Benzene, methyldinitro-: Human health tier II assessment

27 November 2014

CAS Number: 25321-14-6

Preface

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

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

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

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

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

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

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

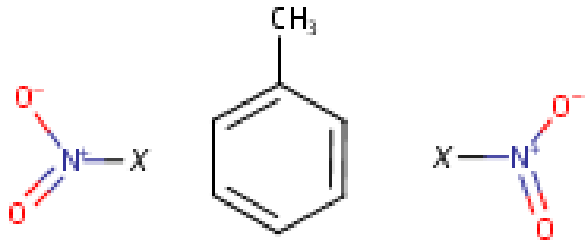
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	Dinitrotoluene DNT Tg-DNT
Structural Formula	
Molecular Formula	C7H6N2O4
Molecular Weight (g/mol)	182.1
SMILES	c1(N(=O)=O)c(N(=O)=O)c(C)ccc1

Import, Manufacture and Use

Australian

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

International

The chemical, technical grade dinitrotoluene (Tg-DNT) comprises mixed isomers of dinitrotoluene (up to six isomers are possible, although 2,4-DNT and the 2,6- isomer normally dominate). The chemical typically comprises approximately 76 % 2,4-DNT (CAS No. 121-14-2) and 20 % 2,6-DNT (CAS No. 606-20-2).

The following international uses have been identified through:

- Galleria Chemica;
- the Substances and Preparations in the Nordic countries (SPIN) database;
- the US National Library of Medicine's Hazardous Substances Data Bank (HSDB); and
- various international assessments (IARC, 1996; OECD, 2004; EURAR, 2008; ACGIH, 2011; ATSDR, 2013).

The chemical has reported commercial use including as a:

- gelatinising, plasticising and waterproofing agent in both commercial and military explosive compositions; and
- modifier for smokeless powders in the munitions industry.

The chemical has reported site-limited use including as a chemical intermediate in the production of:

- toluenediisocyanates, precursors to polyurethane polymers (major use);
- trinitrotoluene (TNT);
- dyes and dyestuffs; and
- rubber chemicals.

The chemical is registered under the Registration, Evaluation, Authorization and Restrictions of Chemicals (REACH) legislation for 'intermediate use only' (REACH).

Restrictions

Australian

No known restrictions have been identified.

International

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

- European Union (EU) Cosmetics Regulation 1223/2009 Annex II—List of substances prohibited in cosmetic products;
- New Zealand Cosmetic Products Group Standard—Schedule 4: Components cosmetic products must not contain; and

- Association of South East Asian Nations (ASEAN) Cosmetic Directive Annex II Part 1: List of substances which must not form part of the composition of cosmetic products.

A primary component of the chemical, 2,4-DNT, is listed in Annex XIV 'The Authorization List' with a sunset date of 21 August 2015 (date from which placing on the market and use of a substance is prohibited, unless an authorisation is granted) (ECHA, 2014).

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):

- Carc. Cat. 2; R45 (carcinogenicity)
- Muta. Cat. 3; R68 (mutagenicity)
- Repr. Cat. 3; R62 (reproductive toxicity)
- T; R23/24/25 (acute toxicity)
- Xn; R48/22 (repeated dose toxicity)

Exposure Standards

Australian

The chemical has an exposure standard of 1.5 mg/m^3 time weighted average (TWA). A skin notation (Sk—absorption through the skin may be a significant source of exposure) is assigned (Safe Work Australia).

Guidance on the interpretation of workplace exposure standards for airborne contaminants advises that 'exposure to carcinogens should be eliminated or minimised so far as is reasonably practicable' (Safe Work Australia, 2013).

International

The following exposure standards are identified (Galleria Chemica):

An exposure limit (occupation exposure limit—OEL, permitted exposure limit—PEL) of 0.15–1.5 mg/m^3 (TWA) and 0.3–5 mg/m^3 short-term exposure limit (STEL) in different countries such as the USA, China, Canada (Yukon), Denmark, Norway, Mexico, Sweden and South Africa.

The American Conference of Governmental Industrial Hygienists (ACGIH) recommends a threshold limit value (TLV) of 0.2 mg/m^3 (TWA) for dinitrotoluene (CAS No. 25321-14-6). This value is 'intended to minimise the potential for heart disease and possible reproductive effects. It should also provide protection against methemoglobinemia'. A skin notation is assigned (ACGIH, 2011).

Health Hazard Information

The chemical typically comprises approximately 76 % 2,4-DNT (CAS No. 121-14-2) and 20 % 2,6-DNT (CAS No. 606-20-2).

Toxicokinetics

The toxicokinetics of the chemical and its components 2,4-DNT and 2,6-DNT have been investigated in laboratory animals and in humans (EURAR, 2008; ACGIH, 2011; ATSDR, 2013, NICNASa; NICNASb).

The available animal data demonstrate that the chemical can be readily absorbed following oral exposure. Biological monitoring in workers exposed to Tg-DNT indicated that dermal exposure and inhalation (in the form of dusts and aerosols) exposure could be a significant route of entry for the chemical in humans.

Once absorbed, the chemical and its metabolites are distributed throughout the body with a similar pattern of distribution in all animal species. In radiolabelling studies for the component isomers, levels of radioactivity were typically higher in the liver and kidneys. There is insufficient evidence to determine if there is accumulation in these organs (NICNASa; NICNASb).

The majority of absorbed chemical is excreted in the urine. Metabolites detected in the urine of workers exposed to Tg-DNT are dinitrobenzoic acids (2,4- and 2,6-), 2,4-(amino,nitro)-benzoic acids, and dinitrobenzyl alcohol glucuronides (2,4- and 2,6-).

Bile is an important route of excretion. In male rats, a larger percentage of the administered dose is excreted in the bile than in females. The liver and intestinal microflora both appear to play an important role in the metabolism of the chemical. The chemical is initially oxidised in the liver by cytochrome P-450 to form dinitrobenzyl alcohols. The glucuronide conjugate of this metabolite is eliminated via urine or excreted in bile. In the bile, this metabolite is released into the gut where it is metabolised by intestinal microflora (by hydrolysis and reduction of the nitro group) to form aminonitrobenzyl alcohols. A portion of the aminonitrobenzyl alcohols are reabsorbed and transported back to the liver where they are metabolised into reactive species capable of binding to DNA (see **Genotoxicity**). Data indicate that sulfation is important in biotransforming the chemical into reactive metabolites (NICNASa; NICNASb).

Acute Toxicity

Oral

The chemical is classified as hazardous with the risk phrase ‘Toxic if swallowed’ (T; R25) in HSIS (Safe Work Australia). The available median lethal dose (LD50) values (286–660 mg/kg bw) in rats (OECD, 2004) do not support this classification. However, methaemoglobinaemia was observed following a single exposure to relatively low doses of the primary component 2,4-DNT (NICNASa). An amendment of the classification based on these non-lethal effects is recommended (refer **Recommendation** section).

Increased levels on both methaemoglobin and Heinz bodies were observed in cats following a single exposure to 2,4-DNT at 50 mg/kg bw (oral) and 20–40 mg/kg bw (intraperitoneally (i.p.)). No effects were observed following oral exposure to 10 mg/kg bw. Humans and cats are more sensitive to methaemoglobin formation than rodents (EU RAR, 2008).

Dermal

The chemical is classified as hazardous with the risk phrase ‘Toxic in contact with skin’ (T; R24) in HSIS (Safe Work Australia). No data are available for Tg-DNT.

The available LD50 value for the primary component 2,4-DNT (>2500 mg/kg bw) does not support this classification (NICNASa). An amendment to the classification is recommended (refer **Recommendation** section) based on the following:

- observed methaemoglobinaemia following a single exposure (oral and i.p.) to relatively low doses of the primary component (see **Acute toxicity: Oral**); and
- skin absorption is significant source of exposure (ACGIH, 2011).

Inhalation

The chemical is classified as hazardous with the risk phrase 'Toxic by inhalation' (T; R23) in HSIS (Safe Work Australia). No data are available for Tg-DNT or the primary component 2,4-DNT to evaluate this classification. The secondary component 2,6-DNT has high acute toxicity in a six-hour exposure study in rats with a median lethal concentration (LC50) of 0.43 mg/L (NICNASb).

An amendment to the classification (refer **Recommendation** section) is recommended, based on the following:

- observed methaemoglobinaemia following a single exposure (oral and i.p.) to relatively low doses of the primary component (see **Acute toxicity: Oral**); and
- the chemical is absorbed through all routes of exposure.

Observation in humans

Secondary effects to methaemoglobin formation such as headache, dizziness, nausea and drowsiness have been reported in workers exposed to the chemical (ACGIH, 2011). Historical cases of cyanosis and anaemia were found in munition and DNT-production factories. It is assumed that these workers were handling large amounts of the chemical without protective equipment (ATSDR, 2013).

Corrosion / Irritation

Skin Irritation

Occlusive administration of the chemical to the interior side of the ear of rabbits for 24 hours did not result in irritation (OECD, 2004). The components 2,4-DNT and 2,6-DNT are reported to be a mild irritants in rabbits. Effects were not sufficient to warrant classification (NICNASa; NICNASb).

Eye Irritation

Slight irritation was observed following application of the chemical in a non-guideline study in rabbits. Effects were reversible within seven days (OECD, 2004). The components 2,4-DNT and 2,6-DNT are not considered to be irritating to rabbit eyes (NICNASa; NICNASb).

Sensitisation

Skin Sensitisation

Based on the available data the chemical is considered to have a low potential for sensitisation. The primary component (2,4-DNT) was negative in a guinea pig maximisation test (NICNASa). Although the secondary component, 2,6-DNT, produced a mild sensitisation response in guinea pigs, the human sensitising potential of the chemical is considered to be low (NICNASb).

Observation in humans

Patch tests or photo-patch tests in healthy humans showed no allergic potential of DNT (unspecified isomer), although a single case of positive photo-patch test reaction was reported for a worker with skin problems (OECD, 2004).

Repeated Dose Toxicity

Oral

The chemical is classified as hazardous with the risk phrase ‘Danger of serious damage to health by prolonged exposure if swallowed (Xn; R48/22)' in HSIS (Safe Work Australia). The data available support this classification; in addition, given that the chemical is absorbed by all routes of exposure it is recommended that the classification should be applied to all routes (refer to **Recommendation** section).

Effects observed in available studies are consistent with the data available for several species for the component chemicals 2,4-DNT and 2,6-DNT (NICNASa; NICNASb). The target organs for toxicity appears to be the blood, liver and male reproductive system (see **Reproductive and developmental toxicity**).

In a 28-day pilot study with Tg-DNT in Fischer 344 (F344) rats, methaemoglobin levels were significantly elevated for animals treated at the highest dose (150 mg kg/bw/day). Whilst gross pathology indicated changes to the lungs, liver, kidneys and spleen, histopathological examinations were not conducted (OECD, 2004).

In a two-year study, F344 rats were exposed to Tg-DNT in doses of about 3.5, 14 and 35 mg/kg bw/day via the food. Lesions in the kidneys and liver, and testicular changes were observed. Signs of hepatotoxicity were present in the low-dose group. The lowest observed adverse effect level (LOAEL) was established as 3.5 mg/kg bw/day (OECD, 2004).

Dermal

No data are available.

Inhalation

No data are available.

Observation in humans

There is equivocal evidence of a link between exposure to the chemical for prolonged periods (>5 months) and ischaemic heart disease (ACGIH, 2011, ATSDR, 2013, NIOSH).

Genotoxicity

The chemical is classified as hazardous—Category 3 mutagenic substance—with the risk phrase ‘Possible risk of irreversible effects’ (Xn; R68) in HSIS (Safe Work Australia). The available data support this classification.

The chemical is mutagenic in several strains of *Salmonella typhimurium* (TA 98, TA 1538 and TM 677) with and without metabolic activation. The chemical was negative in a Hypoxanthine-Guanine Phosphoribosyl Transferase (HPGRT) assay with Chinese Hamster Ovary (CHO) K1 cells, a poorly documented mammalian cell gene mutation assay in mouse lymphoma cells (TK±) and an in vitro unscheduled DNA synthesis (UDS) test with primary rat hepatocytes (OECD, 2004; ATSDR, 2013). However, the component chemicals, 2,4-DNT and 2,6-DNT were positive in several in vitro assays (NICNASa; NICNASb).

The chemical induced dose-dependent DNA repair in the rat liver after 10–250 mg/kg bw oral doses. The presence of gut flora in rats, with its metabolic capacity, was a prerequisite for the induction of liver UDS by DNT in vivo (OECD, 2004).

DNA adduct formation has been observed in several organs, mainly the liver, in rats exposed to the component chemicals, 2,4-DNT and 2,6-DNT (NICNASa; NICNASb).

The chemical is not considered to cause dominant lethal mutations in rats and mice (OECD, 2004).

Significant increases in chromatid-type chromosomal aberrations were observed in workers at a DNT and TNT factory (ATSDR, 2013).

Carcinogenicity

The chemical is currently 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.

The chemical is a potent hepatocarcinogen in rats (IARC, 1996; ATSDR, 2013).

Carcinogenic effects were observed in a two-year study in F344 rats including skin/subcutaneous tissue fibromas in males, mammary gland fibroadenomas in females and hepatocarcinomas in both sexes. The hepatocarcinogenic effect increased dose dependently and was obvious from 3.5 mg/kg bw/day in males and 14 mg/kg bw/day in females.

The chemical administered for one year at 35 mg/kg bw/day to F344 rats produced liver tumours in approximately 50 % of animals.

The chemical demonstrated weak hepatocyte-initiating activity and hepatocyte-foci promoting ability (IARC, 1996; ACGIH, 2011; ATSDR, 2013).

The US National Institute for Occupational Safety and Health (NIOSH) found an increased risk of liver, bile duct, and gall bladder cancers in workers exposed to DNT, based on six reported cases. No such increase was detected in a previous study based on a smaller group of workers from the same and another munitions factory in the United States. Sufficient data are not available to draw conclusions on the carcinogenicity in humans (IARC, 1996; NIOSH, 1996; OECD, 2004).

The International Agency for Research on Cancer (IARC) overall evaluation is that the component chemicals 2,4-DNT and 2,6-DNT are ‘possibly carcinogenic to humans’ (Group 2B) based on 'sufficient evidence in experimental animals' and 'inadequate evidence in humans' for the carcinogenicity of the chemical (IARC, 1996).

Reproductive and Developmental Toxicity

The chemical is classified as hazardous—Category 3 substance toxic to reproduction—with the risk phrase ‘Possible risk of impaired fertility’ (Xn; R62) in HSIS (Safe Work Australia). The available data support this classification.

Effects in the male reproductive system, including decreased sperm production and testicular atrophy, were observed in a long-term feeding study in rats (see **Repeated dose toxicity** section). The no observed adverse effect level (NOAEL) value for testicular effects was 3.5 mg/kg bw/day. Similar effects were observed for the component chemicals 2,4-DNT and 2,6-DNT (NICNASa; NICNASb).

In a developmental toxicity study with Tg-DNT, pregnant rats were exposed orally for 14 days during gestation. Effects on foetal haematological parameters and organ weights were observed in the presence of maternal toxicity. Foetal malformations were not observed, even at dose levels that produced significant maternal toxicity. There was no effect on post natal development (OECD, 2004; ATSDR, 2013; IARC, 2013). In a three-generation study with 2,4-DNT in CD rats, reduced fertility was observed at the highest dose (34 mg/kg bw/day). A NOAEL of 3.9 mg/kg bw/day was established. No anomalies were detected in any of the offspring (NICNASa).

Reproductive effects such as an increased rate of spontaneous abortions and decreased sperm count have been observed in workers exposed to Tg-DNT. However, these studies have a number of limitations for evaluating adverse effects of the chemical in humans, such as exposure to other chemicals, small exposure populations in the studies and a lack of historical individual exposure monitoring (EURAR, 2008; ATSDR, 2013).

Other Health Effects

Neurotoxicity

Neurotoxic effects after intermediate or chronic duration exposure to the primary component chemical, 2,4-DNT, were observed in a number of species, with symptoms ranging from tremors, convulsions and ataxia to paralysis. Dogs appear to be most sensitive to the chemical with effects appearing to be cumulative. The NOAEL for male and female Beagle dogs was 0.2 mg/kg bw/day of 2,4-DNT on the basis of neurotoxicity (incoordination and paralysis) derived from a 24-month study (NICNASa).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include systemic long-term effects (carcinogenicity, mutagenicity, reproductive toxicity) and systemic acute effects (acute toxicity from oral/dermal/inhalation exposure). The chemical can also cause harmful effects following repeated exposure.

Public Risk Characterisation

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

Occupational Risk Characterisation

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

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

Limited data are available to assess the adequacy of the current exposure standard. *Guidance on the interpretation of workplace exposure standards for airborne contaminants* advises that 'exposure to carcinogens should be eliminated or minimised so far as is reasonably practicable' (Safe Work Australia, 2013). Given that lower exposure standards appear achievable (based on international exposure standards), Safe Work Australia should consider whether current controls adequately minimise the risk to workers.

The data available support amendments 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.

It is recommended that Safe Work Australia consider whether current controls adequately minimise the risk to workers. A Tier III assessment could be necessary to provide further information to determine whether the current exposure controls offer adequate protection to workers.

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]
Acute Toxicity	Toxic: danger of very serious irreversible effects through inhalation, in contact with skin and if swallowed (T; R39/23/24/25)	Causes damage to organs - Specific target organ tox, single exp Cat. 1 (H370)
Repeat Dose Toxicity	Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed (Xn; R48/20/21/22)	May cause damage to organs (kidney) 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 2 - May cause cancer (T; R45)*	May cause cancer - Cat. 1B (H350)
Reproductive and Developmental Toxicity	Repro. Cat 3 - Possible risk of impaired fertility (Xn; R62)*	Suspected of damaging fertility - Cat. 2 (H361f)

[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 oral/dermal/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) Draft Toxicological Profile for dinitrotoluenes. Accessed August 2014 at http://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=165

American Conference of Governmental Industrial Hygienists (ACGIH) 2011. Documentation of the Threshold Limit Values for Chemical Substances, ACGIH Signature Publications, 7th Edition. Dinitrotoluene

ECHA (2014). Authorisation List. European Chemicals Agency, Helsinki, Finland. Accessed August 2014 at http://echa.europa.eu/web/guest/addressing-chemicals-of-concern/authorisation/recommendation-for-inclusion-in-the-authorisation-list/authorisation-list.

European Union Risk Assessment Report (EURAR) 2008 for 2,4-Dinitrotoluene (CAS No. 121-14-2). Accessed August 2014 at http://echa.europa.eu/documents/10162/b1176fd0-799d-4c08-a908-755a1c82181f

Galleria Chemica. Accessed October 2014 at https://jr.chemwatch.net/galleria/

Hazardous Substances Data Bank (HSDB). National Library of Medicine. Accessed on October 2014 at http://toxnet.nlm.nih.gov.

International Agency for Research on Cancer (IARC) 1996. Volume 65: Printing Processes and Printing Inks, Carbon Black and Some Nitro Compounds. 2,4-Dinitrotoluene, 2,6-dinitrotoluene and 3,5-dinitrotoluene. Accessed October 2014 at http://monographs.iarc.fr/ENG/Monographs/vol65/mono65-9.pdf

National Industrial Chemicals Notification and Assessment Scheme (NICNASa). Tier II human health assessment for 1-methyl-2,4-dinitrobenzene (121-14-2). Australian Government Department of Health. Accessed October 2014 at http://www.nicnas.gov.au

National Industrial Chemicals Notification and Assessment Scheme (NICNASb). Inventory Multi-Tiered and Prioritisation (IMAP) Human Health Tier II Assessment for Cadmium sulfide. Available at http://www.nicnas.gov.au

National Institute for Occupational Safety and Health (NIOSH) 1996. Worker Health Study Summaries. Research on long-term exposure. Munitions Workers (Dinitrotoluene Exposure). Accessed October 2014 at http://www.cdc.gov/niosh/pgms/worknotify/dinitrotoluene.html#dnt

OECD (2004). SIDS Initial Assessment Report (SIAR) on dinitrotoluene (isomers mixed) (25321-14-6). Accessed October 2004 at http://webnet.oecd.org/HPV/UI/SIDS_Details.aspx?key=ff783183-3680-43e0-b0f7-994ec0770483&idx=0

REACH Dossier. Dinitrotoluene (CAS No. 25321-14-6). Accessed October 2014 at http://echa.europa.eu/web/guest/information-on-chemicals/registered-substances.

Safe Work Australia (SWA). Guidance On The Interpretation Of Workplace Exposure Standards For Airborne Contaminants. April 2013. Accessed at http://www.safeworkaustralia.gov.au/sites/swa/about/publications/pages/workplace-exposure-standards-airborne-contaminants

Safe Work Australia (SWA). Hazardous Substances Information System (HSIS). Accessed October 2014 at http://hsis.safeworkaustralia.gov.au/HazardousSubstance

Substances in Preparations in Nordic Countries (SPIN). Accessed October 2014 at http://188.183.47.4/dotnetnuke/Home/tabid/58/Default.aspx

Last update 27 November 2014

Share this page