

Explosive lead salts: Human health tier II assessment



11 April 2014

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Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
1,3-Benzenediol, 2,4-dinitro-, lead(2+) salt (1:1)	13406-89-8
Lead azide (Pb(N₃)₂)	13424-46-9
1,3-Benzenediol, 2,4,6-trinitro-, lead(2+) salt (1:1)	15245-44-0
1,3-Benzenediol, 2,4,6-trinitro-, lead(2+) salt, monobasic	51331-05-6
Lead, (2-methyl-4,6-dinitrophenolato-O1) (nitrato-O)-.mu.-oxodi-, monohydrate	79357-62-3

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

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ACRONYMS & ABBREVIATIONS

Grouping Rationale

The compounds in this group are organic and inorganic salts of lead that function as initiating or primary explosives. These types of explosives decompose through weak stimulus, such as mechanical shock or spark, and produce a detonation wave. Detonation of these primary explosives can then initiate a secondary explosive such as pentaerythritol tetranitrate (PETN) or nitroglycerin (Boileau et al., 2000; Meyer et al., 2007).

Import, Manufacture and Use

Australian

Based on a report from the Department of Defence, lead styphnate (CAS No. 15245-44-0) has been shown to have site-limited use as a component of ammunition for military use (Spear et al., 1987).

International

The following international uses have been identified through European Union Registration, Evaluation and Authorisation of Chemicals (EU REACH) dossiers; the European Chemical Agency (ECHA); Galleria Chemica; and the US National Library of Medicine's Hazardous Substances Data Bank (HSDB).

Lead azide (CAS No. 13424-46-9) and lead styphnate (CAS No. 15245-44-0) have reported commercial use including:

- in manufacturing initiating explosives in primers (percussion caps for small calibre and rifle ammunition) and in detonators (civilian and military use).

Mixtures composed of controlled crystals of lead azide (CAS No. 13424-46-9) and lead styphnate (CAS No. 15245-44-0) are essential components of a detonator charge. As pure lead azide is highly sensitive to shock making transportation of the chemical difficult, technical grade lead azide is usually manufactured to contain only 92–96 % lead azide. For this reason, lead styphnate is added to improve the explosivity of lead azide in ammunition or explosive articles (Boileau et al., 2000; Meyer et al., 2007).

Lead dinitroresorcinate (CAS No. 13406-89-8) has reported commercial use including:

- in manufacturing electric fuse heads and similar electric igniters.

Restrictions

Australian

Lead and lead compounds are listed in the Poisons Standard (the Standard for the Uniform Scheduling of Medicines and Poisons—SUSMP) (SUSMP, 2013) under the following Schedules:

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 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 high.'

'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.'

In addition, under the Customs (Prohibited Imports) Regulations 1956, importing cosmetic products containing more than 250 mg/kg (0.025 % w/w) of lead or lead compounds (calculated as lead), except products containing more than 250 mg/kg of lead acetate designed for use in hair treatments, is prohibited unless written permission is granted by the Minister (Australian Government, 2013).

Further information regarding the restriction of lead in cosmetics is provided in the NICNAS Existing Chemicals information sheet: *Lead in cosmetics* (November, 2008).

Certain lead compounds in this group are listed in the *Australian Code for the Transport of Explosives by Road and Rail* (3rd edition) as follows (SafeWork Australia):

- the transport of lead azide, lead styphnate, and 2,4-dinitroresorcinol are '*prohibited, when neither desensitised nor phlegmatised, without Competent Authority approval*';
- lead azide and lead styphnate, when wetted with less than 20 % water, or mixture of alcohol and water, by mass, are both classified as Class 1.1D explosives (UN No 0129 and 0130, respectively).

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

Lead compounds appear on the following:

- Health Canada list of prohibited and restricted cosmetic ingredients (The 'Hotlist').
- The EU Cosmetics 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.
- The Thailand Cosmetic Act—Prohibited substances.

Lead azide (CAS No. 13424-46-9) and lead styphnate (CAS No. 15245-44-0) are listed on the candidate list of substances of very high concern (SVHC) for eventual inclusion in Annex XIV (ECHA, 2011). In the EU, companies may have legal obligations if the chemical that they produce, supply, or use is included on the candidate list whether on its own, in mixtures, or present in articles.

Existing Worker Health and Safety Controls

Hazard Classification

Lead azide (CAS No. 13424-46-9) and lead styphnate (CAS No. 15245-44-0) are individually listed in the Hazardous Substances Information System (HSIS) and classified as hazardous, with the following risk phrases for human health (Safe Work Australia):

Xn; R20/22 (Harmful by inhalation and if swallowed)

R33 (Danger of cumulative effects)

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

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

Although the remaining lead compounds are not individually listed in HSIS, by default, they are covered by the generic 'lead compounds' classification as hazardous with the same risk phrases as above, for human health.

Exposure Standards

Australian

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

Time weighted average (TWA): 0.15 mg/m³ 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 = 0.05 mg/m³ [Bulgaria, Canada, China, Italy, Malaysia, USA]

TWA = 0.10 mg/m³ [Austria, New Zealand, Republic of South Africa, Sweden]

TWA = 0.15 mg/m³ [Argentina, Egypt, EU (Directive 98/24/EC), Malta, Singapore]

TWA = 0.20 mg/m³ [Thailand]

STEL: 0.10 mg/m³ [Austria]

STEL: 0.15 mg/m³ [Canada]

STEL: 0.45 mg/m³ [Argentina, Egypt]

Health Hazard Information

Following synthesis, these chemicals are tightly controlled within articles such as detonators. The anion component of these chemicals will be destroyed upon ignition. Thus, human exposure to these chemicals is expected to be very low. However, exposure to the lead component, through lead ignition byproducts and residuals, was shown to occur (Gelberg & Depersis, 2009). Thus, the main concern regarding effects on human health is expected to be driven by the lead component (cation) of these compounds. While there are limited data available on these specific chemicals, data sources for determining the hazard of 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 through inhalation (NICNAS, 2007).

When ingested, different factors influence the absorption of inorganic lead compounds in the human gastrointestinal tract, 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 are estimated to absorb up to 10 %. While soluble lead compounds are expected to be bioavailable following ingestion, results from rodent feeding studies indicate that water-insoluble lead compounds are also bioavailable once exposed to stomach acids (LDAI, 2008). This route of absorption can also be dependent on particle size, with smaller particles being absorbed more readily than larger ones.

In an oral repeated dose toxicity study, rats were dosed with 0, 200, 500 or 1000 ppm lead acetate for four, eight or 12 weeks. Measured blood lead concentrations (PbB) ranged from 40–100 µg/dL. Kidney lead levels were highest at four weeks exposure to the chemical. For all test groups, urinary lead excretion was highest at four weeks exposure, then decreased with continued exposure to lead (REACH).

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

Absorption through the skin (dermal) has been shown to be 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. In another study, where 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 has been shown to accumulate in bone; this accumulation is considered to be a biomarker for long-term lead exposure. Lead stored in bone can be released into the blood after exposure has ceased. When in the blood, 99 % of lead is bound to proteins within red blood cells (erythrocytes) (NICNAS, 2007). Inorganic lead is distributed in the body independently of the source compound and route of exposure. Within bone, distribution is not uniform and lead accumulates in areas that are undergoing active calcification at the time of exposure. The spatial distribution of lead in bone is similar between children and adults, although adults generally have a higher concentration (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.

While PbB concentration is a reflection of recent exposure, it does not capture the more significant impact and slower elimination kinetics of the chemical in bone. 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 (ASTDR, 2007).

In 2009, a survey was conducted on 598 individuals who were exposed to lead from target shooting. The route of exposure was likely to be through inhaling lead particles as a consequence of the combustion of lead azide and/or lead styphnate in priming compounds found in ammunition. It was found that individuals with non-occupational exposure (hobby-related activities) had higher levels of lead in their blood, ≥ 60 µg/dL, compared with individuals who had occupational exposure (range employees, protective services employees, and tradesmen) (Gelberg & DePersis, 2009).

Acute Toxicity

Oral

All the compounds in this group are either listed with individual classifications or covered by the generic 'lead and lead compounds' hazard classification with the risk phrase 'Harmful if swallowed' (Xn; R22) in HSIS (Safe Work Australia).

While there are no experimental data available specific to these chemicals, data from observations in humans exposed to lead compounds are presented in the following sections. In the absence of more comprehensive information, there is insufficient evidence to support a recommendation to amend the classification for this group of chemicals.

Dermal

Several lead compounds were reported to exhibit low acute toxicity in animal tests. Dermal median lethal dose (LD50) values in rats are reported to be >2000 mg/kg bw (REACH).

Inhalation

All the compounds in this group are either listed with individual classifications or covered by the generic 'lead and lead compounds' hazard classification with the risk phrase 'Harmful by inhalation' (Xn; R20) in HSIS (Safe Work Australia).

While there are no experimental data available specific to these chemicals, data from observations in humans exposed to lead compounds are presented in the following sections. In the absence of more comprehensive information, there is insufficient evidence to support a recommendation to amend the classification for this group of chemicals.

Observation in humans

The majority of the data have been collected from accidental or intentional exposure following ingestion or inhalation, and there are rich data regarding the dose-effect in humans (ASTDR, 2007; NICNAS, 2007).

Exposure can cause encephalopathy (the signs of which include hyperirritability, ataxia, convulsions, stupor and coma) in addition to gastrointestinal effects such as colic (the effects can be displayed as: abdominal pain, constipation, cramps, nausea, vomiting, anorexia and weight loss) (WHO, 1995; ASTDR, 2007). It was recorded that signs of acute toxicity were observed in adults with a PbB (blood lead concentration) level ranging from 50–300 µg/dL. However, this is challenged in a more recent study, which only noted signs of encephalopathy in adults with PbB levels >460 µg/dL (ASTDR, 2007; NICNAS, 2007).

Colic is indicative of an effect on the gastrointestinal system and is typically displayed as an early sign of exposure to lead (ASTDR, 2007; NICNAS, 2007). Colic has been noted in individuals exposed to high levels of lead and has been linked to 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 in the kidney (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 >60 µg/dL (ASTDR, 2007; NICNAS, 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 to irritate the skin. No effects were reported in skin irritation assays in rabbits citing OECD Test Guideline (TG) 404 using lead oxide, dibasic lead phosphite and dibasic lead phthalate (REACH).

Eye Irritation

In general, lead compounds were not reported to irritate the eyes or having cause serious eye damage. In eye irritation assays (OECD TG 405) using New Zealand White rabbits, exposure to dibasic lead phthalate or lead oxide did not result in classifiable irritation effects (REACH). All symptoms reported were fully reversible within the seven day and 72 hour observation periods, respectively.

Sensitisation

Skin Sensitisation

Several lead compounds were reported to be non-sensitisers (REACH). It was reported that the compounds produced 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 (ASTDR, 2007).

Repeated Dose Toxicity

Oral

All the compounds in this group are either listed with individual classifications or covered by the generic 'lead and lead compounds' hazard classification with the risk phrase 'Danger of cumulative effects' (R33) in HSIS (Safe Work Australia).

In an experiment conducted on rats (10 animals/dose group), lead azide (CAS No. 13424-46-9) was orally administered at 20, 40, or 60 mg/animal/day for 44 weeks (Fairhall et al., 1943). All of the animals died in the 60 and 40 mg groups by week nine and 14, respectively. Lead was detected in the liver, kidney, and bone tissues (these were the only organs assessed). No other pathological examinations were performed.

While limited data are available for the chemicals in this group, data available from animals studies on other lead compounds, and observations in humans are provided in the following sections. In the absence of more comprehensive information, there is insufficient evidence to support a recommendation to amend the classification for this group of chemicals.

A lowest observed adverse effect level (LOAEL) of 200 ppm (corresponding to PbB levels of 40–60 µg/dL) was derived for lead acetate from a repeated dose toxicity study in Sprague Dawley (SD) rats following the guidelines set out in a US EPA chronic feeding study (REACH). Lead acetate was administered in drinking water (which was freely accessible [*ad libitum*]) to male rats (18 animals/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 at four weeks of exposure.

Dermal

While no data are available for the lead compounds in this group, no significant adverse effects were reported following repeated dermal exposure to several other lead compounds.

In a report available on repeated dose toxicity during dermal exposure, rats were exposed to lead acetate, lead oleate, lead arsenate or tetraethyl lead for 24 hours (REACH). 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 both test groups. However, comparatively greater absorption of lead was reported in the groups where the skin had been mechanically injured.

Inhalation

While no data are available for the chemicals in this group, no significant adverse effects were reported following repeated inhalation exposure to lead nitrate (REACH).

Aerosolised lead nitrate was administered to mice (Swiss Webster) by inhalation at 2.5 mg/m³ per day for 14 or 28 days. It was determined, considering the 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 when compared with the control group. Increased lung weight was noted in both test groups and an increase in 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. Therefore 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 (ASTDR, 2007; NICNAS, 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 adversely affecting memory.

Haematological effects

Lead exposure affects 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 in occupationally exposed adults for a decrease in haemoglobin to be measured is 50 µg/dL. For children the threshold is estimated to be PbB 40 µg/dL.

Cardiovascular effects

Studies investigating the effect of PbB on blood pressure in humans are not conclusive (ASTDR, 2007; NICNAS, 2007). The cardiovascular 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 exposure is characterised by abnormalities in the kidney (specifically in the proximal tubule cells), glomerular sclerosis (hardening or scarring) and interstitial fibrosis (abnormal formation of fibrous tissue). The deterioration in renal function is characterised by increased levels of enzymes and proteins in excreted urine (enzymuria and 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 ≤20 µg/dL; enzymuria and proteinuria are reported at >30 µg/dL; and severe deficits in function and pathological changes are reported in association with PbB levels ≥50 µg/dL.

Genotoxicity

In general, lead compounds are considered genotoxic to mammalian cells.

The genotoxic effects of lead were reviewed and presented by the 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). However, 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

A review conducted by the International Agency for Research on Cancer (IARC) in 1980, which was updated in 1987 and again in 2006, indicated that there was sufficient evidence in experimental animals and limited evidence in humans for the carcinogenicity of inorganic lead compounds (IARC, 1980; IARC, 1987; IARC, 2006). The review resulted in the classification of inorganic lead compounds as probably carcinogenic to humans (Group 2A).

A subsequent review by the International Lead Association (LDAI, 2008) concluded that there is consistent evidence from studies in rodents that soluble lead compounds, or those that are considered bioavailable, are carcinogenic in animals; notably, reproducible renal tumours have been reported in male rats following administration of high levels of lead in food or water.

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

Reproductive and Developmental Toxicity

All the compounds in this group are either listed with individual classifications or covered by the generic 'lead and lead compounds' hazard 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).

In a reproductive and developmental toxicity screening test in SD rats, lead acetate was administered in drinking water to nine females at 0.6 % weight per volume (w/v) (equivalent to 502 mg/kg bw/day) on 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 in the test group had PbB levels >200 µg/dL.

In a subsequent reproductive and developmental toxicity screening test in SD rats, lead acetate was administered in 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, on 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 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 in general populations living near industrial plants. Although the evidence reported is predominantly qualitative and dose-effect relationships have largely not been established (WHO, 1995; NICNAS, 2007), it has been suggested that moderately high PbB levels in humans could result in spontaneous abortion, pre-term delivery, alterations in sperm and decreased male fertility (ASTDR, 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 in humans >10 µg/dL can affect paediatric intellectual development (Donovan, J, 1996; ASTDR, 2007).

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 <10 µg/dL, there is uncertainty attached to these estimates of reported effects (ASTDR, 2007). Even so, the US Centers for Disease Control and Prevention (CDC) has a reference level of 5 µg/dL, above which it is recommended that public health action be initiated (CDC).

Risk Characterisation

Critical Health Effects

In addition to the risk associated with the detonation of these chemicals for which high level exposure controls are already in place, the critical health effects for risk characterisation include systemic long-term effects (reproductive and developmental toxicity, carcinogenicity and mutagenicity). The chemical may also cause harmful effects following repeated exposure and harmful systemic effects following a single exposure.

Public Risk Characterisation

The use of lead and lead compounds in products available to the public in Australia is restricted; the restrictions are listed in the Poisons Standard (SUSMP, 2013).

It is anticipated that the potential public exposure to lead from ammunition and explosive applications will depend on their proximity to the application sites and/or handling articles containing explosive lead compounds. While it is possible that the public will be exposed to lead or lead compounds, the risk can be managed by following the appropriate guidelines (see Recommendation section).

Occupational Risk Characterisation

Given the critical 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

Assessment of the chemical is considered to be sufficient, providing 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

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, 2013). 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) have been published by the Department of Sustainability, Environment, Water, Population and Communities—now the Department of the Environment (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)], the *National standard for the control of inorganic lead at work* [NOHSC:1012 (1994)] and the *Australian Code for the Transport of Explosives by Road and Rail* (3rd edition). These codes of practice, in addition to the Model Work Health and Safety Regulations, 2011, are available from the SWA website.

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

If empirical data become available indicating that a lower (or higher) classification is appropriate, this may be used to amend the default classification for that chemical.

Hazard	Approved Criteria (HSIS) ^a	GHS Classification (HCIS) ^b
Acute Toxicity	Harmful if swallowed (Xn; R22)*	Harmful if swallowed - Cat. 4 (H302)
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)
Reproductive and Developmental Toxicity	Repro. Cat 3 - Possible risk of impaired fertility (Xn; R62)* Repro. Cat 1 - May cause harm to the unborn child (T; R61)*	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 the instruction on the label.

Advice for industry

Control measures

Control measures to minimise the risk from exposure to the chemicals 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.

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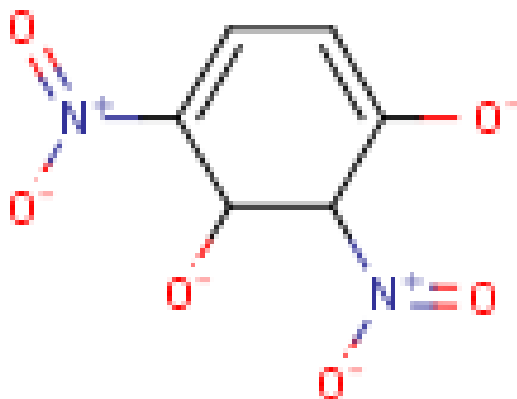
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Last Update 11 April 2014

Chemical Identities

Chemical Name in the Inventory and Synonyms	1,3-Benzenediol, 2,4-dinitro-, lead(2+) salt (1:1) 2,4-Dioxo-3-plumbabicyclo[3.3.1] Resorcinol, 2,4-dinitro-, lead(2+) salt (1:1) Lead(2+), 2,4-dinitroresorcinolate Lead(II), 2,4-dinitrobenzene-1,3-diolate
CAS Number	13406-89-8
Structural Formula	



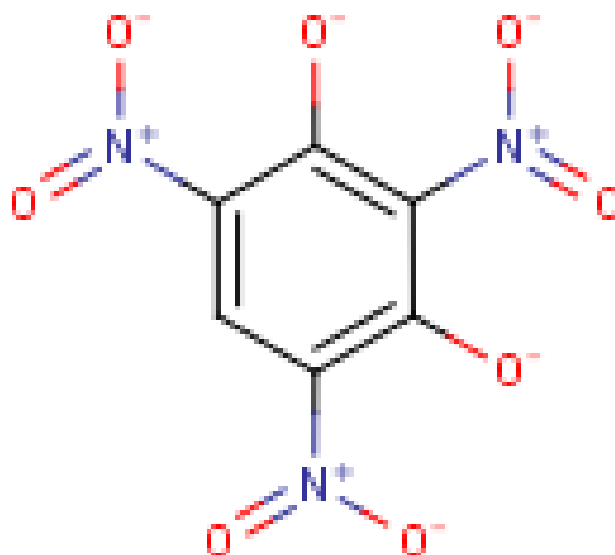
Molecular Formula	C6H4N2O6.Pb
Molecular Weight	407.31

Chemical Name in the Inventory and Synonyms	Lead azide (Pb(N3)2) Lead diazide Plumbous azide
CAS Number	13424-46-9
Structural Formula	



Molecular Formula	N6Pb
Molecular Weight	291.24

Chemical Name in the Inventory and Synonyms	1,3-Benzenediol, 2,4,6-trinitro-, lead(2+) salt (1:1) Lead styphnate Lead trinitroresorcinate Lead tricininate Resorcinol, 2,4,6-trinitro-, lead(2+) salt (1:1) trinitroresorcin lead
CAS Number	15245-44-0
Structural Formula	

 Pb^{2+}

Molecular Formula	C6H3N3O8.Pb
Molecular Weight	450.27

Chemical Name in the Inventory and Synonyms	1,3-Benzenediol, 2,4,6-trinitro-, lead(2+) salt, monobasic 50-56-6A Lead 2,4,6-trinitroresorcinate, monobasic
CAS Number	51331-05-6
Structural Formula	No Structural Diagram Available

Molecular Formula	Unspecified
Molecular Weight	

Chemical Name in the Inventory and Synonyms	<p>Lead, (2-methyl-4,6-dinitrophenolato-O1)(nitrato-O)-mu.-oxodi-, monohydrate</p> <p>Lead 4,6-dinitro-o-cresylate hydroxide nitrate Diplumboxane, 1-(2-(2-methyl-4,6-dinitrophenoxy))-3-(nitrooxy)-, hydrate (1:1) lead dinitroorthocresolate</p>
CAS Number	79357-62-3
Structural Formula	
Molecular Formula	C7H5N3O9Pb2.H2O
Molecular Weight	707.54

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