Selected organic hydroperoxides: Human health tier II assessment

01 July 2016

Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
Hydroperoxide, 1,1-dimethylethyl	75-91-2
Hydroperoxide, 1-methyl-1-phenylethyl	80-15-9
2-Butanone, peroxide	1338-23-4

Preface

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

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

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

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

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

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

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

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

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

Disclaimer

ACRONYMS & ABBREVIATIONS

Grouping Rationale

The chemicals in this group all have the general molecular formula ROOH. Two of the chemicals tert-butyl hydroperoxide (TBHP, CAS No. 75-91-2) and cumene hydroperoxide (CHP, CAS No. 80-15-9) are tertiary hydroperoxides that share the general molecular formula, R-C(CH_3)$_2$-O-OH (OECD 2008). Methyl ethyl ketone peroxide (MEKP, CAS No. 1338-23-4) is a reaction mixture consisting of dimers (50%), trimers (25%), and monomeric peroxy compounds (OECD, 2007; HSDB). The major component has the structure, HOOC(R)$_2$-O-OC(R_2)-OOH (see **Structural formula**).

Whilst the systemic metabolites of the chemicals in this group differ, their toxicity is driven by the reactivity of the hydroperoxide moiety, particularly the generation of reactive free radicals. This is supported by the available toxicological data.

The chemicals in this group have similar chemical properties. Organic peroxides are oxidising, flammable and could undergo heat-induced decomposition. The predominant uses of the chemicals are similar.

Import, Manufacture and Use

Australian

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

The chemicals are used in the manufacture of other chemicals. Specifically, the main reported use for MEKP is in the manufacture of polymers and in polyester resins. It is also used as a spray putty hardener and catalyst for fibreglass and polyester/vinyl surface coatings.

The total volume introduced into Australia, reported under previous mandatory and/or voluntary calls for information, was ≤100 tonnes for TBHP and ≤1000 tonnes for MEKP.

Australian safety data sheets (SDS) for MEKP and CHP indicate their use in industrial hardeners and in adhesives.

International

The following international uses have been identified through:

- the European Union (EU) Registration, Evaluation, Authorization and Restriction of Chemicals (REACH) dossiers (REACHa; REACHb; REACHc);
- the Organisation for Economic Co-operation and Development Screening Information Data Set (OECD SIDS);
- 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) and various international assessments (NTP, 1993; OECD, 2007; EU RAR 2008; OECD, 2008).

The chemicals in this group are predominantly used as intermediates in polymer initiators and raw materials in manufacturing other chemicals. These chemicals also have commercial use in adhesives, plastic hardeners and in auto body repair kits.

The chemicals MEKP and CHP have identified consumer uses in adhesives (REACHb; REACHc). The chemical MEKP has reported domestic use in two consumer two-part auto body repair kits, although these are reported to be discontinued. CHP has reported domestic use in home maintenance products (such as adhesives) up to a concentration of 3 % (HPD).

The chemicals are expected to be completely consumed during reactions.

In addition, MEKP has reported cosmetic use as a cosmetic biocide; an oxidising agent; and a preservative. MEKP is included in the US Personal Care Products Council's INCI dictionary with the identified functions of anti-acne agents; antifungal agents; antimicrobial agents; cosmetic biocides; oxidizing agents; and preservatives. However, there is currently no documented use of the chemical in the US (PCPC, 2011).

Restrictions

Australian

The chemical MEKP is listed in the *Poisons Standard—the Standard for the Uniform Scheduling of Medicines and Poisons* (SUSMP) in Schedule 5 (SUSMP, 2015).

Schedule 5 chemicals are described as 'Substances with a 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.' Schedule 5 chemicals are labelled with 'Caution' (SUSMP, 2015).

International

No specific restrictions have been identified.

Existing Worker Health and Safety Controls

Hazard Classification

The chemical CHP (CAS No. 80-15-9) is classified as hazardous, with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia):

- T; R23 (acute toxicity);
- Xn; R21/22 (acute toxicity);
- Xn; R48/20/22 (repeated dose toxicity); and
- C; R34 (corrosivity).

The chemical MEKP (CAS No. 1338-23-4) is listed on the HSIS, but no specific risk phrases are listed (Safe Work Australia). A chemical described as methyl ethyl ketone peroxide trimer (no CAS No.) is listed with the following risk phrases for human health:

- Xn; R65 (aspiration toxicity)
- Xi; R38 (irritation)
- R43 (sensitisation)

TBHP (CAS No. 75-91-2) is not listed on the HSIS (Safe Work Australia).

Under the Approved Criteria for Classifying Hazardous Substances, organic hydroperoxides are to be classified with the risk phrase C; R34 except where evidence to the contrary is available (Safe Work Australia, 2004). Classifications for physical hazards also apply (Safe Work Australia, 2004).

Exposure Standards

Australian

The chemical MEKP has an exposure limit of 1.5 mg/m^3 (0.2 ppm) peak limit (HSIS).

International

The following exposure standards are identified (Galleria Chemica).

The chemical TBHP has an exposure limit of 5 mg/m^3 (TWA) in Latvia.

The chemical CHP has an exposure limit of 1 mg/m^3 (TWA) in Latvia and Russia.

The chemical MEKP has an exposure limit of 1.5 mg/m^3 or 0.2 ppm (TWA) in different countries such as Argentina, Canada, Chile, China, Croatia, Estonia, France, Finland, Indonesia, Ireland, Malaysia, Mexico, New Zealand, Singapore, South Africa, South Korea, Sweden, Switzerland, Taiwan, United Arab Emirates, United Kingdom, and Spain. In other countries, the following are the occupational exposure limit (OEL) values of the chemical:

- 1.4 mg/m^3 or 0.2 ppm in Peru;

- 0.7 ppm in Greece;
- 1 mg/m^3 in Norway, Denmark and Iceland; and
- 0.2 ppm in Belgium, Colombia, Italy, Nicaragua, Portugal and Uruguay.

The American Conference of Governmental Industrial Hygienists (ACGIH) ceiling limit for the chemical is 0.2 ppm threshold level value. 'This value is intended to minimize the potential for skin and eye irritation and possible adverse effect in the liver and kidney that were reported from animal studies with MEKP' (ACGIH, 2011).

Health Hazard Information

The chemical MEKP is only available in the presence of diluents which are used in the manufacturing process to reduce the potential explosion hazard. Most studies used a commercial product made of about 40% MEKP and 60% dimethyl phthalate (DMP) (NTP, 1993).

Toxicokinetics

Based on the available data, the chemicals in this group are expected to be readily absorbed following oral and inhalation exposure, and to a lesser extent following dermal absorption (EU RAR, 2008; REACHc).

The major metabolic pathway for organic hydroperoxides is generally a two-electron reduction to the corresponding alcohol by glutathione (GSH) peroxidases. Insufficient reductive capacity for peroxides or exhaustion of the GSH supply can result in one-electron oxidation generating the peroxyl radical or one-electron reduction generating the alkoxy radical (OECD, 2007; EURAR 2008; OECD 2008; Wiley VCH). The formation of free radicals prior to depletion of GSH cannot be ruled out (EU RAR, 2008).

Specific toxicokinetic data are only available for TBHP. In rat studies, the chemical was rapidly absorbed, metabolised and widely distributed following oral and subcutaneous exposure. The half-life of TBHP was 12 hours in the first phase (2-36 hours) and 50 hours in the second phase (36-96 hours) (EURAR, 2008; REACHa). The metabolite, 2-methylpropan-2-ol (tert-butanol; CAS No. 75-65-0), is oxidised to 2-methyl-1,2-propanediol and 2-hydroxyisobutyric acid. TBHP and its metabolites are excreted in the urine, faeces or expired air (EURAR, 2008). A number of in vitro studies using human endothelial cells, intact skin samples of the mouse, rat liver microsomes and isolated rat liver nuclei demonstrated that TBHP could also generate free radical compounds through a one-electron oxidation and reduction when reducing agents are depleted. In a 14-day gavage study in rats, an increase in free radicals was observed in the liver, kidney and blood (EU RAR, 2008).

The chemical CHP is expected to be initally metabolised to cumyl alcohol (CAS No. 536-60-7) (OECD, 2008).

The suggested metabolites for MEKP are acetic acid (CAS No. 64-19-7), ethyl acetate (CAS No. 141-78-6), methyl ethyl ketone (butanone, CAS 78-93-3) and hydrogen peroxide (CAS No. 7722-84-1) (OECD, 2007; REACHc).

The chemicals are reported to have lipid peroxidising ability, with MEKP considered the most potent (NTP, 1993; OECD, 2007; EU RAR, 2008). Pre-treatment with antioxidants such as vitamin E has been shown to reduce the extent of lipid peroxidation (NTP, 1993).

Acute Toxicity

Oral

The chemical CHP is classified as hazardous with the risk phrase ‘Harmful if swallowed’ (Xn; R22) in HSIS (Safe Work Australia). The available data (median lethal dose—LD50—382 mg/kg bw) support this classification (OECD, 2008). Reported signs of toxicity include extensive urinary blood content in rats exposed to 400 mg/kg bw/day.

The chemicals TBHP and MEKP have moderate acute toxicity based on animal tests following oral exposure. The LD50 of TBHP in rats is 560 mg/kg (70 % TBHP) (EURAR, 2008). The LD50 for MEKP in rats is 681 mg/kg bw (42% MEKP in DMP to 1017 mg/kg bw (40% MEKP in corn oil) (NTP, 1993; REACH). In the TBHP-exposed Sprague Dawley (SD) rats, depression, pallor, loss of righting reflex, lacrimation, hypothermia, haematuria, and gastric erosions were observed (REACHa). Classification is considered warranted (refer **Recommendation** section).

The reported human toxicity for MEKP (see **Observation in humans section**) also supports the recommendation for classification (NTP, 1993; OECD, 2007; EURAR, 2008).

Dermal

The chemical CHP is classified as hazardous with the risk phrase ‘Harmful in contact with skin’ (Xn; R21) in the HSIS) (Safe Work Australia). The available data from a non-guideline study in rats, which reported LD50 values between 500 and 1500 mg/kg bw, support this classification (REACHb). Reported signs of toxicity include haematuria.

In an acute dermal toxicity study, TBPH has a moderate acute toxicity in rabbits. In this study, New Zealand White (NZW) rabbits were dermally exposed to doses of 250-2000 mg/kg TBHP for 24 hours under an occlusive condition. The reported LD50 was 628 mg/kg (EURAR, 2008; REACHa). Cyanosis, ataxia, lethargy,

breathing difficulty, red nasal discharge, prostration, involuntary eye movement, convulsions and vocalisation were observed in the chemically treated rats before their death. Histopathologically, the treated animals displayed dark coloured lungs, liver, spleen and urinary bladder and severely irritated necrotic skin even in lower doses (EURAR, 2008).

In a non-guideline study, MEKP (35—39 % in DMP) was applied to rabbits at doses of 1000, 2000, 4000 and 8000 mg/kg bw (n=2 per dose). The LD50 was reported as 4000 mg/kg based on the death of one animal. It is not clear whether the skin had been abraded. Whilst the data are inconclusive for classification, given effects observed for TBHP and CHP, classification (acute dermal toxicity) for all the chemicals in this group is considered warranted (see **Recommendation** section).

Inhalation

The chemical CHP is classified as hazardous with the risk phrase 'Toxic by inhalation' (T; R23) in the HSIS (Safe Work Australia). The available data from a non-guideline study in rats (median lethal concentration—LC50—1.37 mg/L/4h) support this classification (REACHb). Reported signs of toxicity include irregular breathing. This classification is also supported for TBHP, which has a reported LC50 of 1.85 mg/L/4h (100 % TBHP). Changes in the lungs such as hyperaemia, with haemorrhages on the lung surface, were reported (EU RAR, 2008; OECD, 2008).

The chemical MEKP has lower acute inhalation toxicity compared with other members of this group; however, classification is still warranted. In an inhalation study conducted similarly to OECD Test Guideline (TG) 403, rats were exposed to MEKP in DMP in a sealed chamber for four hours. The calculated LC50 was 17 mg/L (combined sexes). Focal haemorrhages and congestion in the lungs were noted (REACHc). The 4-hour inhalation observable LC50 for non-blended MEKP products is reported to range from 15.4 mg/L (2162.2 ppm; male rats) to 53.6 mg/L (female rats; 7525.4 ppm). Clinical signs included bradypnoea, dyspnoea, hypoactivity, flaccidity, ataxia, sedation, hypothermia and prostration (OECD, 2007).

Observation in humans

A number of case reports indicated that accidental and intentional ingestion of MEKP by humans caused widespread damage in a number of tissues (Mittleman et al., 1986; Karhunen et al., 1990; Bates et al., 2001; van Enckevort et al., 2008. These include the following:

- liver necrosis with severe ulceration with subsequent scarring and clinical changes including temporary cardiac arrest, abdominal burns, and breakdown of muscle tissues (rhabdomyolysis);
- respiratory failure from ingesting solution of MEKP in DMP;
- digestive tract stenosis, gastric burns, severe metabolic acidosis, oesophageal and gastric necrosis;
- haemolysis; and
- respiratory distress.

In some cases, ingestion of MEKP resulted in death (Karhunen et al., 1990; Subbalaxmi et al., 2010).

Corrosion / Irritation

Corrosivity

The chemical CHP is classified as hazardous with the risk phrase 'Causes burns' (C; R34) in HSIS (Safe Work Australia). Under the Approved Criteria for Classifying Hazardous Substances, organic hydroperoxides are to be classified with the risk phrase C; R34 except where evidence to the contrary is available (Safe Work Australia, 2004). The data available support classification for all chemicals in this group.

The chemicals TBHP, MEKP and CHP have been reported to cause skin necrosis in animal studies. In these studies, the skin (shaved, intact or abraded) of NZW rabbits were exposed to either 0.5 mL of 70 % of TBHP or 0.5 mL of 33 % MEKP in DMP for 24 hours under occlusive conditions. The results showed that all of the TBHP-treated rabbits were depressed and cyanotic within an hour of exposure and half of them died within 24 hours of exposure. Moderate to severe oedema (score: 3.5 for both intact and abraded) and mild to moderate erythema (score: 0.33 for intact and 0.84 for abraded) were observed at the time of death or within 24 hours of exposure for the surviving animals. These effects were still present at 72 hours and by 14 days, the skin of surviving rabbits became necrotic (EU RAR, 2008). Within four hours of MEKP exposure, the animals exhibited well-defined erythema, slight to moderate ischaemia, haemorrhages and severe oedema (REACHc). Scores for erythema and oedema at 24 hours were 4 and 2.67 respectively. Similarly to TBHP, these effects were not reversible after 72 hours. After eight weeks, scar tissue and slightly disturbed hair growth were observed at the application sites (REACHc).

The chemical CHP was also tested in a non-guideline study in rabbits. Necrosis was observed following application of undiluted substance. Moderate erythema was identified following application of a 10 % of CHP solution (REACHb). The maximum non-irritating concentration was reported to be 35 % for TBHP (EU RAR, 2008) and 1.5 % for MEKP (NTP, 1993).

Respiratory Irritation

There is evidence of respiratory irritation in acute and repeated dose inhalation toxicity studies with TBHP, MEKP and CHP (see **Acute toxicity:inhalation** and **Repeated dose toxicity:inhalation** sections). In a sensory irritation test, Swiss Webster mice were exposed for 30 minutes to concentrations between 13 and 82

ppm (48–303 mg/m^3) TBHP. The breathing frequency was reduced in the majority of animals. Similar treatment related effects could not be established in a comparable study in rats; however, in a five-day study in SD rats (refer **Repeated dose toxicity** section), signs of respiratory irritation including dry rales were reported (EU RAR, 2008).

Eye Irritation

Corrosive chemicals are also considered to cause irreversible effects on the eyes. In test guideline-compliant eye irritation studies in rabbits, exposing one eye of each animal to 0.1 mL of 70 % TBHP or MEKP in DMP produced severe eye irritation. All treated animals displayed irreversible iritis; keratitis; corneal opacities; corneal ulcers; and keratoconus (EU RAR, 2008; REACHa; REACHc). The chemical CHP also produced corneal injury, iritis and necroid eyelids in a non-guideline study in rabbits. No corneal damage was produced following exposure to 1 % solution, whilst 5 % resulted in moderate corneal injury (REACHb).

Sensitisation

Skin Sensitisation

The chemical TBHP was found to induce skin sensitisation in guinea pig maximisation test (GPMT), mouse lymph node assay (LLNA) and in a mouse ear swelling test (MEST) (EU RAR, 2008; NTP, 2009; REACHa).

In the GPMT, Hartley guinea pigs were exposed to TBHP at induction concentrations of 0.7 % and 2.1 % and challenge concentration of 21 %. Six out of ten treated animals were positive for delayed contact sensitisation (EU RAR, 2008; REACHa).

The chemical was also positive for skin sensitisation in female Balb/c mice in LLNA and MEST (NTP, 2009). In the LLNA test, animals were exposed to 0.5, 1%, 2.5%, 5%, 10 % and 25% TBHP suspended in acetone. The results demonstrated an increase in the draining lymph node cell proliferation from the 1.0 % TBHP concentration. However, when compared with the controls, the increase in lymph node cell proliferation only reached significance at concentrations of 2.5 and 25 %. ‘None of the doses administered resulted in a Stimulation Index (SI) of three or greater’(NTP, 2009).

The animals in the MEST study were exposed to 1%, 2.5%, 5%, 10 % and 25 % TBHP during the sensitisation phase, and 25 % during the challenge phase. The results indicated a dose-related increase (but not significant) in ear swelling at 24 hours after the challenge exposure. However, after 48 hours post challenge exposure, this increase became statistically significant in animals exposed to concentrations of 2.5 % and above (NTP, 2009). Classification of this chemical is considered warranted (see **Recommendation** section).

The chemical MEKP is not considered to be a skin sensitiser (OECD, 2007). In a GPMT, very slight or well-defined erythema was noted all animals following challenge with a 5 % MEKP concentration. A second challenge application was performed to determine whether the observed reactions were irritant effects and/or delayed contact hypersensitivity. Very slight erythema was observed on the left flank (test substance at 1% (w/w)) and the right flank (vehicle) of 2/20 and 1/20 animals, respectively. The slight, non-persistent and low incidence of cutaneous reactions noted after the application of the second challenge were attributed to the irritant properties of MEKP (OECD, 2007; REACHc).

No data are available for CHP.

Repeated Dose Toxicity

Oral

The chemical CHP is classified as hazardous with the risk phrase ‘Harmful: danger of serious damage by prolonged exposure if swallowed’ (Xn; R48/22) in HSIS (Safe Work Australia). Limited data are available to evaluate this classification. Death of 4/5 rats following exposure to 19 mg/kg bw three times weekly for seven weeks was reported (REACHb).

Based on the available data, MEKP and TBHP are not considered to cause systemic toxicity following repeated oral exposure. Effects observed are considered to be related to the corrosive nature of the chemicals.

In a combined repeat-dose and reproduction/developmental toxicity study (OECD TG 422), Wistar rats (n=12/sex) were fed daily diet containing 3, 10 or 30 mg/kg bw/day (70 % TBHP) for 45 days. Apart from mild renal alterations (resembling male rat specific ∝2u-globulin nephropathy), no significant treatment-related effects were observed. The NOAEL for systemic toxicity in this study was 21 mg/kg bw/day (100 % TBHP) (EU RAR, 2008; REACHa). Local effects on stomach and/or forestomach were observed at higher doses in non-guideline studies in rats and mice (EU RAR, 2008).

Although liver and kidney damage were reported for rats exposed to 97 mg/kg bw of MEKP three times weekly for seven weeks (non-guideline study) (ACGIH, 2011), these effects were not observed in more recent studies. Results from the combined reproductive/developmental toxicity screening study (OECD TG 421) with MEKP (32% with a mixture of diluents) in rats showed reductions in body weight and food consumption. Macroscopic and microscopic changes in the stomach were also reported.

Clinical changes such as gasping, laboured breathing and/or rales were also observed. The NOAEL was 50 mg/kg bw/day (OECD, 2007). The chemical, MEKP did not cause significant changes in F344/DuCrl and SPF rats exposed to doses 20, 65, 200 mg/kg bw for 28 days (REACHc).

Dermal

In repeated dose dermal toxicity studies with MEKP and TBHP, skin necrosis, inflammation, and epidermal hyperplasia were observed (NTP, 1993; OECD, 2007; EU RAR, 2008; OECD, 2008). Significant treatment related systemic effects were not reported.

Inhalation

Limited data are available for the chemicals in this group. The chemical CHP is classified as hazardous with the risk phrase 'Harmful: danger of serious damage by prolonged exposure through inhalation' (Xn; 48/R20) in HSIS (Safe Work Australia). Limited data are available to evaluate this classification. In a three-month non-guideline study, rats were exposed daily for six hours/day, five days/week to 1, 6, 31 and 124 mg/m^3 of CHP delivered as aerosol. The no observed adverse effect concentration (NOAEC) was established as 31 mg/m^3 (approximately 5 ppm) based on tissue irritation at the site of contact, including ulceration and inflammation of the cornea and eyes, nasal turbinates and the stomach lining (REACHb).

The inhalation toxicity of TBHP following repeated exposure was investigated in Wistar rats (OECD TG 412-compliant study). In this study, the animals were exposed head only to 7.4, 22.2 or 66.7 mg/m^3 of TBHP for 6 hours a day, 5 days a week for 28 days. Hyperplasia or metaplasia of the nasal cavity transitional epithelium was observed in rats exposed to the highest dose (66.7 mg/m^3). No other treatment-related effects were reported. The no observed adverse effect concentration (NOAEC) reported in this study was 22.2 mg/m^3 (approximately 6 ppm) (REACHa).

Genotoxicity

Based on the weight of evidence, from the available in vitro and in vivo genotoxicity studies, the chemicals are considered to be genotoxic. The observed genotoxic effects are likely due to free radical formation and the ensuing DNA interaction. Positive results were reported from several in vitro tests for gene mutation and clastogenicity. A number of in vivo tests produced positive results, including in germ cells providing evidence of mutagenicity at the site of contact. Classification is considered warranted (see **Recommendation** section).

TBHP

In vitro, TBHP is mutagenic in vitro in the following tests (EURAR, 2008; NTP, 2009; REACHa):

- bacterial gene mutation in *Salmonella typhimurium* strains TA98, TA100, and TA1537 with or without metabolic activation; TA102 and TA2638A without metabolic activation (test guideline compliant);
- bacterial gene mutation in a number of *Escherichia coli* strains such as IC3789, IC3821 and IC188 (no metabolic activation) and IC203 (with and without metabolic activation);
- SOS-chromotest (to detect bacterial DNA damage) in E.coli strains PQ37 without metabolic activation;
- recombination in *Saccharomyces cerevisiae* D4;
- reverse mutation in *Neurospora crassa*;
- forward gene mutation in mouse lymphoma L5178Y cells with or without metabolic activation;
- chromosomal aberration (CA) in Chinese hamster ovary (CHO) cells with or without metabolic activation (test guideline compliant);
- chromosomal damage in Chinese hamster V79 lung cells without metabolic activation;
- DNA base damage at low concentration in SP2/0 derived murine hybridoma cells without metabolic activation; and
- DNA fragmentation and strand breaks in rat hepatocytes.

In vivo, results from the CFT-Swiss mice study demonstrated DNA damage (strand breaks) in testes and epididymal sperm following intraperitoneal (i.p.) administration of TBHP (Kumar et al., 2002). In this study, mice were treated with 15, 30, and 60 µmol/100g bw/day of TBHP. The observed DNA damage was dose-related in the testes but only the highest concentration caused damage in epididymal sperm (Kumar et al., 2002). In Wistar rats, changes in sperm morphology and strand breaks in testes and epididymal sperm were also observed following an i.p. injection of 150 – 660 µmol/kg bw/day of TBHP for either five or 14 days (EURAR, 2008).

In another study, TBHP gave positive result in a dominant lethal assay in CFT-Swiss mice following i.p. administration of the chemical for five days (EURAR, 2008). This study was conducted following test guidelines similar to OECD.

The chemical was also positive for sex chromosome loss and nondisjunction in *Drosophila melanogaster*. These positive results are considered indicative of a local mutagenic effect. Whilst a comet assay with five-day inhalation exposure produced a negative result in lung tissues, this is not considered sufficient to rule out local mutagenicity. The nasal and tracheal epithelium which would receive higher exposures compared with the lung tissue could not be examined (ECHA, 2014).

The chemical TBHP induced DNA adducts in the liver and stomach following oral exposure to a dose exceeding the LD50. A limited comet assay in rat liver after subcutaneous exposure was negative. Increased chromosome aberrations in the bone marrow were observed in male Mongrel albino rats exposed to 107 mg/m^3 TBHP for 2.5 and 4 months. However, there were a number of deviations from OECD test guidelines and study documentation was limited (EU RAR, 2008). In another guideline compliant bone marrow micronucleus test, the chemical tested negative in Swiss mice and in rats exposed to TBHP via a single intravenous (i.v.) injection and inhalation exposure (for five days), respectively (EU RAR, 2008). It is likely that the negative result was due to the rapid conversion of TBHP to tert-butanol. Overall, it is considered that the chemical is unlikely to cause inheritable genetic damage through oral, inhalation and dermal exposure (EU RAR, 2008).

CHP

Limited data are available. In vitro, CHP was mutagenic in several bacterial *S. typhimurium* strains (TA97, TA98, TA100, TA102 and TA1535) and *E. coli* strains, with or without metabolic activation. The chemical also induced DNA strand breaks in human leukaemia cells. In vitro, chromosome aberration studies were not identified (OECD, 2007; REACHb). Both positive and negative results have been reported in dominant lethal assays. Adult male Swiss mice were administered 0.15 mmol /kg bw of CHP via i.p. for five consecutive days and were mated with virgin females for a period of five weeks. A marginal increase in dead implants was observed only during the first week (OECD, 2008). Prior to mating, single doses of CHP (34 and 90 mg/kg; 0.22 mmol/kg and 0.59 mmol/kg, respectively) were administered via i.p. to 5 or 7 male mice, respectively. No effects on the number of total implants, as comprised by live implants, early and late foetal deaths were observed. However, because matings were only conducted for one week post-treatment, this study was adequate for measuring effects on mature sperm, but was inadequate for measuring induction of dominant lethal mutations over the entire course of spermatogenesis (REACHb).

Results from an in vivo study in adult CFT-Swiss mice indicated that multiple doses of CHP induced dose-dependent DNA damage (strand breaks) in testes and epididymal sperms. In this study, mice were treated with 7.5, 15, and 60 µmol/100g bw/day CHP via daily i.p. administration for five days (Kumar et al., 2002).

MEKP

The chemical MEKP (suspended in dimethyl sulfoxide) did not produce mutations in *S. typhimurium* strains TA 98, TA 100 and TA 1537 with or without metabolic activation (NTP, 1993). A weak mutagenic effect was observed in one study with strain TA 1535, but this was not replicated in other studies (OECD, 2007). The chemical gave positive mutagenic responses in mouse lymphoma L5178Y cells assay (no metabolic activation), and induced sister chromatid exchange (SCE) and chromosomal aberration in CHO with and without metabolic activation (NTP, 1993; OECD, 2007).

In vivo, MEKP induced DNA interstrand crosslinks and DNA–protein crosslinks in the brain of rats following i.p. injection (NTP, 1993). In a 13-week dermal toxicity study, the chemical did not cause an increase in the frequency of micronucleated erythrocytes in the peripheral blood sample from the exposed male and female mice (NTP, 1993).

Carcinogenicity

Carcinogenicity studies have been conducted to investigate the potential for the chemicals TBHP and MEKP to initiate and/or promote tumours (NTP, 1993; OECD, 2007; EURAR, 2008; NTP; REACHa; REACHc). Whilst the chemicals on their own did not initiate tumours in rats and mice, evidence for their tumour promoting activities have been reported as outlined in the following sections. Carcinogenicity at sites of first contact from oral and inhalation exposure cannot be ruled out (EU RAR, 2008).

TBHP

In a skin painting study, skin tumours were identified in mice (species not reported) exposed to 16.6 % of TBHP (in benzene) for 45 weeks. These mice were pre-exposed to a tumour initiating agent, 4-nitroquinoline 1-oxide (NTP). However, the study lacked an appropriate control group. In another long term study (up to 51 weeks), topically-applied TBHP was highly effective in promoting malignant conversion of UV-induced papilloma in Sencar and SKH-1 mice (NTP). A TBHP metabolite, tert-butanol, has been shown to cause tumours in the renal tubules of male rats following chronic exposure in drinking water (NTP). It was suggested that these tumours were initiated by the irreversible binding of the chemical to alpha-2u-globulin (Borghoff et al., 2001; NICNAS). In adddition, exposure to TBHP is not considered likely to result in tert-butanol at levels that can induce tumours (EU RAR, 2008).

MEKP

No two-year carcinogenicity studies of MEKP were identified in the literature to examine its direct carcinogenic potential. However, evidence for the tumour promoting activity of MEKP was observed in a long-term dermal study (46 weeks) in male and female hairless albino rats (n=24). In this study, the animals were irradiated with ultraviolet (UV) light (280-320 nm wavelengths) for 18 weeks for tumour initiation. The animals were then exposed to MEKP (topical application in diethyl maleate solvent) three weeks after the cessation of irradiation twice a day for 25 weeks. Compared with experimental controls, exposure to the MEKP resulted in a statistically significant number of tumours in mice with UV pre-exposure (Logani et al., 1984). The chemical was reported to possess in vivo lipid peroxidising activity which was suggested to promote the UV-initiated skin tumours.

The underlying mechanism for the tumour-promoting activity by organic peroxides has been suggested to be associated with the metabolic activation of these compounds to free radicals (possibly with reactive oxygen species) which leads to enhanced lipid peroxidation and ultimately DNA damage. This has been demonstrated in intact skin tissue through a pathway associated with one-electron reduction. The ability of the organic peroxides to penetrate into viable skin determines the free radical generation (NTP).

Reproductive and Developmental Toxicity

Several studies (test guideline compliant) have been conducted to investigate the reproductive and developmental toxicity of chemicals in this group (OECD, 2007; EURAR, 2008; OECD, 2008; REACHa; REACHb; REACHc). Overall, none of the chemicals in this group are considered toxic to reproduction or development. The chemical TBHP did not cause reproductive and developmental toxicity following oral exposure in Wistar rats at doses 3-30 mg/kg bw/day. Additionally, oral exposure to MEKP did not induce signicant reproductive and developmental changes in Crj: CD(SD) rats gavaged with 25, 50, 75 mg/kg/day of the chemical.

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include the following:

- acute toxicity from oral, dermal and inhalation exposure;
- local effects (corrosivity and respiratory irration); and
- systemic long-term effects (mutagenicity, also carcinogenicity at the site of contact following repeated exposure cannot be ruled out).

The potential for skin sensitisation cannot be ruled out for TBHP and CHP.

Public Risk Characterisation

TBHP

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

MEKP

Based on the available use data, widespread use in cosmetic and domestic products is not expected. The chemical is currently listed on Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons* (SUSMP). A number of warning statements, first aid instructions and safety directions relating to eye and skin contact apply. The current controls are considered adequate to minimise the risk to public health posed by domestic and cosmetic products containing the chemical, therefore, the chemical is not considered to pose an unreasonable risk to public health.

CHP

The general public could be exposed through the skin or inhalation when using domestic products containing the chemical. However, based on limited US information derived from the National Library of Medicine (NLM) Household Products Database, the concentration in these products is not considered to be sufficiently high to cause corrosive effects. In addition, the risk of mutagenicity/carcinogenicity at site of first contact is considered to be low as these products are not likely to be used on an ongoing basis by members of the public. Therefore, the risk to public health is not considered to be unreasonable and further risk management is not considered necessary for public safety.

Occupational Risk Characterisation

During product formulation, oral, dermal, ocular and inhalation 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 chemicals at lower concentrations could also occur while using formulated products containing the chemicals. The level and route of exposure will vary depending on the method of application and work practices employed.

Given the critical systemic long-term, systemic acute and the local health effects, the chemicals could pose an unreasonable risk to workers unless adequate control measures to minimise dermal, ocular and inhalation exposure are implemented. This assumes that oral exposure is prevented by personal hygienic measures. In addition, organic peroxides are strong oxidising agents and highly unstable which could cause serious explosion and fire hazards. Hence, chemicals 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 the appropriate controls.

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

Currently, an exposure limit in Australia only applies to MEKP. Given the potential for both ocular and respiratory irritation and that the chemicals TBHP and CHP could be mutagenic at the site of first contact, Safe Work Australia should consider whether current controls adequately minimise the risk to workers.

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 for TBHP and CHP 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 chemicals are recommended for classification and labelling under the current approved criteria and adopted GHS as below. This assessment does not consider classification of physical and environmental hazards.

Certain classification only apply to one or two members of the group as follows:

- acute toxicity (T; R23) applies to CHP and TBHP
- acute toxicity (Xn; R20) applies to MEKP;
- repeat dose toxicity (T; R48/20/22) applies to CHP (existing classification); and
- skin sensitisation (Xi; R43) applies to TBHP.

Classification for respiratory irritation is covered by the corrosivity classification under the Approved Criteria for Classifying Hazardous Substances, however a separate GHS classification is required.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Harmful if swallowed (Xn; R22) Harmful in contact with skin (Xn; R21) Toxic by inhalation (T; R23) Harmful by inhalation (Xn; R20)	Harmful if swallowed - Cat. 4 (H302) Harmful in contact with skin - Cat. 4 (H312) Fatal if inhaled - Cat. 2 (H330) Harmful if inhaled - Cat. 4 (H332)
Irritation / Corrosivity	Causes burns (C; R34)	May cause respiratory irritation - Specific target organ tox, single exp Cat. 3 (H335) Causes severe skin burns and eye damage - Cat. 1B (H314)
Sensitisation	May cause sensitisation by skin contact (Xi; R43)	May cause an allergic skin reaction - Cat. 1 (H317)
Repeat Dose Toxicity	Harmful: danger of serious damage to health by prolonged exposure through inhalation and if swallowed (Xn; R48/20/22)*	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)

[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 chemicals should be used according to the instructions on the label.

Advice for industry

Control measures

Control measures to minimise the risk from oral, dermal, ocular and inhalation 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 chemicals are used. Examples of control measures which could minimise the risk include, but are not limited to:

- using closed systems or isolating operations;

- using local exhaust ventilation to prevent the chemicals from entering the breathing zone of any worker;
- health monitoring for any worker who is at risk of exposure to the chemicals, if valid techniques are available to monitor the effect on the worker's health;
- 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 chemicals.

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 chemicals 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 these chemicals has not been undertaken as part of this assessment.

References

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

Bates N, Driver CP, Bianchi A 2001. Methyl ethyl ketone peroxide ingestion: toxicity and outcome in a 6-year-old child. Pediatrics 108(2) pp. 473-476.

Borghoff S, Prescott J, Janszen D, Wong B, Everitt J 2001. a2u-Globulin nephropathy, renal cell proliferation, and dosimetry of inhaled tert-butyl alcohol in male and female F-344 rats. Toxicological Sciences 61(1) pp. 176-186.

Chang RH, Yeh PY, Shu CM, Duh YS 2001. Thermal hazard analysis of methyl ethyl ketone peroxide. Proceedings of the 17th Annual Conference of Asia Pacific Occupational Safety and Health Organization, Taipei, Taiwan, ROC, September 24–29. Accessed May 2015 at **http://users.cis.fiu.edu/~chens/PDF/thermal.pdf**

ChemIDPlus Advanced. Accessed May 2015 at **http://chem.sis.nlm.nih.gov/chemidplus/**

CosIng. Cosmetic Ingredients and Substances. Accessed May 2015 at **http://ec.europa.eu/consumers/cosmetics/cosing/**

ECHA (2014) Committee for Risk Assessment (RAC) Opinion proposing harmonised classification and labelling at EU level of tert-butyl hydroperoxide. Accessed June 2015 at **http://echa.europa.eu/opinions-of-the-committee-for-risk-assessment-on-proposals**-for-harmonised-classification-and-labelling/-/substance-rev/5502/term?_viewsubstances_WAR_echarevsubstanceportlet_SEARCH_CRITERIA_NAME=tert-butyl+hydroperoxide&_viewsubstances_WAR_echarevsubstanceportlet_SEARCH_CRITERIA_EC_NUMBER=200-915-7

European Union Risk Assessment Report (EU RAR) 2008. Tertiary butyl hydroperoxide (CAS No. 75-91-2). Accessed May 2015 at **https://echa.europa.eu/documents/10162/13630/trd_rar_hh_netherlands_tbhp_en.pdf**

Galleria Chemica. Accessed May 2015 at **http://jr.chemwatch.net/galeria/**

Hazardous Substances Data Bank (HSDB). National Library of Medicine. Accessed May 2015 at **http://toxnet.nlm.nih.gov**

International Agency for Research on Cancer (IARC) (1985). Evaluation of the carcinogenic risk of chemicals to humans. IARC Monograph Volume 36. Allyl compounds, aldehydes, epoxides and peroxides. Accessed May 2015 at **http://monographs.iarc.fr/ENG/Monographs/vol1-42/mono36.pdf**

Karhunen PJ, Ojanpera I, Lalu K, Vuori E 1990. Peripheral zonal hepatic necrosis caused by accidental ingestion of methyl ethyl ketone peroxide. Human& Experimental Toxicology 9 pp. 197-200.

Kumar R, Doreswamy K, Shrilatha B, Muralidhara 2002. Oxidative stress associated DNA damage in testis of mice: induction of abnormal sperms and effects on fertility. Mutation Research 513(1-2) pp.103-111.

Logani MK, Sambuco CP, Forbes PD, Davies RE 1984. Skin-tumour promoting activity of methyl ethyl ketone peroxide-a potent lipid-peroxidizing agent. Food and Chemical Toxicology 22(11) pp. 879-882.

Mitsui M, Furukawa F, Sato M, Enami T, Imazawa T, Nishikawa A, Takahashi M 1993. Carcinogenicity study of 1,1-bis(tert-butylperoxy)-3,3,5-trimethylcyclohexane in B6C3F1 mice. Food and Chemical Toxicology 31(12) pp. 929-933.

Mittleman RE, Romig LA, Gressmann E 1986. Suicide by ingestion of methyl ethyl ketone peroxide. Journal of Forensic Sciences 31(1) pp. 312-320.

National Industrial Chemicals Notification and Assessment Scheme (NICNAS). Tier II human health assessment for 2-propanol, 2-methyl- (CAS No. 75-65-0). Australian Government Department of Health. Available at **http://www.nicnas.gov.au**

National Toxicology Program (NTP). Summary of data for chemical selection. tert-Butyl hydroperoxide (CAS No. 75-91-2). Accessed May 2015 at **http://ntp.niehs.nih.gov/ntp/htdocs/chem_background/exsumpdf/butylhydroperoxide_508.pdf**

NTP 1993, NTP technical report on toxicity studies of methyl ethyl ketone peroxide (CAS No. 1338-23-4) in dimethyl phthalate (CAS No. 131-11-3). Accessed May 2015 at **https://ntp.niehs.nih.gov/ntp/htdocs/st_rpts/tox018.pdf**

NTP, 2009. Report on the assessment of contact hypersensitivity to tert-Butyl hydroperoxide in female BALB/c mice (CASRN: 75-91-2). Accessed May 2015 at **http://ntp.niehs.nih.gov/testing/types/imm/abstract/imm20605/index.html**

OECD (2007). SIDS Initial Assessment Profile (SIAP) on 2-butanone, peroxide (MEKP). Accessed at June 2015 at **http://webnet.oecd.org/HPV/UI/handler.axd?id=90126ccf-c516-44be-81af-750fd593aa85**

OECD (2008). SIDS Initial Assessment Profile (SIAP) on hydroperoxide, 1-methyl-1-phenylethyl (CHP) and hydroperoxide, 1,1-dimethylpropyl (TAHP). Accessed at June 2015 at **http://webnet.oecd.org/HPV/UI/handler.axd?id=9d4c8a7a-3ef6-467e-a05d-523c348371c3**

Personal Care Products Council (INCI Dictionary). Accessed May 2015 at **http://www.ctfa-gov.org/jsp/gov/GovHomePage.jsp**

REACH Dossier (REACHa). Tert-butyl hydroperoxide (CAS No. 75-91-2). Accessed May 2015 at **http://echa.europa.eu/web/guest/information-on-chemicals/registered-substances**

REACH Dossier (REACHb). alpha,alpha-dimethylbenzyl hydroperoxide(CAS No. 80-15-9). Accessed June 2015 at **http://echa.europa.eu/web/guest/information-on-chemicals/registered-substances**

REACH Dossier (REACHc). 2-Butanone, peroxide (CAS No. 1338-23-4). Accessed May 2015 at **http://echa.europa.eu/web/guest/information-on-chemicals/registered-substances**

Safe Work Australia (SWA). Approved Criteria for Classifying Hazardous Substances, 3rd Edition [NOHSC: 1008 (2004)]. Accesssd Mar 2015 at **http://www.safeworkaustralia.gov.au/sites/SWA/about/Publications/Documents/258/ApprovedCriteria_Classifying_Hazardous_Substances_NOHSC1008-2004_PDF.pdf**

Safe Work Australia (SWA). Hazardous Substances Information System (HSIS). Accessed May 2015 at **http://hsis.safeworkaustralia.gov.au/HazardousSubstance**.

Safe Work Australia 2012. Managing risks of hazardous chemicals in the workplace. Code of practice. Accessed May 2015 at **http://www.safework.sa.gov.au/uploaded_files/CoPManagingRisksHazardousChemicals.pdf**

Subbalaxmi MV, Abkari S, Srinivasan VR, Krishnaprasad A 2010. Methyl ethyl ketone peroxide ingestion: a rare cause of corrosive chemical poisoning. The National Medical Journal of India 23(3) pp. 150-511.

Substances in Preparations in Nordic Countries (SPIN). Accessed April 2015 at **http://188.183.47.4/dotnetnuke/Home/tabid/58/Default.aspx**

The Poisons Standard (the Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP)) 2015.Accessed May 2015 at **http://www.comlaw.gov.au/Details/F2015L00128**

US Household Products Database. US Department of Health and Human Services. Accessed May 2015 at **http://householdproducts.nlm.nih.gov/advancedsearch.htm**

van Enckevort CC, Touw DJ, Vleming LJ 2008. N-acetylcysteine and hemodialysis treatment of a severe case of methyl ethyl ketone peroxide intoxication. Clinical toxicology 46(1) pp. 74-78.

Wiley VCH. Organic peroxides: dibenzoyl peroxide vol. 3, completed 1990. Accessed May 2015 at **http://onlinelibrary.wiley.com/doi/10.1002/3527600418.mb0opexgrpe0003/pdf**

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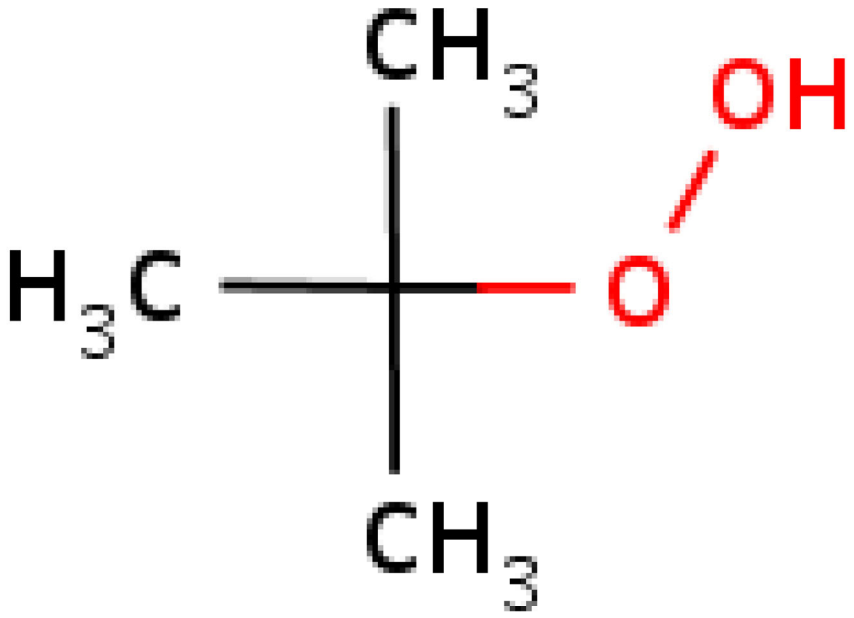
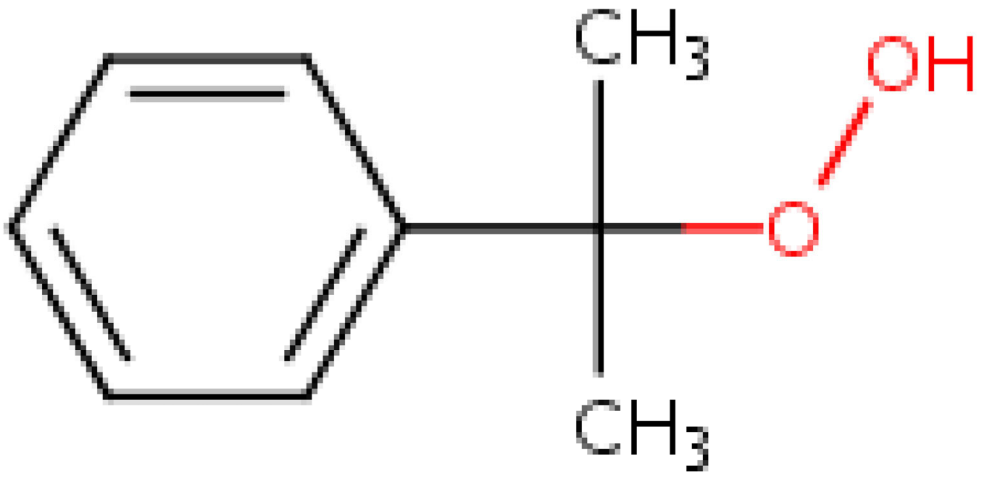
Chemical Identities

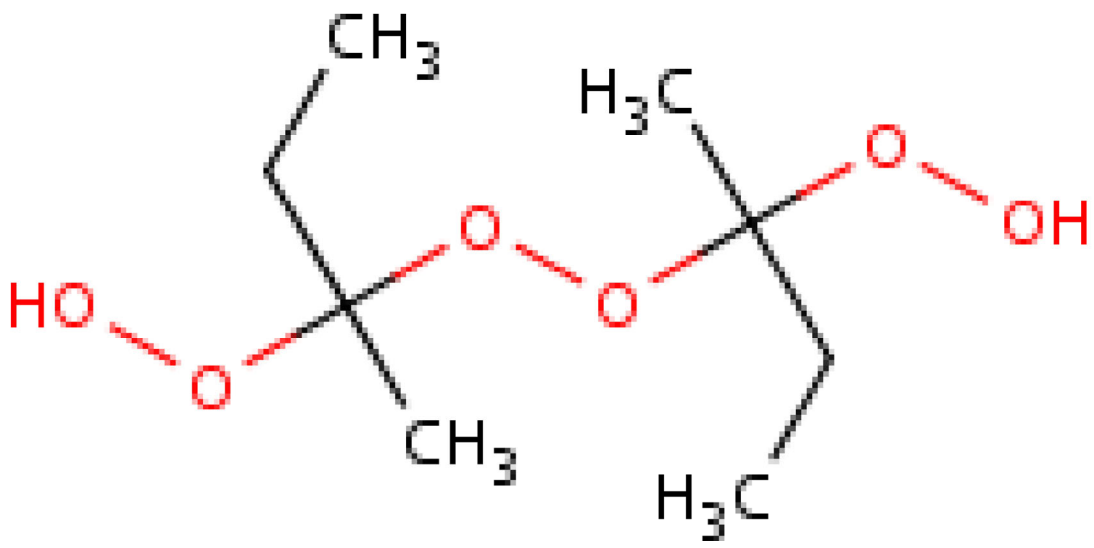
Chemical Name in the Inventory and Synonyms	**Hydroperoxide, 1,1-dimethylethyl** tert-butyl hydroperoxide

	1,1-dimethylethyl hydroperoxide 2-hydroperoxy-2-methylpropane Perbutyl H TBHP
CAS Number	75-91-2
Structural Formula	
Molecular Formula	C4H10O2
Molecular Weight	90.12

Chemical Name in the Inventory and Synonyms	**Hydroperoxide, 1-methyl-1-phenylethyl** cumene hydroperoxide hydroperoxide, .alpha.,.alpha.-dimethylbenzyl CHP
CAS Number	80-15-9
Structural Formula	

	
Molecular Formula	C9H12O2
Molecular Weight	152.19

Chemical Name in the Inventory and Synonyms	**2-Butanone, peroxide** methyl ethyl ketone peroxides MEK peroxide Butanox LPT Ketonox Esperfoam FR
CAS Number	1338-23-4
Structural Formula	



Molecular Formula	Unspecified
Molecular Weight	210.22

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