternal or developmental toxicity (teratogenicity) was observed following administration of two subsequent single oral doses of the chemical at 480 mg/kg bw to pregnant rats on GD 12 and 13 (WHO, 2003). However, at 1000 mg/kg bw, maternal toxicity and embryotoxicity were observed in both rats (chemical administered on GD 12 and 14) and mice (chemical administered on GD 10 only).

In studies in pregnant sheep, stillbirths, low birth weights of lambs, dystocia (difficult births) and retention of placenta were reported following administration of the chemical at 50 mg/kg bw day, daily, for two to six months (WHO, 2003).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include the systemic long-term effects of potential carcinogenicity and developmental toxicity. The chemical can also cause harmful effects following a single oral exposure.

Public Risk Characterisation

Although use in cosmetic/domestic products in Australia is not known, the chemical is reported to be used in domestic products overseas at concentrations up to 7 % (HSDB).

The chemical is currently listed on Schedule 6 of the SUSMP, with a number of warning statements, first aid instructions and safety directions applying to the use of this chemical. The current controls are considered adequate to minimise the risk to public health posed by domestic products containing the chemical; therefore; the chemical is not considered to pose an unreasonable risk to public health.

Occupational Risk Characterisation

During product formulation, exposure may occur, particularly where manual or open processes are used. These could include transfer and blending activities, quality control analysis, and cleaning and maintaining equipment. Worker exposure to the chemical at lower concentrations could also occur while using formulated products containing the chemical. The level and route of exposure will vary depending on the method of application and work practices employed.

Given the critical systemic long-term health effects, the chemical could pose an unreasonable risk to workers unless adequate control measures to minimise exposure are implemented. The chemical should be appropriately classified and labelled to ensure that a person conducting a business or undertaking (PCBU) at a workplace (such as an employer) has adequate information to determine the appropriate controls.

Additionally, guidance on the *Interpretation of workplace exposure standards for airborne contaminants* advises that exposure to carcinogens should be eliminated or minimised as far as reasonably practicable (Safe Work Australia, 2013).

NICNAS Recommendation

Current risk management measures are considered adequate to protect public and workers' health and safety, provided that all requirements are met under workplace health and safety, and poisons legislation as adopted by the relevant state or territory. No further assessment is required.

Regulatory Control

Public Health

Products containing the chemical should be labelled in accordance with state and territory legislation (SUSMP, 2015).

Work Health and Safety

The chemical is recommended for classification and labelling under the current Approved Criteria and adopted GHS as below. This assessment does not consider classification of physical and environmental hazards.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Harmful if swallowed (Xn; R22)*	Harmful if swallowed - Cat. 4 (H302)
Carcinogenicity	Carc. Cat 3 - Limited evidence of a carcinogenic effect (Xn; R40)*	Suspected of causing cancer - Cat. 2 (H351)

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Reproductive and Developmental Toxicity	Repro. Cat 3 - Possible risk of harm to the unborn child (Xn; R63)*	Suspected of damaging the unborn child - Cat. 2 (H361d)

[a] Approved Criteria for Classifying Hazardous Substances [NOHSC:1008(2004)].

[b] Globally Harmonized System of Classification and Labelling of Chemicals (GHS) United Nations, 2009. Third Edition.

[*] Existing Hazard Classification. No change recommended to this classification

Advice for consumers

Products containing the chemical should be used according to the instructions on the label.

Advice for industry

Control measures

Control measures to minimise the risk from exposure to the chemical should be implemented in accordance with the hierarchy of controls. Approaches to minimise risk include substitution, isolation and engineering controls. Measures required to eliminate, or minimise risk arising from storing, handling and using a hazardous chemical depend on the physical form and the manner in which the chemical is used. Examples of control measures that could minimise the risk include, but are not limited to:

- using closed systems or isolating operations;
- health monitoring for any worker who is at risk of exposure to the chemical, if valid techniques are available to monitor the effect on the worker's health;
- minimising manual processes and work tasks through automating processes;
- work procedures that minimise splashes and spills;
- regularly cleaning equipment and work areas; and
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the chemical.

Guidance on managing risks from hazardous chemicals are provided in the *Managing risks of hazardous chemicals in the workplace—Code of practice* available on the Safe Work Australia website.

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. Guidance in selecting personal protective equipment can be obtained from Australian, Australian/New Zealand or other approved standards.

Obligations under workplace health and safety legislation

Information in this report should be taken into account to help meet obligations under workplace health and safety legislation as adopted by the relevant state or territory. This includes, but is not limited to:

- ensuring that hazardous chemicals are correctly classified and labelled;
- ensuring that (material) safety data sheets ((M)SDS) containing accurate information about the hazards (relating to both health hazards and physicochemical (physical) hazards) of the chemical are prepared; and
- managing risks arising from storing, handling and using a hazardous chemical.

Your work health and safety regulator should be contacted for information on the work health and safety laws in your jurisdiction.

Information on how to prepare an (M)SDS and how to label containers of hazardous chemicals are provided in relevant codes of practice such as the *Preparation of safety data sheets for hazardous chemicals—Code of practice* and *Labelling of workplace hazardous chemicals—Code of practice*, respectively. These codes of practice are available from the Safe Work Australia website.

A review of the physical hazards of the chemical has not been undertaken as part of this assessment.

References

Environment Canada and Health Canada, 2008. Screening Assessment for the Challenge: Thiourea. Accessed October 2015 at https://www.ec.gc.ca/ese-ees/default.asp?lang=En&n=CE2D78C6-1

European Commission Cosmetic Ingredients and Substances (CosIng) Database. Accessed October 2015 at http://ec.europa.eu/consumers/cosmetics/cosing/

Galleria Chemica. Accessed October 2015 at http://jr.chemwatch.net/galleria/

Hazardous Substances Data Bank (HSDB). US National Library of Medicine. Accessed October 2015 at http://toxnet.nlm.nih.gov

International Agency for Research on Cancer (IARC) 2001. Some Thyrotropic Agents: Thiourea. Vol. 79. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Accessed October 2015 at http://monographs.iarc.fr/ENG/Monographs/vol79/mono79-24.pdf

Maximale Arbeitsplatz-Konzentration (MAK) 2012. Thiourea [MAK Value Documentation, 1990]. The MAK Collection for Occupational Health and Safety. 302-3114. Accessed October 2015 at http://onlinelibrary.wiley.com/doi/10.1002/3527600418.mb6256e0001/full

REACH Dossier (REACH). Thiourea (CAS No. 62-56-6). Accessed October 2015 at http://echa.europa.eu/web/guest/information-on-chemicals/registered-substances

Safe Work Australia (SWA). Hazardous Substances Information system (HSIS). Accessed October 2015 at http://hsis.safeworkaustralia.gov.au/HazardousSubstance

SPIN (Substances in Preparations in Nordic Countries) Database. Accessed October 2015 at http://195.215.202.233/DotNetNuke/default.aspx

The Poisons Standard (the Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP)) 2015. Accessed October 2015 at https://www.comlaw.gov.au/Details/F2015L00844

US National Library of Medicines, Household Products Database, Health& Safety Information on Household Products. Accessed October 2015 at http://householdproducts.nlm.nih.gov/

World Health Organisation (WHO) 2003. Concise International Chemical Assessment Document (CICAD) 49: Thiourea. Accessed October 2015 at http://www.who.int/ipcs/publications/cicad/en/cicad49.pdf

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