1,2,4-Benzenetricarboxylic acid, tris(2-ethylhexyl) ester: Human health tier II assessment

01 July 2016

CAS Number: 3319-31-1

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.

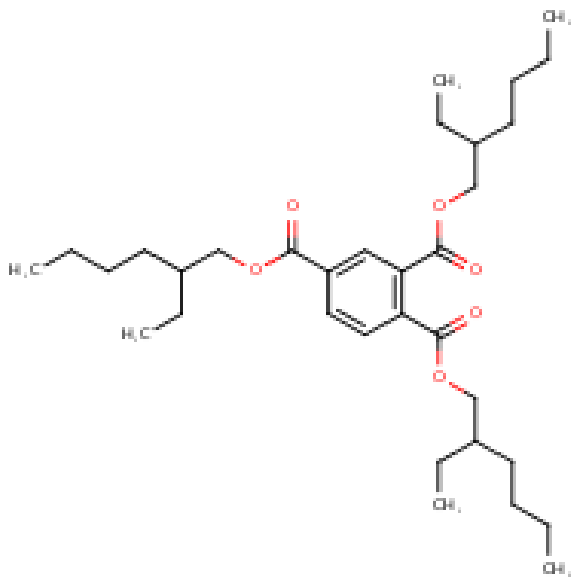
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	tris-2-ethylhexyl trimellitate triethylhexyl trimellitate trioctyltrimellitate (TOTM)
Structural Formula	
Molecular Formula	C33H54O6
Molecular Weight (g/mol)	546.78
Appearance and Odour (where available)	Yellow oily liquid

SMILES	C(=O)(c1c(C(=O)OCC(CCCC)CC)cc(C(=O)OCC(CCCC)CC)cc1)OCC(CCCC)CC

Import, Manufacture and Use

Australian

The following Australian industrial uses were reported under previous mandatory and/or voluntary calls for information.

The chemical has reported cosmetic use as a softener.

The chemical has reported commercial use as an additive in construction materials.

International

The following international uses have been identified through:

- the European Union (EU) Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) dossiers (REACH);
- the Organisation for Economic Co-operation and Development Screening information data set International Assessment Report (OECD, 2002);
- Galleria Chemica;
- the Substances and Preparations in Nordic countries (SPIN) database;
- the European Commission Cosmetic Ingredients and Substances (CosIng) database;
- the United States (US) Personal Care Products Council International Nomenclature of Cosmetic Ingredients (INCI) Dictionary;
- the US National Library of Medicine's Hazardous Substances Data Bank (HSDB);
- Cosmetic Ingredient Review (CIR, 2015);
- US Consumer Product Safety Commission (CPSC, 2010).

The chemical has reported cosmetic uses as perfuming agent and skin conditioning agent or emollient.

The chemical has reported domestic uses, including in:

- machine wash liquids/detergents;
- automotive care products; and
- fragrances and air fresheners.

The chemical has reported commercial use in:

- lubricants, greases and hydraulic fluids;
- manufacture of polymers;
- manufacture of polyvinyl chloride (PVC) for use in electrical cable and wires;

- construction and building materials (e.g. flooring, metal, wooden and plastic materials);
- paints and coatings; and
- adhesives.

Non-industrial uses of the chemical in articles listed below have also been identified internationally:

- furniture;
- toys;
- curtains;
- foot-wear;
- leather products;
- paper and cardboard products; and
- haemodialysis tubing.

The chemical was listed and recommended for evaluation under the EU Community Rolling Action Plan (CoRAP) 2015-2017 (EUCoRAP, 2015)

Restrictions

Australian

No known restrictions have been identified.

International

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

- Taiwan Standard List of Application and Maximum Levels of Preservatives in Cosmetics.

Existing Work Health and Safety Controls

Hazard Classification

The chemical is not listed on the Hazardous Substances Information System (HSIS) (Safe Work Australia).

Exposure Standards

Australian

No specific exposure standards are available.

International

No specific exposure standards are available.

Health Hazard Information

The chemical is a primary plasticiser used in polyvinyl chloride (PVC) products. The physico-chemical properties of the chemical are very similar to those of diethylhexyl phthalate (DEHP); hence, the chemical can be a potential substitute for DEHP as a primary plasticiser in plastic products. In Australia, the use of DEHP in cosmetics and in certain plastic products (for example, plastic toys, childcare articles, and eating vessels and utensils) is restricted due to its reproductive and developmental toxicity, with a potential endocrine disruption mechanism, and carcinogenicity. The chemical DEHP may also cause adverse systemic effects following repeated exposure (NICNAS, 2010; NICNAS IMAP; Australian Competition and Consumer Commission (ACCC), 2011).

The chemical is included in the list of chemicals for evaluation on the EU Community Rolling Action Plan (CoRAP) 2015-2017 (EU CoRAP, 2015). It is anticipated that the current assessment may be revisited if the outcomes of the EU evaluation are significantly different from this assessment.

Toxicokinetics

In a study conducted in four male Sprague Dawley (SD) rats, the chemical was administered by oral gavage at a single dose of 100 mg/kg bw to determine the rates of absorption, metabolism and excretion of the chemicals. The chemical was partially hydrolysed in the gastrointestinal tract to 2-ethylhexanol and the corresponding di-ester (diethylhexyl trimellitate). Following further hydrolysis, 2-ethylhexanol and a single isomer of mono-(2-ethylhexyl) trimellitate are produced. Following absorption, 2-ethylhexanol and the monoester were extensively metabolised into metabolites eliminated in the urine and as expired CO_2. Overall, 94.4 % of the dose was recovered. Excretion occurred mainly in the faeces (75 % of the administered dose) with 16.3 % found in the urine and 1.9 % in expired air. The metabolites in the urine were identified as mono-(2-ethyhexyl) trimellitate, 2-ethylhexanol, 2-ethylhexanoic acid and 2-heptanone. The elimination of the chemical in the urine and in CO_2 was biphasic. The elimination half lives for expired CO_2 were 3.1 to 4.3 hours and 31 to 42 hours for urine (REACH; OECD, 2002; CPSC, 2010; CIR, 2015).

The distribution and elimination of the chemical was also determined in SD rats (five animals) dosed intravenously (iv) with 10.5 mg/kg bw radiolabelled chemical. The study reported a rapid initial distribution (distribution half life of 46.2 min) and slow clearance (plasma clearance rate of 40.5 mL/kg/hour) of the chemical from the body. The amounts of radioactivity recovered in the urine and faeces after 14 days were 3.3 % and 16.9 %, respectively. The renal clearance rate was 13 mL/kg.hour. In the same study, 28 rats (four/group) were dosed iv with 15.6 mg/kg bw radiolabelled chemical and sacrificed at 1, 6, 24, 48, 72, 168 and 338 hours after dosing. At necropsy, the majority of the chemical was distributed in the liver, lungs and spleen. The peak radioactivity in the liver was 71.6 % of the dose at 24 hours and 18.6 % of the dose in the lungs at one hour, followed by a rapid decline of the radioactivity (CIR, 2015).

The dermal absorption of the chemical was investigated using Franz cells (full thickness skin samples excised from female nude mice and specific pathogen-free pigs). The receptor medium contained 40 % ethanol and the donor medium had 5.4 mM chemical in 40 % ethanol and pH 7.4 buffer. The chemical was not found in the receptor medium after 12 hours, indicating that there was no dermal absorption of the chemical (CIR, 2015).

Acute Toxicity

Oral

The chemical has low acute toxicity based on a result from an animal test conducted in accordance with OECD Test Guideline (TG) 401. Following oral exposure, the median lethal dose (LD50) in Crj:CD (SD) rats (five animals/sex) was >2000/mg/kg bw.

No toxicity effects were seen at 2000 mg/kg bw/day during an observation period of 14 days (REACH; OECD, 2002; CPSC, 2010; CIR, 2015).

Dermal

The chemical has low acute toxicity based on results from animal tests following occlusive dermal exposure. The LD50 in New Zealand White rabbit (three animals/sex) was >2 mL/kg bw (approximately 2000 mg/kg bw; chemical density of 0.999 g/mL) (REACH; OECD, 2002; CIR, 2015). The LD50 in a guinea pig (one animal; unspecified sex) was >20 mL/kg bw (approximately 20 000 mg/kg bw; chemical density 0.999 g/mL) (CPSC, 2010; CIR, 2015).

Inhalation

The chemical had low acute toxicity in an animal test following inhalation exposure. In a study conducted in accordance with OECD TG 403, SD rats (five animals/sex) were exposed to the chemical (aerosol) at a single inhalation dose of 2600 mg/m^3 for 4 hours. No mortality occurred. At necropsy, reddened patches on the lung were observed. The median lethal concentration (LC50) in rats was >2600 mg/m^3 (approximately >2.6 mg/L) (REACH; OECD, 2002; CPSC, 2010; CIR, 2015).

Corrosion / Irritation

Skin Irritation

The chemical is reported to slightly irritate the skin in animal studies. The effects were not sufficient to warrant hazard classification.

In a skin irritation test, the chemical was applied onto the shaved back (abraded and intact skin) of New Zealand White rabbits (six animals/unspecified sex) at concentrations of 0 and 100 % for 24 hours under semi-occlusive patch and observed up to 72 hours. The chemical produced slight to well defined erythema when applied to intact skin of rabbits at 100 % concentration; however, recovery occurred within 72 hours. There was no difference in the irritation effects observed between the animals with abraded and intact skin (REACH; OECD, 2002; CIR, 2015).

The chemical (neat) applied to skin under an occlusive patch produced reversible irritation effects in guinea pigs (one animal/unspecified sex; single 24–hour exposure) and Californian rabbits (two animals/sex; single 4–hour exposure) (CIR, 2015).

Eye Irritation

The chemical is reported to be a slight eye irritant in animal studies. The effects were not sufficient to warrant hazard classification.

In an eye irritation study (similar to a guideline study), 0.1mL of the chemical (100%) was instilled into the conjunctival sac of one eye of each six New Zealand White rabbits and remained unwashed for 7 days. The left eye remained untreated and served as a control. Minimal conjunctival irritation, which was fully reversible within 7 days after exposure, was reported (REACH; OECD, 2002).

In a non-guideline eye irritation study, the chemical was applied into the eyes of rabbits (unspecified number and strain) with seven day observation. Slight eye irritation effects, which fully reversed in 24 hours after exposure, were reported (REACH; OECD, 2002).

Sensitisation

Skin Sensitisation

The chemical was not found to induce dermal sensitisation when tested in a Buehler test in guinea pigs and in a human repeated insult patch test.

In a Buehler test similar to OECD TG 406, the chemical (neat) was not a primary irritant or skin sensitiser in albino guinea pig (10 animals/group). No skin reactions were seen during the induction phase or subsequent challenge phase (REACH; OECD, 2002).

In a non-guideline Buehler test, the chemical (unspecified purity) was not a sensitiser in guinea pigs (unspecified number of animals and strain) (REACH; OECD, 2002).

Observation in humans

The chemical was tested for dermal irritation and sensitisation in 201 men and women volunteers. The chemical at 1 % (v/v in acetome) was applied to the skin under semi-occlusive path for 3 consecutive weeks followed by 2 weeks rest period. The chemical was found to be non–irritating and non–sensitising in human repeated insult patch test (CPSC, 2010; CIR, 2015).

Repeated Dose Toxicity

Oral

Considering the lowest observed-effect levels (LOELs) available from a 90-day rat study (225 mg/kg bw/day), and based on the minor liver effects reported in various repeated dose toxicity studies, repeated oral exposure to the chemical is not considered to cause serious damage to health.

In a sub-chronic oral toxicity study conducted in accordance with OECD TG 408, SD rats (10 animals/sex/dose) were administered the chemical daily in the diet at doses of 0, 50, 225 and 1000 mg/kg bw/day for 90 days. No treatment–related mortalities were observed at any dose level. At high dose, statistically significant increases in relative liver weights in both sexes and absolute liver weights in females were reported. Treatment–related changes in the liver seen in the high dose animals included: diffused hepatocytic hypertrophy (consisting of increased cytoplasmic eosinophilia), and increased incidence of extramedullary haematopoiesis. In high dose males, decreases in absolute and relative spleen weights were also observed. In females, a slight increase in the incidence of extramedullary haematopoiesis in the spleen was reported. The no observed adverse effect level (NOAEL) was determined to be 225 mg/kg bw/day (REACH; CIR, 2015).

In a sub-acute oral toxicity study similar to OECD TG 407, Crj:CD (SD) rats (five animals/sex/dose) were administered the chemical daily by oral gavage at 0, 100, 300 and 1000 mg/kg bw/day, and to an additional 1000 mg/kg bw/day recovery group for 28 days. No signs of clinical, haematological, biochemical and histopathological toxicity were reported. The NOAEL was determined to be 1000 mg/kg bw/day (REACH; CPSC, 2010; CIR, 2015).

In another sub-acute oral toxicity study, Fischer 344 (F344) rats (five animals/sex/group) were administered the chemical continuously in the diet at 0, 0.2, 0.67 and 2 % (calculated as 0, 184, 650 and 1826 mg/kg bw/day) for 28 days. The chemical, DEHP at 0.067 % was used as a reference compound. No mortalities were observed at any dose level. At 650 mg/kg bw/day and above, statistically significant increases in absolute and relative liver weights were observed in both sexes. Statistically significant reductions in haemoglobin levels and increased leucocyte counts were seen. Statistically significantly increased levels of albumin and slightly lower alkaline phosphatase levels were also reported. In the highest dose group, a slight increase in the number of peroxisomes were reported. There were also statistically significant increases in palmitoyl-CoA activity in both sexes at the highest dose, and in males at 0.2 and 0.67 %. Catalase activity was statistically significantly increased in males, and a decrease in cytoplasmic basophilia in females was reported at the highest dose. The chemical, DEHP at 0.67 % caused a moderate increase in the number of peroxisomes. In all of the other enzyme measurements, the severity of the effects in rats given DEHP at 0.67 % were greater than those given the chemical at a higher dose of 2.0 %. The results demonstrate that the chemical produces similar morphological and biochemical changes to the rat liver as DEHP, although the chemical was much less potent in its action, causing less peroxisome proliferation and enzyme induction than DEHP. The NOAEL was determined to be 184 mg/kg bw/day (REACH; OECD, 2002; CIR, 2015).

Dermal

No data are available for this chemical.

Inhalation

No data are available for this chemical.

Genotoxicity

Based on the weight of evidence from well–conducted in vitro (in accordance with OECD TG for genotoxicity and mutagenicity) and in vivo genotoxicity studies (non-guideline study), the chemical is not considered to be genotoxic.

The chemical gave negative results when tested in following in vitro and in vivo gene mutation and clastogenicity tests (REACH; OECD, 2002; CPSC, 2010; CIR, 2015):

In vitro studies

- Bacterial reverse mutation assay –*Salmonella typhimurium* TA 1535, 1537, 100 and 98, *Escherichia coli* WP2 uvrA
- Mammalian cell gene mutation assay –Mouse lymphoma L5178Y cells
- Mammalian cell gene mutation assay–Chinese hamster ovary (CHO) cells
- Chromosomal aberration assay–Chinese hamster lung fibroblast (V79)
- Chromosomal aberration assay–Human lymphocyte
- DNA damage and repair, unscheduled DNA synthesis in mammalian cells in vitro–Rat hepatocytes

In vivo studies

- Rodent dominant lethal assay–dose concentration of 1400 mg/kg bw chemical (unspecified vehicle) in male Swiss Mice

Urine from rats fed with a diet containing 2000 mg/kg bw of the chemical for 15 days did not induce mutagenic activity with or without metabolic activation in various strains of *S. typhimurium* (CPSC, 2010; CIR, 2015).

Carcinogenicity

There are no carcinogenicity studies available for the chemical. The chemical is not expected to have carcinogenic potential in humans.

The ability of the chemical to induce peroxisome proliferation was investigated because the chemical has been considered as an alternative to DEHP (CPSC, 2010; CIR, 2015). Peroxisome proliferation causes an increase in liver weights and is also believed to induce hepatocarcinogenicity in rodents. The ability of the chemical to induce peroxisome proliferation was investigated in rats fed daily with a diet containing up to 2 % of the chemical. The chemical induced a slight increase in the number of peroxisomes at 2 % (Refer to **Repeat dose toxicity-Oral section**). However, the increase was less than that reported for DEHP at lower concentration of 0.67 %. Furthermore, liver and kidney effects, when related to peroxisome proliferation or a rodent-specific mode of action, are unlikely to be relevant to humans (CPSC, 2010; NICNAS, 2010; NICNAS IMAP; CIR, 2015).

Reproductive and Developmental Toxicity

Based on the limited information available, exposure to the chemical caused a slight reduction in the number of spermatocytes & spermatids in male rats. The available data do not provide adequate evidence of reproductive and developmental toxicity for the chemicals in humans.

In a reproductive/developmental toxicity screening test conducted in accordance with OECD TG 421, Crj:CD:SD rats (12/sex/dose) were dosed with the chemical at 0, 100, 300 and 1000 mg/kg/bw day by oral gavage. Males were dosed from 14 days prior to mating (46 days including mating) and females were dosed from 14 days prior to mating through lactation day (LD) 3. There were no mortalities; clinical signs of toxicity; effects on body weight, food consumption and organ weights; gross pathology; effects on male and female fertility; or foetal developmental toxicity following treatment with the chemical at all doses. No histological changes in the ovaries of treated females were reported. Histopathological examination of the testes revealed a slight reduction in the number of spermatocytes and spermatids in the testes of males dosed with 300 or 1000 mg/kg bw/day, in the absence of effects on the testes weights and gross pathology. A NOAEL of 100 mg/kg bw/day was determined for male fertility and 1000 mg/kg bw/day for female fertility. The NOAEL of 1000 mg/kg bw/day was determined for the offspring (REACH; OECD, 2002; CPSC, 2010; CIR, 2015).

In another study, pregnant SD rats (20 animals) were dosed at 0, 100, 500 or 1050 mg/kg bw/day of the chemical by gavage on gestation days (GD) 6–9, and recovery animals (15 animals) were dosed on GD 6 through LD 20. No treatment related signs of maternal toxicity were reported. There were no effects reported on foetal body weights, litter viability, sexual maturation or development of reproductive tracts (testicular and ovarian effects) in both sexes. An increase in the number of foetuses with displaced testes in the high dose group was reported; however, the values were within the historical control ranges. An increase in the number of male offspring with retained areolar regions at mid-dose but not in the high dose was reported. The NOAEL for maternal and developmental toxicity was 1050 mg/kg/bw/day (CPSC, 2010; CIR, 2015).

In a study conducted in pregnant SD rats (three to four), no effects on testicular testosterone production, foetal viability or maternal body weights were observed following exposure to the chemical by oral gavage at concentrations up to 1000 mg/kg bw/day (CIR, 2015).

Other Health Effects

Endocrine Disruption

The available data do not provide adequate evidence of perturbation to the endocrine system following exposure to the chemical.

The chemical in dimethyl sulfoxide (DMSO, 10^{-3}–10^{-4} mol/L) had no affinity for oestrogen receptor alpha (ERα) when screened in an in vitro competitive assay measuring its binding capacity for the human ERα (CIR, 2015). However, in another study, the chemical exhibited oestrogenic activity in an in vitro test using human osteoblastic (US-O2) reporter gene cell lines for ERα and ERβ (CIR, 2015).

Risk Characterisation

Critical Health Effects

The chemical is not considered to have high toxicity. There may be possible systemic long-term effects (testicular effects and increased liver weights) when exposed repeatedly to high concentrations of the chemical.

Public Risk Characterisation

Considering the range of domestic, cosmetic and personal care products that may contain the chemical, the main route of public exposure is expected to be through the skin. However, based on the chemical's relative high molecular weight and bulky structure, the chemical is expected to have low leachability; hence, low migration rate. The available studies also indicate that the chemical is not significantly absorbed through the skin (CPSC, 2010; CIR, 2015).

The toxicity profile of the chemical indicates that it does not exhibit the reproductive toxicity associated with DEHP.

Overall, the risk to the public posed by cosmetic/domestic products containing the chemical is not considered to be unreasonable and further risk management is not considered necessary for public safety.

Occupational Risk Characterisation

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

Whilst the chemical is not recommended for classification as a hazardous chemical, the chemical could pose an unreasonable risk to workers unless adequate control measures to minimise repeated exposure to high concentrations are implemented.

NICNAS Recommendation

The risk to workers and public from this chemical is not considered to be unreasonable. The chemical is not recommended for classification and labelling under the current approved criteria and adopted GHS. This report does not consider classification of physical hazards and environmental hazards. No recommendations or further assessment is required.

Regulatory Control

Advice for industry

Control measures

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

- minimising manual processes and work tasks through automating processes;
- work procedures that minimise splashes and spills;
- regularly cleaning equipment and work areas; and
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the chemical.

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

Personal protective equipment should not solely be relied upon to control risk and should only be used when all other reasonably practicable control measures do not eliminate or sufficiently minimise risk. Guidance in selecting personal protective equipment can be obtained from Australian, Australian/New Zealand or other approved standards.

Obligations under workplace health and safety legislation

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

- ensuring that hazardous chemicals are correctly classified and labelled;

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

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

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

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

References

Bailey, JE (ed.) 2011, Compilation of Ingredients Used in Cosmetics in the United States (CIUCUS), 1st Edition, Personal Care Products Council, Washington, D.C.

European Chemicals Agency (ECHA) Risk Assessment Committee (RAC) 2008. Opinion on proposing harmonised classification and labelling at EU level of p-tert-butylphenol (CAS No. 98-54-4). Accessed March 2016 at http://echa.europa.eu/documents/10162/a69caf1d-c84c-45e5-b30d-4d18b949eef8

European Union Risk Assessment Report (EU RAR) 2008. p-tertbutylphenol (CAS No. 98-54-4). Accessed March 2016 at http://echa.europa.eu/documents/10162/605c05d5-0ef9-46cf-b5a2-bb8a51ac26e5

Galleria Chemica. Accessed March 2016 at http://jr.chemwatch.net/galleria/

International Fragrance Association (IFRA) Standards: p-Tertbutylphenol (CAS NO. 98-54-4). Accessed March 2016 at http://www.ifraorg.org/en-us/standards

Organisation for Economic Development and Cooperation (OECD) 2000. OECD Screening Information Data Set (SIDS), SIDS Initial Assessment Report (SIAR) on p-tert-butylphenol—(98-54-4). Accessed March 2016 at http://www.inchem.org/documents/sids/sids/98544.pdf.

Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) Dossier. 4-tertbutylphenol (CAS No. 98-54-4). Accessed February 2016 at http://echa.europa.eu/registration-dossier/-/registered-dossier/15260/1

Substances in Preparations in Nordic Countries (SPIN) Database. Accessed March 2016 at http://195.215.202.233/DotNetNuke/default.aspx

US National Library of Medicines, Household Products Database, Health& Safety Information on Household Products. Accessed March 2016 at http://householdproducts.nlm.nih.gov/

Last update 01 July 2016

Share this page