Cadmium oxide (CdO): Human health tier II assessment

04 July 2014

CAS Number: 1306-19-0

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	cadmium monoxide cadmium fume
Structural Formula	
Molecular Formula	CdO
Molecular Weight (g/mol)	128.41
Appearance and Odour (where available)	Odourless dark-brown powder or cubic crystals.
SMILES	O=[Cd]

Import, Manufacture and Use

Australian

No specific Australian use, import, or manufacturing information has been identified.

International

The following international uses have been identified through European Union Registration, Evaluation and Authorisation of Chemicals (EU REACH) dossiers; the Organisation for Economic Cooperation and Development Screening information data set International Assessment Profile (OECD SIAP); EU Risk Assessment Reports (EU RAR); the International Agency for Research on Cancer (IARC) report; the United States (US) National Toxicology Program's Report on Carcinogens (NTP RoC); and Galleria Chemica.

The chemical has reported site-limited use including:

- as a catalyst for oxidation-reduction, dehydrogenation, cleavage and polymerisation reactions;
- as a sensitiser for photochemical reactions; and
- in the manufacture of fibre-structured nickel batteries, cadmium-based pigments and colouring agents, stabilisers and cadmium salts of natural fatty acids.

Restrictions

Australian

Cadmium and cadmium compounds are listed in the *Poisons Standard* (the Standard for the Uniform Scheduling of Medicines and Poisons—SUSMP (SUSMP, 2012)) under the following Schedules:

Appendix I, The uniform paint standard

The following applies to paints containing cadmium or cadmium compounds at >0.1 %. 'The proportion of a substance for the purposes of this Schedule is calculated as a percentage of the element present in the non-volatile content of the paint' (SUSMP 2012).

'A person must not manufacture, sell, supply or use a paint containing >0.1 % of cadmium or cadmium compounds for application to:

- a roof or any surface to be used for the collection or storage of potable water; or
- furniture; or
- any fence, wall, post, gate or building (interior or exterior) other than a building which is used exclusively for industrial purposes or mining or any oil terminal; or
- any premises used for the manufacture, processing, preparation, packing or serving of products intended for human or animal consumption' (SUSMP, 2012).

Additionally, 'a person must not manufacture, sell, supply or use a paint for application to toys unless the paint complies with the specification for coating materials contained in Australian/New Zealand Standard AS/NZS ISO 8124.3:2012 entitled *Safety of toys Part 3: Migration of certain elements* (ISO 8124-03:2010, MOD)' (SUSMP, 2012).

*'**Schedule 6*** except when:

(a) included in Schedule 4; or

(b) in paints or tinters containing 0.1 per cent or less of cadmium calculated on the non-volatile content of the paint or tinter' (SUSMP, 2012).

Schedule 6 substances are considered to have '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' (SUSMP, 2012).

Cadmium and its compounds are also listed as restricted hazardous chemicals in the Australia work health and safety Regulations 2011 for 'use in abrasive blasting at a concentration of greater than 0.1 % as cadmium' (SafeWork Australia).

International

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

- EU Cosmetic Directive 76/768/EEC Annex II: List of Substances which must not form part of the Composition of Cosmetic Products;
- New Zealand Cosmetic Products Group Standard—Schedule 4: Components cosmetic products must not contain; and
- Health Canada List of prohibited and restricted cosmetic ingredients (The "Hotlist").

Cadmium and its compounds are also restricted in the EU under Annex XVII of the European Chemicals Agency (ECHA) REACH Regulation, including in:

- plastic materials;
- paints;
- metal plating; and
- brazing (soldering/welding) fillers.

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):

T+; R26 (Very toxic by inhalation)

T; R48/23/25 (Danger of serious damage to health by prolonged exposure through inhalation and if swallowed)

Carc. Cat. 2; R45 (May cause cancer)

Muta. Cat. 3; R68 (Possible risk or irreversible effects)

Repr. Cat. 3; R62-63 (Possible risk of impaired fertility and of harm to the unborn child)

Exposure Standards

Australian

Cadmium and cadmium compounds have an exposure standard of 0.01 mg/m^3 time weighted average (TWA).

International

The following exposure standards are identified (Galleria Chemica):

An exposure limit (TWA) of 0.01–0.2 mg/m^3 in different countries such as USA, Canada, Latvia and Switzerland.

Health Hazard Information

Toxicokinetics

There is a large amount of information on the toxicokinetics of the cadmium ion in humans. This information is mostly related to general cadmium exposure in workers, and is not specific to particular cadmium compounds.

Cadmium oxide is considered to be relatively water-insoluble. However, in vitro studies have demonstrated the solubility of the chemical to be 94 % in artificial gastric juice and 0.15 % in artificial intestinal juice (EU RAR, 2007). Due to the high solubility of cadmium oxide in gastric juice, water-soluble cadmium compounds such as cadmium chloride are appropriate analogues for orally administered cadmium oxide.

In rodent dietary exposure studies using the chemical, significant accumulation of cadmium was detected in the liver, kidneys, lungs and spleen. Levels in liver and kidneys were reported to be dose-dependent. However, no significant increase in blood or urine levels of cadmium was detected. Absorption rates following oral exposure to low doses of the chemical were reported to be much greater than those determined for exposure to higher doses (EU RAR, 2007).

Animal studies have demonstrated that absorption of the chemical following inhalation exposure ranges from 30 % (dusts, size-dependent) to 50 % (fumes). In humans, an inhalation absorption rate of 10–30 % (dusts, size-dependent) is reported (OECD, 2004). Dermal absorption is expected to be low due to the ionic nature of the chemical.

Following long-term low-level exposure, cadmium is reported to be widely distributed in the body and has a biological half-life of 10–20 years. The greatest accumulation occurs in the kidneys and liver, with only 0.005–0.02 % reported to be excreted in urine and faeces each day. Cadmium is also detectable in the placenta, and can cross the placenta, although foetal concentrations are lower than placental concentrations. Concentrations of cadmium in newborn blood were 40–50 % lower than the levels in maternal blood (EU RAR, 2007). Cadmium is reported to be found in human breast milk at <1 µg/L (OECD, 2004). In tissue, cadmium is bound to metallothionein, a low molecular weight metal-binding protein thought to play a key role in the metabolism and detoxification of cadmium (EU RAR, 2007).

Human data available on cadmium indicate that gastro-intestinal absorption rates are low (5–10 %), and vary depending on the source of the cadmium, presence of zinc in the diet, the body’s iron stores (deficiencies linked with increased cadmium absorption) and the person’s age and physiological condition (young or pregnant and lactating animals have been shown to absorb more cadmium than non-pregnant adult animals) (EU RAR, 2007; OECD, 2004).

It should be noted that higher levels of cadmium (particularly in the kidney) are detected in smokers compared with non-smokers, as cadmium has been shown to accumulate in tobacco plant leaves (WHO, 2010).

Acute Toxicity

Oral

The chemical had high acute toxicity in animals tests following oral exposure.

The oral median lethal dose (LD50) values in rats range from 72–296 mg/kg bw (EU RAR, 2007).

Dermal

No data are available.

Inhalation

The chemical is classified as hazardous with the risk phrase 'Very toxic by inhalation' (T+; R26) in HSIS (Safe Work Australia). The available data support this classification.

The chemical was acutely toxic in animals tests following inhalation exposure to fumes of the chemical. The inhalation median lethal concentration (LC50) value in rats is reported to be 25 mg/m^3 (measured as cadmium). The cause of death following exposure was reported to be pulmonary oedema. Other signs of toxicity included rales (abnormal respiratory sounds) and laboured breathing (EU RAR, 2007).

Observation in humans

There are many case reports of acute poisoning following inhalation of cadmium oxide fumes. Documented signs of toxicity include nausea, fever, difficulties in respiration and severe respiratory irritation. Pulmonary oedema, resulting in mortality, was commonly reported following acute exposure (EU RAR, 2007).

Several cases of cadmium poisoning (compound not specified) from ingesting contaminated food or drinks have been documented. Reported signs and symptoms of toxicity include nausea, vomiting, diarrhoea and abdominal cramps.

Corrosion / Irritation

Respiratory Irritation

While no specific data are available, based on the sublethal symptoms observed in inhalation studies in animals and observations in humans, the chemical is expected to be irritating to the respiratory tract.

Skin Irritation

No data are available.

Eye Irritation

No data are available.

Sensitisation

Skin Sensitisation

While it is unclear whether the chemical is a potential sensitiser, no incidences of skin or respiratory sensitisation have been reported across the cases of occupational exposure to the chemical. However, skin patch tests using two other cadmium compounds (cadmium chloride and cadmium sulfate) were positive in seven out of approximately 150 patients attending a dermatological department between 1979-1981 (EU RAR, 2007).

Repeated Dose Toxicity

Oral

The chemical is classified as hazardous with the risk phrase 'Toxic: danger of serious damage to health by prolonged exposure if swallowed.' (T; R48/25) in HSIS (Safe Work Australia). While there are no experimental data available for this specific chemical, data from animal studies and observations in humans following oral exposure to cadmium compounds support this classification, as presented in the following sections.

A chronic exposure study in male Wistar rats using cadmium chloride reported potential damage to the vertebrae. The animals (10/dose) were orally exposed to cadmium chloride through drinking water for 12 months at 1, 5, 50 mg/L. No treatment-related signs were reported at 1 mg/L. At ≥5 mg/L, increased lumbar spine deformities and a decrease in lumbar spine mineralisation (including calcium, magnesium, zinc, copper, iron and phosphate) were reported. A decrease in mechanical strength of the vertebral column at the fourth lumbar vertebra (L4) was reported at 50 mg/L. The L4 was reported to be fractured in 30 % of animals in this group, while 40 % were reported to have L4 deformities. The remaining animals in this group (30 %) had intact L4s. The NOAEL (no observed adverse effect level) and LOAEL (lowest observed adverse effect level) for this study were reported to be 0.2 mg/kg bw/day and 0.5 mg/kg bw/day, respectively (REACH).

Dermal

No data are available.

Inhalation

The chemical is classified as hazardous with the risk phrase 'Toxic: danger of serious damage to health by prolonged exposure if inhaled.' (T; R48/23) in HSIS (Safe Work Australia). The available data support this classification.

Effects observed in animal studies following repeated-dose exposure to the chemical are reported to be similar to those observed in acute exposure studies including rales, laboured breathing and pneumonia (EU RAR, 2008).

In a 13-week repeated-dose inhalation study in rats, the no observed adverse effect concentration (NOAEC) for the chemical was reported to be 0.025 mg/m^3. At higher doses (≥0.05 mg/m^3), treatment-related lesions in the lungs, including inflammation and fibrosis, were observed. A dose-related increase in hyperplasia was also reported (EU RAR, 2007).

Observation in humans

Exposure to low levels of cadmium over a long period of time has been linked to chronic cadmium poisoning. The effects of cadmium on specific target organs following exposure in humans are summarised and provided below.

Respiratory effects

There are a number of documented case studies of workers chronically exposed to the chemical fumes (EU RAR, 2007). Effects reported include fatigue, respiratory irritation, shortness of breath, decreased lung function and recurrent bronchitis.

It is suggested that an increase in residual levels of the chemical in lungs may lead to chronic obstructive airway disease and (in some cases) mortality, all of which have been documented following exposure (EU RAR, 2007). A lowest observed adverse effect concentration (LOAEC) of 0.0031 mg/L, based on lung effects (increased residual levels of the chemical), was derived from a study on workers exposed to the chemical fumes at <0.5 mg/m^3 over several years.

Renal effects

The kidneys are considered to be the main target organ for cadmium toxicity following repeated oral and inhalation exposure (ATSDR, 2012; EU RAR, 2007). Initial signs of kidney effects following cadmium exposure include tubular dysfunction, a decreased glomerular filtration rate, and increased proteinuria and enzymuria. Renal dysfunction is considered to occur when renal cortex cadmium concentrations reach 200 ppm (equivalent to 5–10 µg/g creatinine) (EU RAR, 2007).

An increased frequency of kidney stones has also been reported in workers exposed to cadmium (18 %), compared with unexposed workers (3 %) (EU RAR, 2007).

Skeletal effects

Oral cadmium exposure is reported to cause bone disease in humans (EU RAR, 2008). While the underlying mechanism is not clearly understood, it is thought that cadmium-induced kidney damage and resulting hypercalcinuria (elevated levels of calcium in the urine) may promote osteoporotic effects in bone (EU RAR, 2007).

In a case study in Japan, a high incidence of Itai-Itai disease was diagnosed in patients from specific geographical locations. It was found that farms in these areas were irrigated by a river being polluted by cadmium sludge from an upstream mine, and the patients may have been exposed to cadmium for over 30 years. Samples of rice taken from those areas were reported to contain cadmium at 0.68 mg/kg, compared with 0.066 mg/kg in other areas (EU RAR, 2007). Itai-Itai disease is characterised by osteomalacia (softening of the bones), osteoporosis, severe renal tubular disease, and is associated with severe pain (WHO, 2011). A limited number of case reports have also documented clinical bone disease in workers exposed to cadmium compounds (EU RAR, 2007).

Genotoxicity

The chemical is classified as hazardous, as a Category 3 mutagen, with the risk phrase ‘Possible risk of irreversible effects’ (Xn; R68) in HSIS (Safe Work Australia). While the available data for the chemical do not support this classification, results for soluble cadmium compounds indicate that absorbed cadmium is a possible mutagen.

In vitro

The chemical did not induce genotoxic effects in *Salmonella typhimurium* bacterial strains at 0.003–3.333 mg/plate, with or without metabolic activation (NTP, 1995; EU RAR, 2007).

In vivo

In an inhalation exposure study in mice, the chemical was reported to not increase the frequency of micronucleated erythrocytes in peripheral blood, following exposure at 0.025–1 mg/m^3 for 13 weeks (NTP, 1995; EU RAR, 2007). However, these results were not considered to be reliable due to deficiencies in the study protocol (EU RAR, 2007).

Other cadmium compounds

Cadmium salts have been shown to induce genotoxic effects in both in vitro (reduction in colony-forming ability and DNA strand breaks in *Escherichia coli*) and in vivo studies (sister chromatid exchange and chromosomal aberration in mice, and DNA strand breaks in rats) (EU RAR, 2007; IARC, 2012).

Observations in humans

Chromosomal aberrations, increased frequency of micronuclei, and sister chromatid exchanges have been detected in humans environmentally exposed to cadmium (EU RAR, 2007). However, the specific cadmium compounds involved have not been identified.

Carcinogenicity

The chemical is classified as hazardous, as a Category 2 carcinogen, with the risk phrase ‘May cause cancer’ (T; R45) in HSIS (Safe Work Australia). The available data support this classification.

In chronic inhalation studies in rats, the chemical (dusts and fumes) induced carcinogenic effects (malignant lung tumours) at 0.03 mg/m^3. However, lower incidences of tumours were reported in rats exposed to fumes, compared with dusts, which was shown to be related to the level of pulmonary deposition of the chemical from the two forms (EU RAR, 2007).

Observations in humans

There are many case studies that explore the link between exposure to cadmium and increased incidences of cancer in workers (NTP, 2011; IARC, 2012).

The International Agency for Research on Cancer (IARC) has classified cadmium and cadmium compounds as 'Carcinogenic to humans (Group 1)' based on sufficient evidence in humans and experimental animals (IARC, 2012). Additionally, the US National Toxicology Program (NTP) has also classified cadmium and cadmium compounds as 'Known to be human carcinogens' (NTP, 2011).

Significantly increased mortalities due to lung cancer were reported in workers in cadmium-processing plants, cadmium recovery plants and those who worked in the nickel-cadmium battery production industry (IARC, 2012). An increased risk of lung cancer was identified in workers with long-term employment in high cadmium-exposure jobs.

A few cases of cancer of the prostate, pancreas and kidney have also been reported following exposure to cadmium (either from occupational exposure or by contamination).

In some of these cases, workers may have been exposed to other chemicals, including arsenic and nickel. Both IARC and the US NTP concluded that the increase in lung cancers could not be solely due to co-exposure to other chemicals (NTP, 2011; IARC, 2012). However, the Agency for Toxic Substances and Disease Registry (ATSDR) concluded that the interpretation of these observations in humans is complicated by co-exposure with other metals, and that there is a 'lack of significant relationship between cadmium exposure and duration' (ASTDR, 2012).

Reproductive and Developmental Toxicity

The chemical is classified as hazardous, as a Category 3 reproductive and developmental toxin, with the risk phrases 'Possible risk of impaired fertility' (Xn; R62) and 'Possible risk of harm to the unborn child' (Xn; R63) in HSIS (Safe Work Australia). While the available data for this specific chemical do not conclusively support these classifications, results for soluble cadmium compounds indicate that absorbed cadmium is a possible reproductive and developmental toxin. In the absence of more comprehensive information, there is insufficient evidence to support a recommendation to amend these classifications.

Reproductive toxicity

In a study in male and female F344/N rats and male and female B6C3F1 mice, animals were exposed to the chemical by inhalation at 0.025, 0.5, 0.1, 0.25 or 1 mg/m³ for 13 weeks. Decreased spermatid counts and increased oestrous cycle lengths were reported in rats in the highest dose groups. These effects were not observed in mice (NTP, 1995). A reproductive LOAEL of 1 mg/m^3 was reported for this study based on the effects observed in rats (EU RAR, 2007).

In a study in female Wistar rats, animals were exposed to cadmium chloride by oral gavage at 0.04, 0.4, 4 and 40 mg/kg bw/day for 14 weeks. Increased oestrous cycle lengths and decreased body weights compared with controls were reported in rats in the highest dose group (40 mg/kg bw/day) after six weeks of treatment; this was not reported for other dose groups. A reproductive NOAEL of 4 mg/kg bw/day and LOAEL of 40 mg/kg bw/day were reported for this study (NICNAS).

Developmental toxicity

In a developmental toxicity study in Sprague Dawley (SD) rats, animals were exposed to the chemical by inhalation at 0.05, 0.5 or 2 mg/m^3 on gestation days 4–19. Exposure-related foetal skeletal variations (reduced ossification of the pelvis and the sternebrae) and reduced foetal weights were reported (statistically significant in the highest dose group). However, significantly decreased maternal body weights and reduced absolute liver and kidney weights were also recorded at the highest dose, in addition to one mortality at gestation day 17. Signs of toxicity were observed in dams in all treatment groups. The effects included dyspnoea (difficult or laboured breathing) and hypoactivity (NTP, 1995). A maternal NOAEL of <0.05 mg/m^3 and a developmental NOAEL of 0.5 mg/m^3 were reported for this study (EU RAR, 2007).

In another study, Swiss mice were exposed to the chemical by inhalation at 0.05, 0.5 or 2 mg/m³ on gestation days 4–17. An increased frequency in reduced ossification of the sternebrae was reported in foetuses (statistically significant at the highest dose), while significantly reduced foetal weights were reported at ≥0.5 mg/m^3. Signs of toxicity, including dyspnoea and hypoactivity, were observed in dams from all treatment groups, in addition to significantly reduced maternal body weights and five mortalities (euthanised moribund) from the highest dose group (NTP, 1995). A maternal NOAEL of <0.05 mg/m^3 and a developmental NOAEL of 0.05 mg/m^3 were reported for this study (EU RAR, 2007).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include systemic long-term effects (carcinogenicity, specific organ effects, mutagenicity, reproductive toxicity and developmental toxicity), and systemic acute effects (acute toxicity from oral and inhalation exposure). The chemical is also a potential respiratory irritant.

Public Risk Characterisation

Given the uses identified for the chemical, it is unlikely that the public will be exposed. Hence, the public risk from this chemical is not considered to be unreasonable.

Occupational Risk Characterisation

Given the critical health effects, the chemical may pose an unreasonable risk to workers unless adequate control measures to minimise inhalation exposure to the chemical 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 appropriate controls.

The data available support an amendment to the hazard classification in HSIS (refer to **Recommendation** section).

NICNAS Recommendation

Assessment of the chemical is considered to be sufficient, provided that the recommended amendment to the classification is adopted, and labelling and all other requirements are met under workplace health and safety and poisons legislation as adopted by the relevant state or territory.

Regulatory Control

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 hazards and environmental hazards.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Toxic if swallowed (T; R25) Very toxic by inhalation (T+; R26)*	Toxic if swallowed - Cat. 3 (H301) Fatal if inhaled - Cat. 1 (H330)
Repeat Dose Toxicity	Toxic: danger of serious damage to health by prolonged exposure through inhalation (T; R48/23)* Toxic: Danger of serious damage to health by prolonged exposure if swallowed (T; R48/25)*	Causes damage to organs through prolonged or repeated exposure through inhalation - Cat. 1 (H372) Causes damage to organs through prolonged or repeated exposure if swallowed - Cat. 1 (H372)
Genotoxicity	Muta. Cat 3 - Possible risk of irreversible effects (Xn; R68)*	Suspected of causing genetic defects - Cat. 2 (H341)
Carcinogenicity	Carc. Cat 2 - May cause cancer (T; R45)*	May cause cancer - Cat. 1B (H350)
Reproductive and Developmental Toxicity	Repro. Cat 3 - Possible risk of harm to the unborn child (Xn; R63)* Repro. Cat 3 - Possible risk of impaired fertility (Xn; R62)*	Suspected of damaging fertility - Cat. 2 (H361f) 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 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 which may minimise the risk include, but are not limited to:

- using closed systems or isolating operations;
- using local exhaust ventilation to prevent the chemical from entering the breathing zone of any worker;
- 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;
- air monitoring to ensure control measures in place are working effectively and continue to do so;
- 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 assist with meeting 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

European Union Risk Assessment Report (EU RAR) 2007. Final report for cadmium oxide. Part II - Human Health. Accessed September 2013 at http://esis.jrc.ec.europa.eu/

European Union Risk Assessment Report (EU RAR) 2008. Summary report for cadmium metal and cadmium oxide. Accessed September 2013 at http://esis.jrc.ec.europa.eu/

Galleria Chemica. Accessed at September 2013 at http://jr.chemwatch.net/galleria/

International Agency for Reseach on Cancer (IARC) 2012. IARC monographs on the evaluation of carcinogenic risks to humans. A Review of Human Carcinogens: Cadmium. Volume 100 C. Accessed September 2013 at http://monographs.iarc.fr/ENG/Monographs/vol100C/index.php

National Industrial Chemicals Notification and Assessment Scheme (NICNAS). Inventory Multi-tiered Assessment and Prioritisation (IMAP) Human Health Tier II Assessment for Cadmium chlorides and sulfates. Australian Government Department of Health. Available at http://www.nicnas.gov.au

National Toxicology Program (NTP) 1995. Technical Report on toxicity studies of cadmium oxide administered by inhalation to F344/N Rats and B6C3F1 Mice. U.S. Department of Health and Human Services. Accessed September 2013 at http://ntp.niehs.nih.gov/

National Toxicology Program (NTP) 2011. Report on Carcinogens, Twelfth Edition: Cadmium and cadmium compounds. U.S. Department of Health and Human Services. Accessed September 2013 at http://ntp.niehs.nih.gov/?objectid=03C9AF75-E1BF-FF40-DBA9EC0928DF8B15

OECD (2004). SIDS Initial Assessment Profile (SIAP): Cadmium oxide and cadmium metal. Accessed September 2013 at http://webnet.oecd.org/HPV/

REACH Dossier. Cadmium oxide (CAS No. 1306-19-0). Accessed September 2013 at http://echa.europa.eu/web/guest/information-on-chemicals/registered-substances

Safe Work Australia (SWA). Hazardous Substances Information System (HSIS). Accessed September 2013 at http://hsis.safeworkaustralia.gov.au/HazardousSubstance

Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) 2012. Australian Government Department of Health and Ageing. Therapeutic Goods Administration. The SUSMP No. 3, 2012. Accessed September 2013 at http://www.comlaw.gov.au/Details/F2012L01200

World Health Organisation (WHO) 2010. Exposure to cadmium: A major public health concern. International Programme on Chemical Safety. Accessed September 2013 at http://www.who.int/ipcs/assessment/public_health/cadmium/en/

World Health Organisation (WHO) 2011. Cadmium in Drinking-water. Background document for the development of WHO Guidelines for Drinking-water Quality, Fourth edition. Accessed September 2013 at http://www.who.int/water_sanitation_health/publications/2011/dwq_guidelines/en/index.html

Last update 04 July 2014

Share this page