Lead phthalates: Human health tier II assessment

07 February 2014

Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
1,2-Benzenedicarboxylic acid, lead(2+) salt (1:1)	6838-85-3
1,2-Benzenedicarboxylic acid, lead salt	16183-12-3
1,2-Benzenedicarboxylic acid, lead(2+) salt	18608-34-9
Lead, [1,2-benzenedicarboxylato(2-)]oxodi-	57142-78-6
Lead, [1,2-benzenedicarboxylato(2-)]dioxotri-	69011-06-9

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

Grouping Rationale

This group of five chemical compounds consists of lead salts of phthalates. These compounds have been included in this group due to the expectation that the physicochemical properties will not vary greatly, with the compounds within this group having related end uses. The toxicity of these chemicals which is of most concern is considered to result from the presence of the lead component (cation). The lead compounds with an unspecified oxidation state will predominantly contain lead in the +2 oxidation state. In addition, information outlined in the Organisation for Economic Co-operation and Development's (OECD) guideline on Grouping of Chemicals (OECD, 2007) provided guidance on the grouping of these chemicals based on physico-chemical or toxicological criteria.

Import, Manufacture and Use

Australian

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

International

For the group, the following international uses have been identified through Galleria Chemica and the Substances in Preparations in Nordic Countries (SPIN) database.

Reported site-limited uses including:

- as a heat stabiliser in plastics.

Restrictions

Australian

Lead and lead compounds are listed in the Poisons Standard (the Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP, 2012)) in:

Appendix I, Uniform Paint Standard

Lead compounds are not permitted to be used in domestic or industrial paints 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.

Appendix C

Lead compounds in paints, tinters, inks or ink additives except in preparations containing = 0.1 % of lead calculated on the non-volatile content of the paint, tinter, ink or ink additive.

Appendix C substances, other than those included in the Schedule 9, are considered of such danger to health as to warrant prohibition of sale, supply and use. These substances are poisons prohibited from sale, supply or use because of their known potential for harm to human and/or animal health.

Schedule 6

Lead compounds unless specified in Appendix C or:

(a) when included in Schedule 4 or 5;

(b) in paints, tinters, inks or ink additives;

(c) in preparations for cosmetic use containing 100 mg/kg or less of lead;

(d) in pencil cores, finger colours, showcard colours, pastels, crayons, poster paints/colours or coloured chalks containing 100 mg/kg or less of lead; or

(e) in ceramic glazes when labelled with the warning statement: *CAUTION - Harmful if swallowed. Do not use on surfaces which contact food or drink.* Written in letters not less than 1.5 mm in height.

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.

Schedule 5

Lead compounds in preparations for use as hair cosmetics, unless specified in Appendix C.

Schedule 5 substances are considered to have low potential for causing harm, the extent of which can be reduced through the use of appropriate packaging with simple warnings and safety directions on the label.

International

The risk of exposure to lead and lead compounds has been recognised internationally, which has resulted in broad restrictions regarding occupational and public exposure.

Cosmetics

The lead compounds contained in this group appear on the following:

- Health Canada List of Prohibited and Restricted Cosmetic Ingredients (The "Hotlist").
- The EU Cosmetic Directive 76/768/EEC Annex II: List of Substances which must not form part of the Composition of Cosmetic Products.
- The New Zealand Cosmetic Products Group Standard - Schedule 4: Components Cosmetic Products Must Not Contain.
- The Thailand Cosmetic Act - Prohibited Substances.

Existing Worker Health and Safety Controls

Hazard Classification

The compounds in this group are not individually listed in the Hazardous Substances Information System (HSIS) (Safe Work Australia) and therefore, by default, are covered by the lead and lead compounds classification as hazardous with the following risk phrases for human health:

Repr. Cat. 1; R61 (Reproductive toxicity - may cause harm to the unborn child)

Repr. Cat. 3; R62 (Reproductive toxicity - possible risk of impaired fertility)

Xn; R20/R22 (Harmful by inhalation and if swallowed)

Xn; R33 (Danger of cumulative effects)

Exposure Standards

Australian

Lead, inorganic dusts and fumes (as lead) have the following exposure standards reported in HSIS (Safe Work Australia), which apply to the lead compounds in this assessment:

Time Weighted Average (TWA): 0.15 mg/m^3 for lead compounds (as lead).

Short-Term Exposure Limits (STEL): No specific exposure standards are available.

International

For lead compounds in general the following exposure limits were identified:

TWA: 0.20 mg/m^3 [Thailand, USA (Idaho)]

TWA: 0.15 mg/m^3 [Argentina, Canada (Northwest Territories, Yukon), Egypt, European Union, Gibraltar, India, Malta, Singapore, Slovak Republic]

TWA: 0.10 mg/m^3 [Austria, Maximum Allowable Concentration (MAK), New Zealand, Republic of South Africa, Sweden]

TWA: 0.05 mg/m³ [Bulgaria, Canada (Alberta, British Columbia, Prince Edward Island, Nova Scotia, Ontario), China, Italy, Malaysia, USA (Hawaii, Michigan, North Carolina, Oregon, Washington, Wyoming)]

TWA: 0.15 ppm [Luxembourg]

STEL: 0.45 mg/m³ [Argentina, Canada (Northwest Territories, Yukon), Egypt]

STEL: 0.40 mg/m³ [Austria MAK]

STEL: 0.15 mg/m³ [Canada (Ontario, Saskatchewan)]

Health Hazard Information

The health hazard associated with each end point is considered to be due to the lead cation. While the common names of these chemicals refer to them as phthalates, these chemicals are not closely related to the phthalate plasticisers. The plasticisers which are of concern for reproductive effects are diesters of phthalic acid (NICNAS, 2010), while the current compounds are salts. The reproductive concerns relate only to phthalate esters. The phthalate component is not considered to contribute to the final recommendation, as toxic effects of the phthalate ion are not expected to be observed at levels that are reasonably achievable given the toxicity of the lead component. Although limited experimental data are available for some members of this group, and the effects of the phthalate ion are also reflected in these studies, data sources for determining the hazard for the lead cation include animal studies on well characterised organic and inorganic lead compounds, and a large amount of literature on observations in humans.

Toxicokinetics

Inorganic lead compounds can be absorbed orally, dermally or via inhalation (NICNAS, 2007).

When ingested, the absorption of inorganic lead compounds in the human gastrointestinal tract is influenced by different factors, the most significant being age. Children (up to the age of eight) are estimated to absorb up to 50 % of the lead dose they ingest, while adults would absorb up to 10 % of the dose they ingest. This route of absorption can be dependent on solubility and particle size with smaller particles being absorbed more readily than larger ones.

In an oral repeat dose toxicity study, rats were dosed with 0, 200, 500 or 1000 ppm lead acetate and tested for four, eight or 12 weeks. The blood lead concentration (PbB) level range was 40 - 100 μg/dL and the kidney lead levels were highest at four weeks. For all test groups the urinary lead excretion was highest at four weeks then decreased with continued exposure to lead (REACH).

If inhaled, the size of lead compound particulates can dictate the site of deposition and rate of absorption (NICNAS, 2007).

Absorption via the dermal route is the least efficient (NICNAS, 2007). Less than 0.3 % of lead from lead acetate in cosmetics was absorbed dermally in human male volunteers over a 12 hour period. When lead nitrate was applied to the skin, 30 % of the dose was absorbed. It is not known if the absorption was systemic or confined to the layers of the skin.

Lead stored in bone can be released into the blood after exposure has ceased. Distribution in bone is not uniform and lead has been shown to accumulate in areas that are undergoing active calcification at the time of exposure (NICNAS, 2007). Inorganic lead is distributed in the body independently of the source compound and route of exposure. The spatial distribution of lead in bone is similar between children and adults, although adults generally have a higher concentration. In blood, 99 % of lead is bound to proteins within erythrocytes (NICNAS, 2007).

Mobilisation of lead from bone increases during pregnancy when maternal bone is catabolised to produce the foetal skeleton. It has been shown that up to 80 % of lead in human cord blood comes from maternal bone stores and can be transferred into the foetal skeleton during its formation.

The PbB concentration is a reflection of recent exposure and does not capture the more significant impact and slower elimination kinetics of the chemical in bone (ATSDR, 2007). The accumulation of lead in bone is considered a biomarker for long-term exposure over a lifetime. As a result, the affinity of lead for bone would suggest that lead levels in bone, rather than lead levels in blood, provide more relevant predictive information for some health effects associated with long term exposure.

Acute Toxicity

Oral

The members of this group are not individually listed in HSIS and therefore, by default, they are covered by the 'lead and lead compounds' hazard classification with the risk phrase ‘Harmful if swallowed’ (Xn; R22) in HSIS (Safe Work Australia).

However, the rat oral median lethal dose (LD50) for ***dibasic lead phthalate*** **(CAS No: 69011-06-9)** was reported to be > 2000 mg/kg (REACH). Specific signs of toxicity were not reported.

While the experimental data do not support the classification for dibasic lead phthalate, data available from observations in humans do support the generic 'lead and lead compounds' classification and are presented in the following sections.

Dermal

Dibasic lead phthalate **(CAS No: 69011-06-9)** exhibited low acute toxicity in animal tests as evidenced by reported LD50s in rats of > 2000 mg/kg bw (REACH).

Inhalation

The members of this group are not individually listed in HSIS and therefore, by default, they are covered by the 'lead and lead compounds' hazard classification with the risk phrase ‘Harmful by inhalation’ (Xn; R20) in HSIS (Safe Work Australia). While no data are available for the lead compounds in this group, data available from observations in humans support thisgeneric 'lead and lead compounds' classification.

Although a study using lead oxide reported a median lethal concentration (LC50) of > 5.05 mg/L for rats (REACH), this evidence is insufficient to warrant removal of the generic classification for the chemicals in this group.

Observation in humans

In this section, route specific data are not provided but exposure is reported in terms of absorbed dose. The concentration of lead in the blood is the most commonly reported value. However lead in bone, hair and teeth are also reported in the literature.

Adult Exposure

The majority of the data have been collected from accidental or intentional exposure via ingestion or inhalation, and there are rich data regarding the dose-effect in humans (NICNAS 2007; ATSDR 2007). Exposure can cause encephalopathy (the signs of which include: hyperirritability, ataxia, convulsions, stupor and coma) in addition to gastrointestinal effects such as colic (displayed as: abdominal pain, constipation, cramps, nausea, vomiting, anorexia and weight loss) (ATSDR 2007; WHO 1995). It was recorded that signs of acute toxicity were observed in adults with a PbB level ranging from 50 - 300 µg/dL. However, that is challenged in a more recent study that only noted signs of encephalopathy in adults with PbB levels greater than 460 µg/dL (NICNAS 2007; ATSDR 2007).

Colic is indicative of gastrointestinal impact and is typically displayed as an early effect of exposure to lead (NICNAS 2007; ATSDR 2007). Colic has been noted in individuals exposed to high levels of lead and can be evident as a result of occupational exposure where workers generally register PbB levels between 100 – 200 µg/dL, although symptoms have been reported by workers with PbB levels between 40 – 60 µg/dL.

Exposure to lead has been reported to cause proximal renal tubular damage (NICNAS 2007).

Paediatric Exposure

Data were compiled from a paediatric population regarding the dose-response after acute exposure to lead. Signs of encephalopathy were noted in children with PbB levels between 90 – 800 µg/dL. The mean value reported for PbB levels related to death (327 µg/dL) is similar to that noted for encephalopathy (330 µg/dL). Gastrointestinal effects (abdominal pain, constipation, cramps, nausea, vomiting, anorexia and weight loss) were reported at PbB levels between 60 – 450 µg/dL. Data collected from additional reports indicate that acute encephalopathy was noted in children with PbB levels of 80 – 100 µg/dL and infants at PbB levels of 74.5 µg/dL (NICNAS, 2007).

In paediatric populations, acute colic has also been reported as an effect of poisoning associated with exposure to lead and is noted to occur when the PbB level is greater than or equal to 60 µg/dL (NICNAS 2007; ATSDR 2007). In addition, it has been reported that exposure to lead can inhibit the formation of the haem-containing protein cytochrome P450 (NICNAS, 2007).

Corrosion / Irritation

Skin Irritation

In general, lead compounds are not considered irritating to skin (REACH). No effects were reported in skin irritation assays in rabbits citing OECD TG 404 using ***dibasic lead phthalate*** **(CAS No: 69011-06-9)**.

Eye Irritation

In general lead compounds were not reported to be irritating to eyes or having caused serious eye damage (REACH). In an eye irritation assay (OECD TG 405) in rabbits (New Zealand White) using ***dibasic lead phthalate*** **(CAS No: 69011-06-9)**, all symptoms reported were fully reversible within seven days.

Observation in humans

No studies were located that recorded skin or eye irritation in humans as a result of exposure to lead compounds.

Sensitisation

Skin Sensitisation

Several lead compounds, including ***dibasic lead phthalate*** **(CAS No: 69011-06-9)**, were reported to be non-sensitisers (LDAI, 2008). It was reported that the compounds gave negative results for skin sensitisation in guinea pigs when tested according to OECD TG 406.

Observation in humans

Although altered immune parameters were described in occupational and paediatric groups that were exposed to lead, there were no reports of skin or respiratory sensitisation to lead in humans (ATSDR, 2007).

Repeated Dose Toxicity

Oral

The compounds in this group are not individually listed in HSIS and therefore, by default, are covered by the generic 'lead and lead compounds' hazard classification with the risk phrase ‘Danger of cumulative effects' (R33) in HSIS (Safe Work Australia). While no data are available of the chemicals in this group specifically, data available from animal studies and observations in humans support the 'lead and lead compounds' generic classification and are presented in the following sections.

In a study using lead acetate, the lowest observed adverse effect level (LOAEL) of 200 ppm (corresponding to PbB levels of 40-60 mg/dL) was derived based on body and kidney weights (REACH).

In a repeated dose toxicity study with Sprague Dawley (SD) rats that followed the guidelines set out in a US EPA chronic feeding study, lead acetate was administered via drinking water (ad libitum) to 18 male rats per dose group at 0, 200, 500 or 1000 ppm per day for four, eight or 12 weeks. Decreased body weight and increased kidney weight as a percentage of body weight were reported at all dose ranges from four weeks (REACH).

Dermal

No significant adverse effects were reported following repeated dermal exposure to several lead compounds (REACH).

In a report available on repeat dose toxicity during dermal exposure, rats were exposed to lead compounds for 24 hours. The test groups had lead compounds applied either directly to the skin or to skin that had been mechanically injured. Dermal absorption of lead was shown to occur in all the test groups. However, comparatively greater absorption of lead was reported in the groups where the skin had been mechanically injured.

Inhalation

No significant adverse effects were reported following repeated inhalation exposure to lead nitrate (REACH).

Aerosolised lead nitrate was administered to mice (Swiss Webster) via inhalation at 2.5 mg/m³ per day for 14 or 28 days. It was determined, considering total retention of the inhaled lead, that each mouse received a dose of 80 µg/day of lead.

A statistically significant reduction in the relative size of the spleen and thymus in both test groups was reported. Increased lung weight was noted in both test groups and an increase lead concentration was reported in the liver, lung and kidney, although the 28 day group was noted to show a greater concentration than the 14 day group. There were no apparent differences in body weight and food consumption noted for either test group.

Observation in humans

Lead has multiple modes of action in biological systems; as a result, any system or organ in the body can potentially be affected by lead exposure. For the purposes of this report, the effects of lead toxicity on the most sensitive target organs have been identified and summarised (NICNAS, 2007; ATSDR 2007).

Neurological Effects

Lead encephalopathy is considered the most severe neurological effect of lead exposure in adults. Occupational lead exposure has also been linked to neurotoxicity and studies have shown that the following signs and symptoms have been noted in those recorded to have PbB levels of between 40 – 120 µg/dL: malaise, forgetfulness, irritability, lethargy, headache, fatigue, impotence, decreased libido, dizziness, weakness, paraesthesia, visual motor coordination impairment, cognitive performance impairment, decreased reaction time, mood and coping ability as well as affecting memory.

Haematological Effects

Lead exposure impacts on the haematological system by inhibiting haem synthesis and decreasing the lifespan of erythrocytes, which results in the onset of microcytic and hypochromic anaemia (NICNAS, 2007). It has been estimated that the PbB threshold for a decrease in haemoglobin to be seen in occupationally exposed adults is 50 µg/dL. For children the PbB threshold is estimated to be 40 µg/dL.

Cardiovascular Effects

Studies investigating the effect of PbB on blood pressure in humans are not conclusive (NICNAS, 2007; ATSDR, 2007). The cardiovascular endpoint of concern for humans when exposed to low levels of lead is an increase in systemic blood pressure. Longitudinal occupational studies investigating the possible

relationship between low level lead exposure and blood pressure have been undertaken, with mixed results. Subsequently, based on the available literature, it is suggested that a relationship between low level exposure to lead and increased systemic blood pressure cannot be determined (NICNAS, 2007).

Renal Effects

Nephrotoxicity associated with lead is characterised by proximal tubular nephropathy, glomerular sclerosis and interstitial fibrosis. The deterioration in renal function is characterised by enzymuria, proteinuria and an impaired ability to transport organic anions and glucose, in addition to a decreased glomerular filtration rate. Studies summarised in ATSDR (2007) indicate that an increase in nephrotoxicity is proportional to an increase in PbB levels. Effects on glomerular filtration are reported at or below 20 µg/dL, enzymuria and proteinuria are reported at equal to or greater than 30 µg/dL and severe deficits in function and pathological changes are reported in association with PbB levels ≥50 µg/dL.

Genotoxicity

Lead compounds are considered genotoxic for mammalian cells.

The genotoxic effects of lead were reviewed and presented by the Agency for Toxic Substances and Disease Registry (ATSDR 2007). The majority of the in vitro point mutation tests in bacteria were negative, while mammalian clastogenicity tests were generally positive.

It was reported that in bacterial reverse mutation assays, lead was negative both with and without metabolic activation (REACH). In vitro chromosomal aberration tests using Chinese hamster ovary (CHO) cells and human lymphocytes were positive without metabolic activation. An in vivo micronucleus assay using human peripheral lymphocytes (from those working with lead compounds) was positive below the maximum tolerated dose.

Carcinogenicity

The members of this group are not individually listed in HSIS and therefore, by default, are covered by the generic 'lead and lead compounds' classification which does not classify the compounds as carcinogenic (Safe Work Australia).

However, a review conducted by the International Agency for Research on Cancer (IARC) in 1980, which was updated in 1987 and again in 2006, resulted in the classification of inorganic lead compounds as probably carcinogenic to humans (Group 2A) (IARC, 1980; IARC, 1987; IARC, 2006).

A subsequent review by the International Lead Association (LDAI, 2008) concluded that there is consistent evidence from studies with soluble lead compounds or bioavailable lead compounds to support the conclusion that lead compounds are probably carcinogenic to humans.

This evidence is sufficient to classify the chemicals in this group as potential carcinogens.

Reproductive and Developmental Toxicity

The lead compounds in this group are not listed in HSIS and therefore, by default, are covered by the generic 'lead and lead compounds' hazardous classification with the risk phrases ‘Possible risk of impaired fertility’ (R62) and ‘May cause harm to the unborn child’ (R61) in HSIS (Safe Work Australia). The available data support these classifications.

In a reproductive and developmental toxicity screening test in SD rats, lead acetate was administered via drinking water to nine females at 0.6 % weight per volume (w/v) (equivalent to 502 mg/kg bw/day) at gestation days 5 – 21 (Ronis et al, 1996; LDAI, 2008). A stillbirth rate of 19 % was recorded in the test group compared with a 2 % rate noted in the control group. The dams and offspring had PbB levels > 200 µg/dL.

In a subsequent reproductive and developmental toxicity screening test in SD rats, lead acetate was administered via drinking water to 10 females at 0.05 % w/v, eight females at 0.15 % w/v and nine females at 0.45% w/v, during gestation days 5 – 21 (Ronis et al, 1998). Stillbirth rates of 3(±3), 10(±6) and 28(±8) % were recorded for increasing dose groups respectively. This was compared with a 4(±3) % rate noted in the control group. At birth, the male pups had PbB levels of 40(±1), 83(±8) and 120(±120) µg/dL for increasing dose groups respectively, while the female pups had PbB levels of 42(±7), 67(±16) and 197(±82) µg/dL. A developmental LOAEL of 0.05% (equivalent to 42 mg/kg bw/day) was reported for this study (LDAI, 2008)..

Reproductive toxicity observations in humans

Recent studies have investigated the effect of lead exposure in occupational groups and general populations living near industrial plants. Although evidence reported is predominantly qualitative and dose-effect relationships have largely not been established (NICNAS, 2007; WHO, 1995), it has been suggested that moderately high PbB levels could result in spontaneous abortion, pre-term delivery, alterations in sperm and decreasing male fertility (ATSDR, 2007).

Developmental toxicity observations in humans

Data pertaining to low level exposure to lead contributing to developmental toxicity in infants and young children were recently reviewed. Consensus exists between the reports which suggest that PbB levels greater than 10 µg/dL can affect paediatric intellectual development (ATSDR, 2007; Donovan, J; 1996). In addition, data regarding the effects on children of higher levels of lead exposure were reviewed. Although neurobehavioural deficits were reported in children with PbB levels less than 10 µg/dL, there is uncertainty attached to these estimates of reported effects (ATSDR, 2007). Even so, the US Centers for Disease Control and Prevention (CDC) has a reference level of 5 µg/dL, above which they recommend that public health action be initiated (CDC).

Risk Characterisation

Critical Health Effects

The main critical effects to human health are reproductive and developmental toxicity, potential genotoxicity and limited evidence of carcinogenicity. The chemicals are also expected to cause adverse effects by repeated exposure and may be harmful following acute exposure via inhalation and the oral route.

Public Risk Characterisation

While only site-limited uses have been identified for the chemicals in this group, the use of lead and lead compounds in products available to the public in Australia is restricted as listed in the Poisons Standard (SUSMP, 2012). These restrictions will prevent risks from domestic use of these compounds. It should also be noted that, given these restrictions, any overseas use of lead and lead compounds in paints can be considered to not be relevant to Australia.

Historical use of lead compounds in surface coatings suggests that the potential for the public to be exposed, through flaking paint and during home renovation, still exists. While it is possible that the public will be exposed to lead or lead compounds, the risk can be managed by following appropriate guidelines.

Occupational Risk Characterisation

Given the critical systemic long-term health effects, the chemical may pose an unreasonable risk to workers unless adequate control measures to minimise 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.

NICNAS Recommendation

Current risk management measures are considered adequate for the protection of 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

Current restrictions control the use of lead and lead compounds in cosmetics, paint, tinters, inks or ink additives, which effectively reduces the risk of public exposure. The availability and permissible lead content in products, such as paint, are regulated in terms of availability and concentration (SUSMP, 2012). Products that historically contained lead or lead compounds still pose an exposure risk to the public due to their existence in the public domain.

The National Health and Medical Research Council (NHMRC) of Australia has published recommendations regarding how the public can manage exposure to lead by mitigating the risk (NHMRC, 2009). Methods for the safe approach to painting a house (when there is a likelihood of lead paint having been used previously) has been published by the Department of Sustainability, Environment, Water, Population and Communities (DSEWPaC, 2009).

Work Health and Safety

The health risk to workers from these chemicals is controlled when correct classification and labelling are considered, and adequate control measures to minimise occupational exposure and protective clothing are implemented. Safe Work Australia (SWA) encourages working safely with lead and promotes the *National*

Code of Practice for the Control and Safe Use of Inorganic Lead at Work [NOHSC: 2015 (1994)] and the *National Standard for the Control of Inorganic Lead at Work [NOHSC:1012 (1994)]*. These Codes of Practice, in addition to the *Model Work Health Safety Regulations, 2011* are available from the SWA website.

The chemicals are recommended for classification and labelling under the current Approved Criteria and adopted Globally Harmonized System (GHS) of Classification and Labelling of Chemicals as below. This does not consider classification of physical hazards and environmental hazards.

Please note that the acute oral toxicity classification does not apply to chemicals for which experimental data do not support this classification (***dibasic lead phthalate*** **CAS No. 69011-06-9).**

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Harmful if swallowed (Xn; R22)* Harmful by inhalation (Xn; R20)*	Harmful if swallowed - Cat. 4 (H302) Harmful if inhaled - Cat. 4 (H332)
Repeat Dose Toxicity	Danger of cumulative effects (R33)*	May cause damage to organs through prolonged or repeated exposure - Cat. 2 (H373)
Genotoxicity	Muta. Cat 3 - Possible risk of irreversible effects (Xn; R68)	Suspected of causing genetic defects - Cat. 2 (H341)
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 1 - May cause harm to the unborn child (T; R61)* Repro. Cat 3 - Possible risk of impaired fertility (Xn; R62)*	May damage the unborn child. Suspected of damaging fertility - Repr. 1A (H360Df)

[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 label instructions.

Advice for industry

Control measures

Control measures to minimise the risk from oral and inhalation 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

Agency for Toxic Substances & Disease Registry (ATSDR) Toxicological Profile for Lead. Accessed September 2012 at http://www.atsdr.cdc.gov/toxprofiles/tp13.pdf

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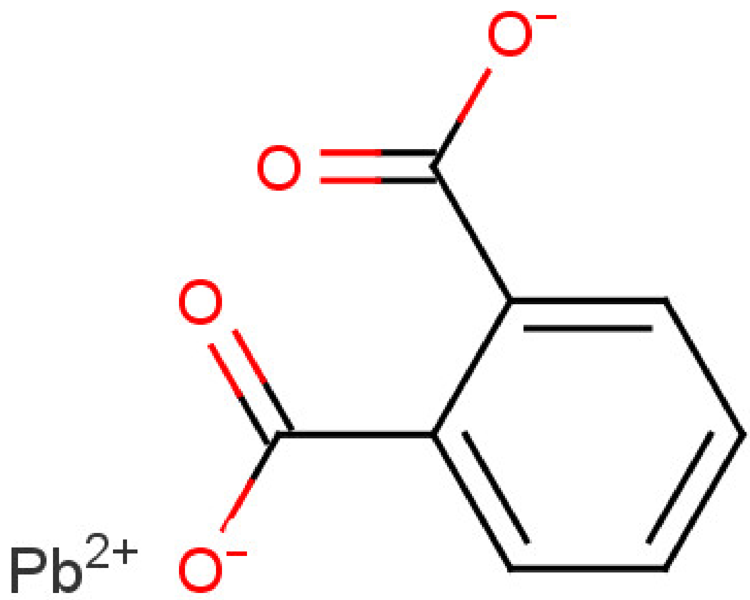
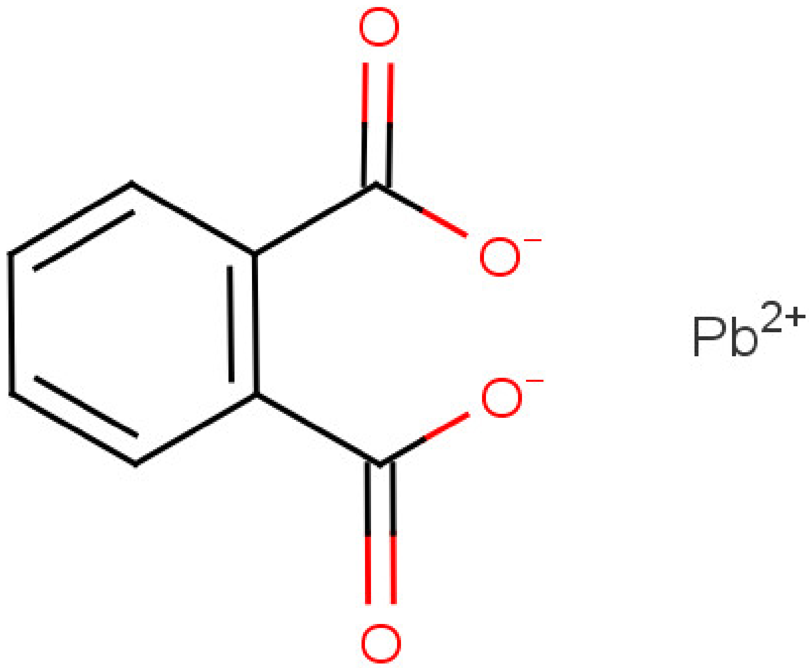
Last Update 07 February 2014

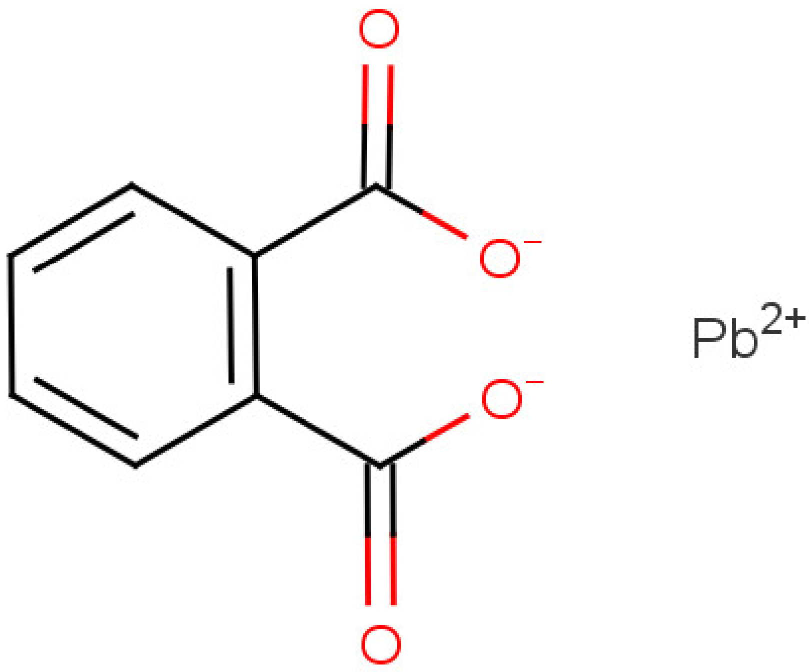
Chemical Identities

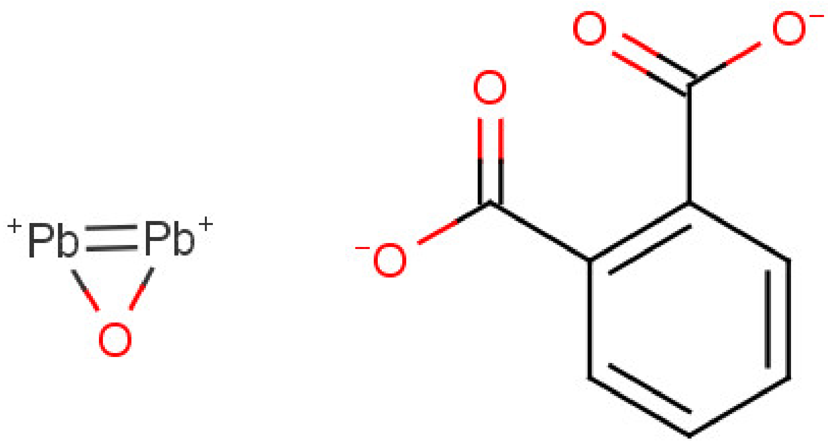
Chemical Name in the Inventory and Synonyms	**1,2-Benzenedicarboxylic acid, lead(2+) salt (1:1)** Lead phthalate Lead(2+) phthalate (1:1) 1,2-Benzenedicarboxylic acid, lead(2+) salt (1:1) Lead phthalate, dibasic Phthalic acid, lead(2+) salt (1:1)
CAS Number	6838-85-3
Structural Formula	
Molecular Formula	C8H6O4.Pb
Molecular Weight	371.32

Chemical Name in the Inventory and Synonyms	**1,2-Benzenedicarboxylic acid, lead salt** Lead phthalate
CAS Number	16183-12-3

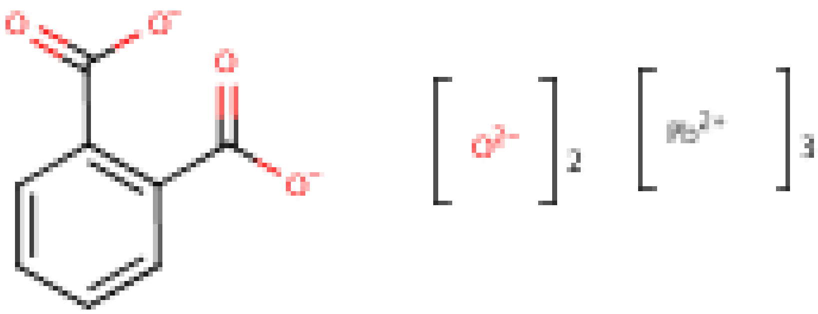
Structural Formula	
Molecular Formula	C8H6O4.xPb
Molecular Weight	371.32

Chemical Name in the Inventory and Synonyms	**1,2-Benzenedicarboxylic acid, lead(2+) salt** Phthalic acid, lead(2+) salt
CAS Number	18608-34-9
Structural Formula	

	
Molecular Formula	C8H6O4.xPb
Molecular Weight	371.32

Chemical Name in the Inventory and Synonyms	**Lead, [1,2-benzenedicarboxylato(2-)]oxodi-** Dibasic lead phthalate (Phthalato(2-))oxodilead Lead, (1,2-benzenedicarboxylato(2-))oxodi-
CAS Number	57142-78-6
Structural Formula	

Molecular Formula	C8H4O5Pb2
Molecular Weight	594.52

Chemical Name in the Inventory and Synonyms	**Lead, [1,2-benzenedicarboxylato(2-)]dioxotri-** Dibasic lead phthalate [phthalato(2-)]dioxotrilead Lead, 1,2-benzenedicarboxylato(2-)dioxotri- [1,2-benzendicarboxylato]dioxotri-Lead 1,2-Benzenedicarboxylic acid, lead complex
CAS Number	69011-06-9
Structural Formula	
Molecular Formula	C8H4O6Pb3
Molecular Weight	

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