Methylphenols (Cresols): Human health tier II assessment

11 April 2014

Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
Phenol, 2-methyl-	95-48-7
Phenol, 4-methyl-	106-44-5
Phenol, 3-methyl-	108-39-4
Phenol, methyl-	1319-77-3

Preface

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

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

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

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

Stage Two of IMAP began in July 2016. We are continuing to assess chemicals on the Inventory, including chemicals identified as a concern for which action has been taken overseas and chemicals that can be rapidly identified and assessed by using

Stage One information. We are also continuing to publish information for chemicals on the Inventory that pose a low risk to human health or the environment or both. This work provides efficiencies and enables us to identify higher risk chemicals requiring assessment.

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

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

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

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

Disclaimer

ACRONYMS & ABBREVIATIONS

Grouping Rationale

Three members of the group: ortho-cresol (o-cresol—CAS No: 95-48-7), para-cresol (p-cresol—CAS No:106-44-5), and meta-cresol (m-cresol—CAS No: 108-39-4) are chemical isomers, differing only in the position of the methyl group relative to the hydroxyl group on the phenol ring. The fourth member, cresol (CAS No: 1319-77-3) is a mixture of these three isomers.

Technical grade cresol (commercial cresol) available in the USA contains about 20 % o-cresol (CAS No: 95-48-7), 30 % p-cresol (CAS No:106-44-5), and 40 % m-cresol (CAS No: 108-39-4). Phenol and xylenols are also present in small amounts as contaminants. Therefore, assessment of cresol is expected to take account of the properties of each of the three individual isomers.

The members of this group have similar molecular formule, molecular weight, and physicochemical properties. While the substitution patterns on benzene rings can lead to major differences in toxicologial properties, the chemicals in this group show a similar hazard profile and have a similar existing hazard classification. Members of this group have also been grouped together for regulatory controls (exposure standard, Poisons Standard).

The members of the group also have similar reported uses.

Import, Manufacture and Use

Australian

The total volume introduced into Australia, reported under previous mandatory and/or voluntary calls for information, was between 100 and 1000 tonnes. No specific use information was available.

The following non-industrial uses have been identified in Australia:

- ingredients in therapeutic goods including antiseptic creams and disinfectant lotions listed on the Australian Register of Therapeutic Goods (ARTG).

International

The following international uses have been identified through European Union Registration, Evaluation and Authorisation of Chemicals (EU REACH) dossiers; the Organisation for Economic Cooperation and Development Screening information data set International Assessment Report (OECD SIAR); Galleria Chemica; Substances and Preparations in the Nordic countries (SPIN) database; the European Commission Cosmetic Ingredients and Substances (CosIng) database; United States (US) Personal Care Product Council International Nomenclature of Cosmetic Ingredients (INCI) Dictionary; the US Agency for Toxic Substances and Disease Registry (ATSDR); and the US National Library of Medicine's Hazardous Substances Data Bank (HSDB).

All chemicals in this group have reported cosmetic use including:

- as a preservative, antimicrobial, and disinfectant agent; and
- in fragrance compounds.

Cresols were not reported to be in use as cosmetic ingredients in the USA, as per US Food and Drug Administration and Cosmetic, Toiletry and Fragrance Association (CTFA, 2001; FDA, 2001; both cited by CIR, 2006). There is also currently no documented use of these chemicals in cosmetic products in the United States (Personal Care Products Council, 2011).

All chemicals in this group have reported domestic use including:

- in paints, lacquers and varnishes; and
- as adhesive (binding) agents and fillers.

The US Household Products Database does not give evidence for use of these chemicals in consumer products, indicating that the chemicals are not likely to be widely available for domestic uses.

All chemicals in this group have reported commercial use including:

- as solvents;
- as disinfectants and fumigants;
- as a preservative or stabiliser in cleaning/washing agents, surface treatment products, paints, and adhesives (binding agents);
- as components in photographic developers;
- in ore flotation and fibre treatment agents;
- in tanning and metal degreasing agents;
- in agents to remove carbon deposits from combustion engines;
- in textile scouring agents;
- in metal cleaning agents and paintbrush cleaners;
- as laboratory chemicals;
- as cutting oils and as additives to lubricating oil;
- as wood preservatives; and

- in non-agricultural pesticides and preservatives.

All chemicals in this group have reported site-limited use including as organic intermediates (main use) in manufacturing:

- phenolic and epoxy resins;
- plasticisers (phosphate esters);
- rubber and plastic antioxidants;
- dyes;
- deodorising and odour-enhancing compounds and fragrances;
- disinfectants and preservatives; and
- photographic chemicals and explosives.

The following non-industrial uses have been identified internationally including:

- in manufacturing herbicides and pharmaceuticals;
- as a disinfectant, bacteriocide, and germicide for animal pathogenic bacteria; and
- as a bacteriocide and bacteriostat in treating crown gall (*Agrobacterium tumefaciens*) by bark treatment for agricultural use (fruit crops).

Restrictions

Australian

All chemicals in this group are listed in the Poisons Standard (Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP, 2013)) in Schedule 6 as follows:

'PHENOL, including cresols and xylenols and any other homologue of phenol boiling below 220°C, **except**:

(a) when separately specified in these Schedules;

(b) when included in Schedule 5; or

(c) in preparations containing 3 per cent or less of such substances'.

Schedule 6 chemicals are labelled with 'Poison'. These are substances with a moderate potential for causing harm, the extent of which can be reduced by using distinctive packaging with strong warnings and safety directions on the label.

The chemicals are also listed in schedules 2, 4 and 5 for non-industrial uses. The Schedule 5 entry relates to use in animal feed.

International

The chemicals are listed on the following (Galleria Chemica):

- Health Canada List of prohibited and restricted cosmetic ingredients (The Cosmetic Ingredient "Hotlist") as 'Mixed cresols (1319-77-3) and derivatives'.
- The Japanese Ministry of Health, Labor, and Welfare has put restrictions on use of cresol (rinse-off and leave-on products) as a preservative in cosmetics to 0.010 g per 100 g (0.0001 %). Cresols are also not permitted for use in cosmetics that are applied on mucous membranes (CIR, 2006).

- The US Cosmetic Ingredient Review (CIR) Expert Panel concluded that o-cresol (CAS No: 95-48-7) and m-cresol (CAS No: 108-39-4) are safe at concentrations up to 0.5 % in cosmetics; however, the available data are insufficient to support the safety of p-cresol (CAS No: 106-44-5) and mixed cresols (CAS No: 1319-77-3) in cosmetic products (CIR, 2006). This conclusion was based on concerns of chemical leukoderma (depigmentation).

Existing Worker Health and Safety Controls

Hazard Classification

Cresols (CAS Nos: 95-48-7, 106-44-5, 108-39-4, 1319-77-3) are hazardous, with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia):

T; R24/25 (Acute toxicity)

C; R34 (Corrosive)

Exposure Standards

Australian

Cresol (CAS No: 1319-77-3) has an exposure standard of 22 mg/m³ (5 ppm) time weighted average (TWA).

International

The following exposure standards are identified (Galleria Chemica):

Cresol (CAS No: 1319-77-3) has an exposure limit (TWA) of 22 mg/m³ (5 ppm) in countries such as Canada, Denmark, Iceland, Ireland, Japan, Norway, Spain, Switzerland, and the United States of America.

Cresol (CAS No: 1319-77-3) also has an exposure limit (STEL) of 9–22 mg/m³ (2–10 ppm) in countries such as Canada, Sweden, Switzerland, and USA.

The American Conference of Governmental Industrial Hygienists (ACGIH) recommends a TLV of 20 mg/m³ TWA (measured as inhalable fraction and vapour). This value is based on upper respiratory tract irritation. A skin notation has also been assigned (ACGIH, 2011).

Health Hazard Information

Cresol (CAS No: 1319-77-3) is a mixture of three individual isomers; technical grade cresol available in the USA contains about 20 % o-cresol (CAS No: 95-48-7), 30 % p-cresol (CAS No: 106-44-5), and 40 % m-cresol (CAS No: 108-39-4). Even though limited data are available for cresol (CAS No: 1319-77-3), considering that cresol is a mixture of three individual isomers, human health hazards for cresol are expected to take account of the properties of each of the three individual isomers, as indicated in this report.

The toxicity of the cresols is clearly dependent on the mode of administration, indicative of acute toxicity due to corrosive effects. While a gavage treatment with m-cresol (CAS No: 108-39-4) and p-cresol (CAS No: 106-44-5) resulted in an oral LD50 values of 242 and 207 mg/kg bw/day, respectively; a dietary two year carcinogenicity study used a maximum dose of 720 mg/kg bw/day of m/p-cresol (60:40) in rats without reports of acute lethality. In reproductive and developmental toxicity studies, treatment with the chemical through gavage at 450 mg/kg bw/day for each individual isomer in corn oil was without acute lethality. However, systemic signs of toxicity in reproductive and developmental toxicity studies occurred at lower doses (30 mg/kg bw/day) than in the dietary studies.

Toxicokinetics

The chemicals in this group are absorbed across the respiratory and gastrointestinal tract and also through intact skin in animals and humans. After absorption, the chemicals are mainly metabolised by the liver; conjugation with glucuronic acid and inorganic sulphate is the primary metabolic pathway. Minor pathways include hydroxylation of the benzene ring and side-chain oxidation. Following oral administration (gavage) of all three isomers under fasting conditions in rabbits, a minimum of 65–84 % of the administered dose was absorbed within 24 hours, based on the recovery of the same amount in the urine. About 60–72 % of the administered dose was recovered in the urine as glucuronide and 10–15 % as ethereal sulfate. Although urinary excretion in the form of conjugates is the main route for elimination from the body, chemicals of this group are also excreted in the bile and then reabsorbed from the gut (enterohepatic circulation) (EC, 2002; OECD, 2005; ATSDR, 2008; NTP, 2008).

Acute Toxicity

Oral

All chemicals in this group are classified as hazardous with the risk phrase ‘Toxic if swallowed’ (T; R25) in HSIS (Safe Work Australia). The available data support this classification for the cresol group as defined in HSIS.

The reported oral median lethal dose (LD50) in rats was 121 mg/kg bw for undiluted o-cresol (CAS No: 95-48-7), 207 mg/kg bw for undiluted p-cresol (CAS No: 106-44-5), and 242 mg/kg bw for undiluted m-cresol (CAS No: 108-39-4) (WHO, 1995; OECD, 2001; ATSDR, 2008; NTP, 2008; REACH). Reported 95 % confidence intervals range were 172–250 for p-cresol (CAS No: 106-44-5) and 190–308 for m-cresol (CAS No: 108-39-4). Even though limited data are available for cresol (CAS No: 1319-77-3), considering that cresol is a mixture of three individual isomers, it is expected that the LD50 for cresol would be between 121–242 mg/kg bw. Reported sublethal signs of toxicity include hypoactivity, tremors, convulsions, salivation, dyspnoea and prostration.

Dermal

All chemicals in this group are classified as hazardous with the risk phrase ‘Toxic in contact with skin’ (T; R24) in HSIS (Safe Work Australia). While the available data for p-cresol (CAS No: 106-44-5) and human incidences support this classification, data for the other two chemicals (o-cresol-CAS No: 95-48-7, m-cresol-CAS No: 108-39-4) do not. However, in the absence of more comprehensive information, the available data are not sufficient to recommend amendment of the current HSIS classification.

The reported dermal median lethal dose (LD50) in rabbits was 890 mg/kg bw for o-cresol (CAS No: 95-48-7), 300 mg/kg bw for p-cresol (CAS No: 106-44-5), and 2050 mg/kg bw for m-cresol (CAS No: 108-39-4). The reported dermal LD50 in rats was 620 mg/kg bw for o-cresol (CAS No: 95-48-7), 750 mg/kg bw for p-cresol (CAS No: 106-44-5), and 1100 mg/kg bw for m-cresol (CAS No: 108-39-4) (WHO, 1995; OECD, 2001; OECD, 2005; ATSDR, 2008; REACH). Even though limited data are available for cresol (CAS No: 1319-77-3), considering that cresol is a mixture of three individual isomers, it is expected that the LD50 for cresol would be 300–2050 mg/kg bw. Reported sublethal signs of toxicity include hypoactivity, tremor, convulsion, salivation, dyspnoea and prostration.

Inhalation

Limited data for acute toxicity following inhalation exposure are available.

Although median lethal concentrations (LC50) of 29, 29, and 58 mg/m³ (equivalent to 0.029–0.058 mg/L) are reported in rats for o-cresol (CAS No: 95-48-7), p-cresol (CAS No: 106-44-5), and m-cresol (CAS No: 108-39-4), respectively and an LC50 of 178 mg/m^3 (0.178 mg/L) in mice has been reported for aerosolised o-cresol; exposure periods are not indicated (OECD, 2001; EC, 2002). It has also been reported that exposure of rats to 710 mg/m³ (equivalent to 0.71 mg/L) of p-cresol (CAS No: 106-44-5) and m-cresol (CAS No: 108-39-4) for one hour caused no mortality and no signs of intoxication were observed. Observed

sublethal effects included irritation of mucous membranes, and neuromuscular excitation that progressed from tremors to clonic convulsions. Haematuria was reported at very high concentrations (OECD, 2001; EC, 2002; ATSDR, 2008, ACGIH, 2011).

Observation in humans

Cases of intentional or accidental ingestion of chemicals in this group by humans resulting in deaths have been reported. There are also two case reports of humans who died following dermal exposure to cresols. Reported effects included irritation of the mouth and throat, abdominal pains, vomiting, increased heart rate, respiratory failure, liver and kidney damage, methaemoglobinaemia, headaches, facial paralysis, drowsiness, cramps, coma and death. It is likely that most of these effects may have resulted from secondary reactions to shock caused by internal burns. The human lethal dose is reported to be 50–500 mg/kg bw (WHO, 1995; OECD, 2005; ATSDR, 2008; NTP, 2008). Some examples of these cases are as follows.

A man died 15 minutes after ingesting a mixture of p-cresol (CAS No: 106-44-5) and m-cresol (CAS No: 108-39-4). A woman died four days after consumption of 250 mL of a disinfectant (approximately 2 g/kg); described as 50 % cresols in a mixture of linseed oil, potassium hydroxide, and water. Death was due to multiple thrombosis and renal failure resulting from acute intravascular haemolysis. A woman recovered after drinking a smaller amount (approximately 100 mL) of the same disinfectant. A woman died from cardiac arrest 26 hours after consumption of 500–750 mL of a concentrated cresol mixture (WHO, 1995; OECD, 2005; CIR, 2006; ATSDR, 2008; NTP, 2008).

A one-year-old baby died while in a coma within four hours after (20 mL of) a cresol derivative (90 % mixed cresols in water) was spilled on his head, covering about 7 % of his body surface. Assuming that all the cresol was absorbed, the lethal dose was estimated to be roughly 2 g/kg bw. However, as the child’s head was washed with soap and water five minutes after the spill, the lethal dose was probably less. In another case, a man died on the tenth day following a fall into a vat of a cresylic acid derivative (cresol content unknown) and suffered burns on 15 % of the body surface. Another fatal case involved a man who worked for two days with an antiseptic solution containing concentrated mixed cresols; dermal absorption of cresol was stated to be responsible for his death (WHO, 1995; CIR, 2006; ATSDR, 2008; NTP, 2008).

Corrosion / Irritation

Corrosivity

All chemicals in this group are classified as hazardous with the risk phrase 'Causes burns' (C; R34) in the HSIS (Safe Work Australia). The available animal and human data support this classification (OECD, 2001; EC, 2002; OECD, 2005; CIR, 2006; ATSDR, 2008; REACH).

Following single application (0.5 mL) of o-cresol (CAS No: 95-48-7) to the clipped skin of rabbits for four hours, corrosive effects were observed. Although scores were not reported, necrosis and severe oedema were noted at four hours, eschar formation (corrosive) at 24 hours, and slight loosening about the edges of scab (showing injury in depth) was noted at 14 days. In another study, application of 0.5 ml of undiluted o-cresol (CAS No: 95-48-7) on shaved intact or abraded rabbit skin resulted in a mean irritation score of eight out of maximum of eight (REACH).

Application of the undiluted p-cresol (CAS No: 106-44-5) and m-cresol (CAS No: 108-39-4) to the intact or abraded skin of six rabbits (for each chemical) caused severe erythema and oedema in each rabbit within 24 hours. These lesions did not disappear by 72 hours and a mean score of eight out of maximum of eight was noted (OECD, 2005; REACH). In another study, the observed tissue damage was also indicative of corrosive effects during the application of p-cresol (CAS No: 106-44-5) and m-cresol (CAS No: 108-39-4) for four hours under semi-occlusive dressing. An undiluted mixture of p-cresol (CAS No: 106-44-5) and m-cresol (CAS No: 108-39-4) was also classified as corrosive in rabbits, as necrosis with severe oedema was noted at four hours following application, and eschar formation within 24 hours (OECD, 2005).

In another study, all three isomers (o-cresol-CAS No: 95-48-7, p-cresol-CAS No: 106-44-5, m-cresol-CAS No: 108-39-4) were applied to epilated or clipped areas on the backs of CBA/J agouti female mice and C57BL/6J male mice three times weekly for six weeks. After the study concluded, the skin and hair samples were examined microscopically. There was no effect on the skin and hair using 0.5 % m-cresol (CAS No: 108-39-4) and 0.5 % o-cresol (CAS No: 95-48-7) after six weeks of application. However, depigmentation and a local caustic and corrosive effect on black mice was noted following repeated application of 0.5 % p-cresol (CIR, 2006).

Respiratory Irritation

Respiratory tract irritation has been observed in animals following single or short-term exposure to a vapour/aerosol mixture of o-cresol (CAS No: 95-48-7) in concentrations ranging from 5–10 to 178 mg/m³. Histopathological changes in the nasal cavity, either through delivery to the nose of the chemicals in feed or of exposure to the vapours of the chemicals, have also been noted in repeated dose feeding studies of p-cresol (CAS No: 106-44-5) and a mixture of p-cresol (CAS No: 106-44-5) and m-cresol (CAS No: 108-39-4) (EC, 2002).

Eye Irritation

Corrosive chemicals are also considered to cause irreversible effects on the eyes; the available eye irritation data for the chemical support this finding.

In an eye irritation study, o-cresol (CAS No: 95-48-7) was administered into the eyes of six rabbits. The eyes were observed up to 72 hours following exposure. A mean score of 97 out of maximum of 110 was achieved. Similarly, administration of 0.1 mL of p-cresol (CAS No: 106-44-5) and m-cresol (CAS No: 108-39-4) caused highly irritating effects in the cornea, iris, and conjunctivae of all six treated rabbits. The mean irritation score at 72 hours was 93 and 87.3 out of maximum 110, respectively, for p-cresol (CAS No: 106-44-5) and m-cresol (CAS No: 108-39-4) (WHO, 1995; OECD, 2001; EC, 2002; OECD, 2005; ATSDR, 2008, REACH).

Observation in humans

Humans have reported corrosive damage to the skin following dermal exposure. Disfiguring scars remained visible in one patient up to one year following exposure. However, skin reaction to cresol was not noted following application as a 1 % solution in alcohol (WHO, 1995; ATSDR, 2008; HSDB).

Burns at the site of dermal exposure (on the scalp) were noted in a one-year-old baby following exposure to 20 mL of a 90 % cresol solution. Additional clinical signs of toxicity included haemorrhagic pulmonary oedema, liver and kidney necrosis, swelling of the brain, and death within four hours (WHO, 1995; CIR, 2006; NTP, 2008; ATSDR, 2008).

In human maximisation tests, o-cresol (CAS No: 95-48-7) and p-cresol (CAS No: 106-44-5) tested negative for skin irritation using a 48-hour closed patch tests at 4 % concentration in petrolatum (CIR, 2006). The mucosal irritation threshold for o-cresol (CAS No: 95-48-7) in humans (8/10) has been determined to be 6 mg/m³ (OECD, 2001; HSDB).

Sensitisation

Skin Sensitisation

Although limited information is available on the skin sensitisation potential of these chemicals, based on the available information, chemicals in this group are not likely to be skin sensitisers.

The chemical p-Cresol (CAS No: 106-44-5) did not produce skin sensitisation in guineas pigs in a modified Draize test, using induction and challenge concentrations of 0.1 % and 10 %, respectively (EC, 2002; OECD, 2005; REACH). Sensitisation was also not observed in guinea pigs following repeated application of a 7.5 % solution of a mixture of p-cresol (CAS No: 106-44-5) and m-cresol (CAS No: 108-39-4) in acetone (EC, 2002; OECD, 2005).

Observation in humans

The chemical p-Cresol (CAS No: 95-48-7) tested negative for skin sensitisation effects in 25 humans using a 4 % concentration (CIR, 2006). Similarly, p-cresol (CAS No: 106-44-5) tested negative for skin sensitisation in a maximisation test conducted on 25 humans using a 4 % concentration of the chemical in petrolatum (OECD, 2005; REACH). Positive effects to 2 % m-cresol (CAS

No: 108-39-4) were noted only in 2/81 patients, who were sensitive to textile dyes (CIR, 2006). Ten humans, who had hand dermatitis, were patch tested using different concentrations of o-cresol (CAS No: 95-48-7) and p-cresol (CAS No: 106-44-5) (in ethanol). Four patients tested positive to 81 % o-cresol (CAS No: 95-48-7), three tested positive to 8.1 % o-cresol (CAS No: 95-48-7), and there were no effects at 0.81 % o-cresol (CAS No: 95-48-7). One patient tested positive at 81 % p-cresol (CAS No: 106-44-5) and there were no effects at 8.1 % p-cresol (CAS No: 106-44-5) (CIR, 2006).

Repeated Dose Toxicity

Oral

Although the chemicals in this group are not considered to cause serious damage to health from repeated oral exposure, lesions have been noted, possibly due to the corrosive nature of these chemicals.

The nasal respiratory epithelium of rats and mice appears to be the most sensitive target for the toxicity of the chemicals in this group. Although it has been suggested that the nasal lesions probably arose by evaporation of the chemicals from the feed and thus there was direct contact of the airborne chemicals with the nasal respiratory epithelium, the systemic effect of chemicals in this group has also not been ruled out (ASTDR, 2008).

Dose-related alterations in the nasal respiratory epithelium of rats and mice were noted following dietary exposure to p-cresol (CAS No: 106-44-5) or to a mixture of m/p-cresol at a 60:40 ratio for 28 days, 13 weeks, and in a two-year carcinogenicity study (see **Carcinogenicity**). However, nasal epithelial lesions were not observed following exposure to o-cresol (CAS No: 95-48-7) or m-cresol (CAS No: 108-39-4) (NTP, 1992; OECD, 2001; EC, 2002; OECD, 2005; NTP, 2008).

The nervous system (see **Neurotoxicity**) as well liver and kidney (increases in liver and kidneys weights), have also been proposed as sensitive targets during repeated oral exposure to chemicals in this group. Although altered liver and kidney functions as well as gross microscopic alterations were not noticed in the intermediate duration studies, even at the highest doses administered (>1000 mg/kg bw/day), histopathological changes were observed in the two-year carcinogenicity study, as described below (see **Carcinogenicity**) (NTP, 1992; OECD, 2001; EC, 2002; OECD, 2005; NTP, 2008).

Several studies have reported repeated oral dose toxicities. The most comprehensive studies in rats and mice were undertaken by National Toxicology Program (NTP), where chemicals in this group were administered for 28 days or 13 weeks (NTP, 1992).

In the 13-week repeated dose toxicity study, groups of F344 rats were fed diets containing 0, 1880, 3750, 7500, 15000, or 30000 ppm o-cresol (CAS No: 95-48-7) and an m/p-cresol mixture (60:40 ratio). The estimated corresponding dose for m/p-cresol was found to be 0, 123, 241, 486, 991, and 2014 mg/kg/day for males and 0, 131, 254, 509, 1024, and 2050 mg/kg bw/day for females. In the same study, groups of B6C3F1 mice were fed diets containing 0, 1250 (199 and 237 mg/kg bw in males and females, respectively), 2500, 5000, 10000, or 20000 ppm o-cresol (CAS No: 95-48-7) and 0, 625, 1250, 2500, 5000, or 10000 ppm m/p-cresol for 13 weeks. Nasal respiratory epithelium irritation was noted in rats and mice receiving feed containing m/p-cresol. Haematology, clinical chemistry, and urinalysis were not affected during the study and there were no deaths. Mild bone marrow hypocellularity in rats and forestomach hyperplasia in mice were noted in animals receiving feed containing the higher concentrations of o-cresol (CAS No: 95-48-7). The severity of nasal respiratory epithelium irritation by o/p cresol was minimal for males/females at 123/131 mg/kg bw/day, mild at 486/509 mg/kg bw/day, and moderate at 2014/2050 mg/kg bw/day (NTP, 1992; OECD, 2001; EC, 2002; OECD, 2005; NTP, 2008).

Groups of F344 male rats were fed diets containing 0, 1500, 5000 or 15000 ppm of 60:40 m/p-cresol (equivalent to approximately 70, 230, or 720 mg cresols/kg bw/day) for 105 weeks in a carcinogenicity study (see **Carcinogenicity**). In the same study, groups of B6C3F1 female mice were also fed diets containing 0, 1000, 3000, or 10000 ppm of 60:40 m/p-cresol (equivalent to approximately 100, 300, or 1040 mg cresols/kg bw/day) for 104–105 weeks. While the mean body weights of the groups fed the highest concentration of cresols were lower than the control groups, survival of animals in all exposed groups was not affected by treatment with the chemicals (NTP, 2008).

A number of non-neoplastic lesions were observed in this study. In rats, significantly increased incidence of hyperplasia of the goblet cells and respiratory epithelium of the nose were noted in all exposed animals. While the incidence of squamous metaplasia of the respiratory epithelium was significantly increased in the two highest concentration groups, the incidence of inflammation was significantly increased only in the rats fed the highest concentration of the chemical. The incidence of hyperplasia of the transitional epithelium of the renal pelvis was significantly increased in the rats fed the highest concentration

of the chemicals; the severity of nephropathy was slightly increased and the incidence of eosinophilic focus of the liver was significantly increased in this group.

In mice, the incidences of respiratory epithelial hyperplasia of the nose were significantly increased in the two highest concentration groups. Significantly increased incidence of bronchiolar hyperplasia of the lung were also noted in all exposed mice. The incidences of thyroid gland follicular degeneration were significantly increased in all exposed groups of mice, while the incidence of eosinophilic focus of the liver was significantly increased in the group fed the highest concentration of the chemicals.

Dermal

No data are available.

Inhalation

Limited information is available on the effects of repeated inhalation exposure of these chemicals.

Exposure of rats to vapours of o-cresol (CAS No: 95-48-7) at 9 mg/m³ for 4–6 hours/day, five days/week for four months, resulted in behavioural depression, leucocytosis, decreased erythroid/myeloid ratio in the bone marrow, increased hexobarbital narcosis time, and morphological changes in respiratory tissues (inflammation and irritation of the upper respiratory tract, oedema, and perivascular sclerosis in the lungs) (WHO, 1995; OECD, 2001).

In another study, mice were exposed to vapours and aerosols of o-cresol (CAS No: 95-48-7) for two hours/day, six days/week for one month, with an average concentration of 50 mg/m³. Clinical signs of toxicity were observed during the exposure periods only and included signs of respiratory irritation, hypoactivity, reduced weight gain, and mummification of the tails of some animals. There were no deaths. Other lesions included degeneration of heart muscle, liver, kidney and nerve cells, and glial elements of the central nervous system (WHO 1995; OECD, 2001; CIR, 2006).

Administration of individual cresol isomers or a mixture of isomers (isomers not specified) by the inhalation route for three to four months in rats at doses ranging from 0.05 to 10 mg/m³ resulted in decreased body weight gain, and histological alterations in the liver and kidney (WHO, 1995).

Genotoxicity

Overall, the data indicate that the chemicals in this group have no mutagenic or genotoxic potential in vivo. Although positive results were obtained for chemicals in this group with some human and animal in vitro tests, there was no evidence for a genotoxic activity in vivo. These results also suggest that chemicals in this group have some potential to interact with DNA and may be clastogenic under certain circumstances (OECD, 2001; EC, 2002; OECD, 2005; ATSDR, 2008; REACH).

Chemicals in this group (o-cresol, CAS No: 95-48-7; p-cresol, CAS No: 106-44-5; m-cresol, CAS No: 108-39-4) did not induce gene mutations in bacterial and mammalian cell systems. The chemicals o-Cresol (CAS No: 95-48-7) and p-cresol (CAS No: 106-44-5) induced chromosome aberration using Chinese Hamster Ovary (CHO) cells in vitro. While m-cresol (CAS No: 108-39-4) was negative for chromosome aberration in CHO cells, positive results were produced in Syrian hamster embryo cells.

While o-cresol (CAS No: 95-48-7) and a mixture of three individual isomers produced positive results for sister chromatid exchange (SCE) in CHO cells and Syrian hamster embryo cells, negative results for sister chromatid exchange in human fibroblasts were obtained for the three individual isomers. The chemical p-Cresol (CAS No: 106-44-5) and a mixture of three individual isomers produced cell transformation in mouse BALB/C-3T3 cells, while o-cresol (CAS No: 95-48-7) and m-cresol (CAS No: 108-39-4) did not.

Although the three individual isomers were negative for forward mutation in mouse lymphoma cells, a mixture of the three isomers at a 1:1:1 ratio tested positive. Increased DNA synthesis was noticed in Syrian hamster embryo cells with metabolic activation for m-cresol (CAS No: 108-39-4) and in human peripheral lymphocytes for p-cresol (CAS No: 106-44-5), but was not seen in rat hepatocytes for o-cresol (CAS No: 95-48-7) and m-cresol (CAS No: 108-39-4). o-Cresol (CAS No: 95-48-7) induced DNA damage in mouse spermatid and human peripheral lymphocytes. DNA adduct formation was noted in rat hepatocytes and

HL-60 cells during incubation with p-cresol (CAS No: 106-44-5). In addition, a weakly positive result for induction of infectious virus particles from SV40-transformed weanling Syrian hamster kidney cells was reported for m-Cresol (CAS No: 108-39-4)

Studies of the genotoxicity of chemicals in this group in animals (in vivo) showed negative results: o-cresol (CAS No: 95-48-7) and a mixture (60:40) of m-cresol (CAS No: 108-39-4) and p-cresol (CAS No: 106-44-5) caused no induction of micronuclei in peripheral blood erythrocytes of mice; m-cresol (CAS No: 108-39-4) did not induce clastogenic activity in mouse bone marrow cells even at clearly toxic dose levels (up to 960 mg/kg bw by gavage); o-cresol (CAS No: 95-48-7) and p-cresol (CAS No: 106-44-5) produced no dominant lethal effects in mice; o-cresol (CAS No: 95-48-7) did not increase SCE in mouse bone marrow, lung or liver cells during in vivo studies, and p-cresol (CAS No: 106-44-5) also did not induce significant increases in SCE frequencies in any of the cell types examined in mice after intraperitoneal injection.

Carcinogenicity

Although there is some evidence of carcinogenicity in animals, the relevance of this to humans is considered questionable.

In a carcinogenicity study, groups of F344 male rats were fed diets containing 0, 1500, 5000 or 15000 ppm of 60:40 m/p-cresol (equivalent to approximately 70, 230, or 720 mg cresols/kg bw/day) for 105 weeks (see **Repeat dose toxicity: oral**). In the same study, groups of B6C3F1 female mice were also fed diets containing 0, 1000, 3000, or 10000 ppm of 60:40 m/p-cresol (equivalent to approximately 100, 300, or 1040 mg cresols/kg bw/day) for 104–105 weeks (NTP, 2008).

A marginally nonstatistically significant increased incidence of renal tubule adenoma was noted in male rats at the top dose tested. The occurrence of renal tubule adenoma in this study was considered as ‘equivocal findings’. The incidence of squamous cell papilloma of the forestomach was significantly greater in mice fed the highest concentration of the chemicals than in the control group. The increased incidences of squamous cell papilloma of the forestomach were considered as neoplastic effects (NTP, 2008). Tumours of the forestomach, following administration by gavage of an irritating or corrosive non-mutagenic chemical, is considered of questionable relevance to humans (GHS, 2013).

Reproductive and Developmental Toxicity

The chemicals in this group do not cause specific reproductive or developmental toxicity. Any reproductive and developmental effects were only observed secondary to maternal toxicity (WHO, 1995; OECD, 2001; EC, 2002; OECD, 2005; ATSDR, 2008; REACH).

In a two-generation reproductive toxicity study, each individual isomer was administered (gavage) to separate groups of Sprague Dawley (SD) rats at 0, 30, 175 or 450 mg/kg bw/day in corn oil for 10 weeks before breeding. All three isomers caused toxic effects in the parental animals, which were mostly limited to the 450 mg/kg bw/day groups in F0 rats. These effects included reduced body weight gain, hypoactivity, ataxia, twitches, tremors, prostration, rapid and laboured respiration, urine stains, perioral wetness, and death. Some clinical signs of toxicity also occurred in the 175 mg/kg bw/day groups in F1 rats. Reproductive function as well as the morphology of reproductive tissues were not affected in these studies, even at doses producing overt parental toxicity. While the NOAEL for fertility was 450 mg/kg bw/day, the NOAEL for general toxicity was 30 mg/kg bw/day. Similarly no effects on reproductive parameters were observed in a two-generation study in mink. Whilst significantly decreased numbers of live pups/litter and increased cumulative days to litter were observed in CD-1 mice in a continuous breeding protocol study with a mixture of m/p cresol, this was only at high doses. The NOAEL was 1390 mg/kg bw/day. No effects were observed in a similar study with o-cresol (highest dose 660 mg/kg bw/day) (WHO, 1995; OECD, 2001; EC, 2002; OECD, 2005; ATSDR, 2008; REACH).

In a 13-week repeated dose study in mice and rats (see **Repeat dose toxicity: oral)**, no adverse effects on the male reproductive system or in sperm parameters were observed in either species. However, the oestrus cycle was lengthened in rats and mice receiving the higher concentrations of o-cresol (CAS No: 95-48-7) and in rats receiving a mixture of p-cresol (CAS No: 106-44-5) and m-cresol (CAS No: 108-39-4) at as low as 7500 ppm (509 mg/kg bw/day) (NTP, 1992; ATSDR, 2008).

A number of studies investigating developmental toxicity are available. Whilst effects of the different isomers were not consistent between studies, in all cases effects were only observed in the presence of maternal toxicity. Effects observed included reduced pup survival and mild foetotoxic effects (including increased incidences of dilated lateral ventricles in the brain and minor skeletal variations). The lowest reported NOAEL for developmental effects was 175 mg/kg bw/day in rats and 50 mg/kg bw/day in rabbits (WHO, 1995; OECD, 2001; OECD, 2002; OECD, 2005; ATSDR, 2008; REACH).

Other Health Effects

Neurotoxicity

In a neurotoxicity study, rats were treated with 0, 50, 175 and 600 mg/kg bw/day of o- and p-cresols or 0, 50, 150 and 450 mg/kg bw/day of m-cresol by gavage in corn oil for 13 weeks. Convulsions were seen only in the groups treated with ≥450 mg/kg bw/day of the isomers. Animals treated with all three isomers showed hypoactivity, rapid laboured respiration, and excessive salivation sporadically at doses of ≥50 mg/kg bw/day. However, significant changes were not found in performance on neurobehavioural test batteries; brain weights; on gross and histopathological lesions in the brain or other nervous tissues in animals treated with any of the isomers. While m-cresol was not lethal at the highest tested dose of 450 mg/kg bw/day, both o-cresol and p-cresol caused mortality at the highest tested dose of 600 mg/kg bw/day (WHO, 1995).

The development of coma in humans following oral poisoning with cresols and adverse clinical signs indicative of neurological impairment (hypoactivity, excessive salivation, laboured respiration, and tremors) have also been reported in rodents following oral gavage. However, gross or microscopic alterations of the brain, spinal cord, or sciatic nerve have never been observed in rodents. It has also been noted that neurological signs seen in oral gavage studies in animals have not been seen in dietary studies or they have occurred at much higher dose levels than in oral gavage studies (ATSDR, 2008).

In a study comparing the effects of m-cresol on neonates and young rats, adverse neurological effects were observed at much lower doses in neonates. The NOAEL was 30 mg/kg bw/day for the neonates compared with 300 mg/kg bw/day in young rats. Observed effects included deep respiration, hypersensitivity on handling, and tremors under contact stimulus (ASTDR, 2008).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include systemic acute effects (acute toxicity by the oral and dermal routes of exposure) and local effects (corrosivity) in a concentration dependent manner.

Although the chemicals can produce neurological effects and there is some evidence of carcinogenicity in animals (with a low likelihood of relevance to humans) at high doses, these are not considered relevant at doses and or routes of exposure relating to occupational exposure.

Public Risk Characterisation

Even though the chemicals in this group have reported cosmetic and domestic uses overseas (see **Import, manufacture and use**), the available North American databases do not give evidence for use of the chemicals in this group in cosmetics as well as consumer products. Therefore, the use of the chemicals in this group in cosmetic and consumer products is not anticipated in Australia. Hence, the public risk from this chemical is not considered to be unreasonable.

However, as the current schedule entry for these chemicals is part of the entry for phenol (see **Restrictions: Australia),** NICNAS has recommended that an amendment to the current entry for phenol in the SUSMP be considered (NICNAS), given that necrosis of human skin has been reported at concentrations as low as 1 %. The possibility of a higher degree of local damage by phenol was also raised as it may diminish the sensation of pain and it was also noted that Canada, New Zealand and the European Union have prohibited the use of phenol in cosmetics. Therefore, a consideration of the schedule entry for the present group of chemicals will be necessary.

Occupational Risk Characterisation

During product formulation, dermal, ocular and inhalation exposure of workers to the chemical may occur, particularly where manual or open processes are used. These may include transfer and blending activities, quality control analysis, and cleaning

and maintenance of equipment. Worker exposure to the chemical at lower concentrations may 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 health effects, the chemical may pose an unreasonable risk to workers unless adequate control measures to minimise dermal, ocular 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.

Based on the available data, the hazard classification in HSIS is considered appropriate.

NICNAS Recommendation

Current risk management measures are considered adequate to protect public and workers' health and safety, provided that all requirements are met under workplace health and safety and poisons legislation as adopted by the relevant state or territory. No further assessment is required.

However, NICNAS has recommended a change to the Scheduling of phenol (NICNAS). The current entry for phenol is complex and includes additional chemicals, including the cresols. Any change to the scheduling of phenol will have flow on effects to these other chemicals (see **Restrictions: Australia)**. This report, which is supportive of the current controls for cresols, should be taken into account in determining whether changes in cutoff should apply to the current entry as written in the SUSMP, or whether separate entries for phenol and cresol should be created.

Regulatory Control

Public Health

It is recommended that the current report should be taken into account in determining whether changes in cutoff should apply to the current entry as written in the SUSMP, or whether this entry should be changed to make a single more restrictive entry for phenol alone. Matters for consideration include:

- the chemical has been reported to cause poisoning in humans by ingestion and skin absorption;
- the chemicals are less corrosive than phenol with corrosive effects not seen at or below 3 %;
- the chemical in preparations for external use containing 3 % or less of such substances is currently included in Schedule 2 for therapeutic uses;
- the use of the chemicals in this group in cosmetic and consumer products is not anticipated in Australia; and
- while more restrictive scheduling of phenol has been recommended by NICNAS (NICNAS), the specific considerations requiring reconsideration of the phenol cutoff do not apply to cresols.

Work Health and Safety

The chemicals in this group are 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 if swallowed (T; R25)* Toxic in contact with skin (T; R24)*	Toxic if swallowed - Cat. 3 (H301) Toxic in contact with skin - Cat. 3 (H311)

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Irritation / Corrosivity	Causes burns (C; R34)*	Causes severe skin burns and eye damage - Cat. 1A (H314)

[a] Approved Criteria for Classifying Hazardous Substances [NOHSC:1008(2004)].

[b] Globally Harmonized System of Classification and Labelling of Chemicals (GHS) United Nations, 2009. Third Edition.

[*] Existing Hazard Classification. No change recommended to this classification

Advice for consumers

Products containing the chemical should be used according to the instruction on the label.

Advice for industry

Control measures

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

- using closed systems or isolating operations;
- using local exhaust ventilation to prevent the chemical from entering the breathing zone of any worker;
- health monitoring for any worker who is at risk of exposure to the chemical if valid techniques are available to monitor the effect on the worker's health;
- air monitoring to ensure control measures in place are working effectively and continue to do so;
- minimising manual processes and work tasks through automating processes;
- work procedures that minimise splashes and spills;
- regularly cleaning equipment and work areas; and
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the chemical.

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

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

Obligations under workplace health and safety legislation

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

- ensuring that hazardous chemicals are correctly classified and labelled;

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

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

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

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

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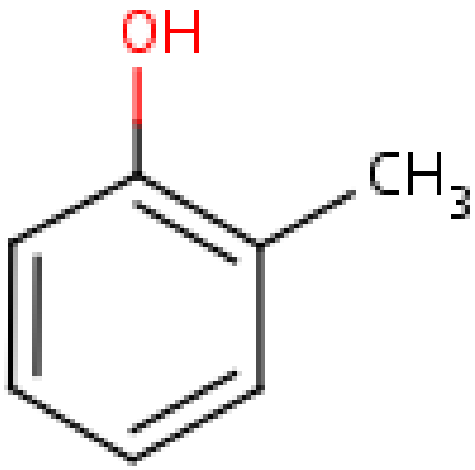
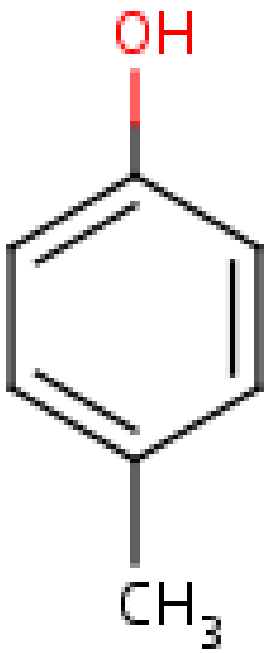
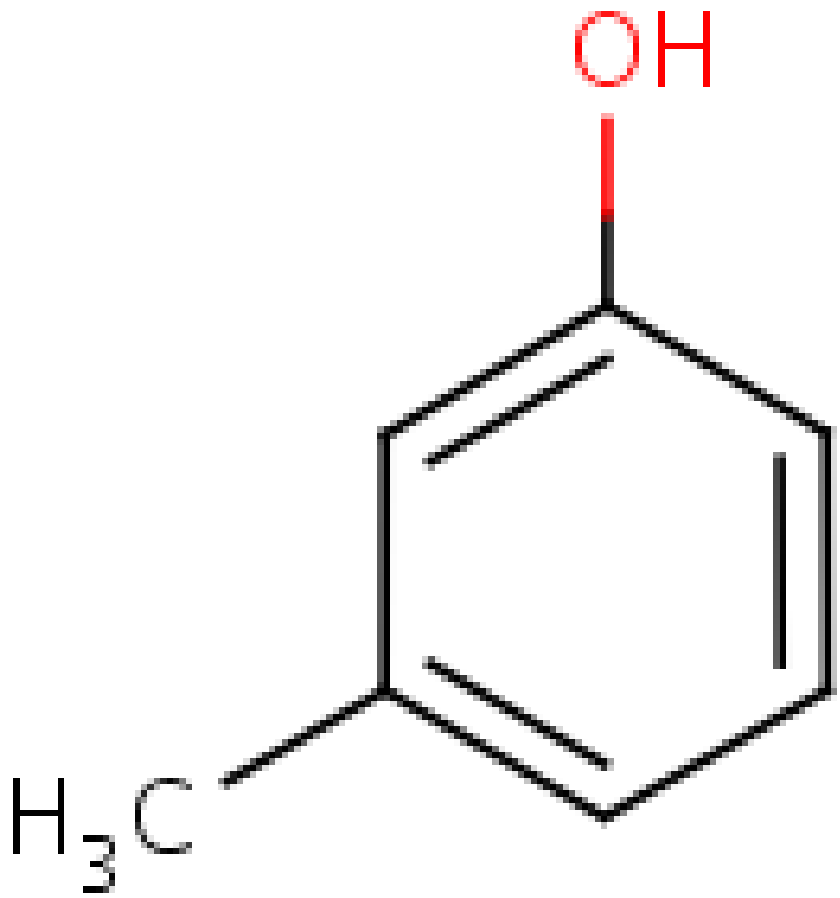
Last Update 11 April 2014

Chemical Identities

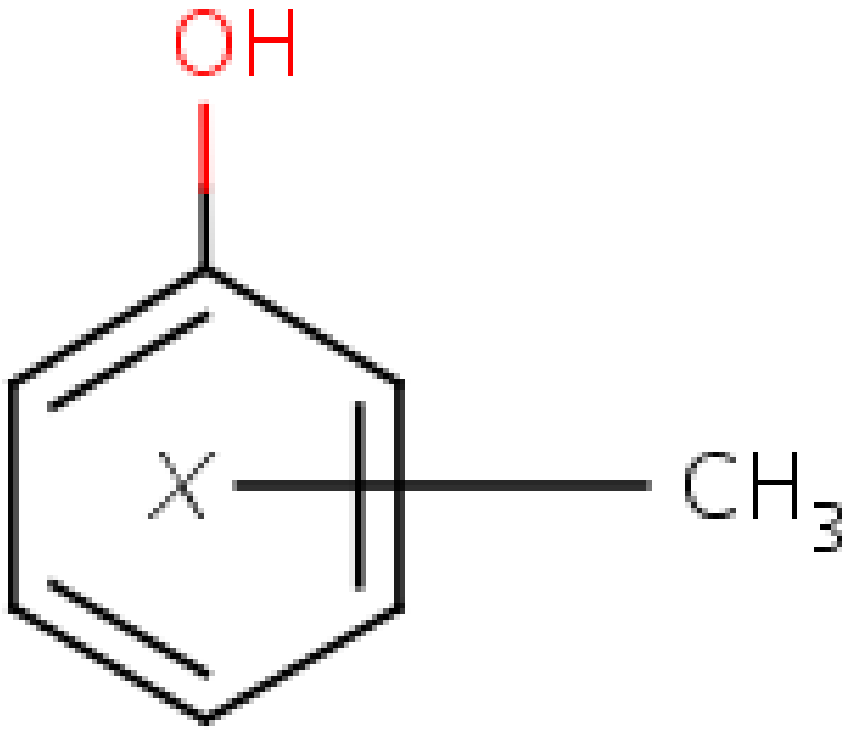
Chemical Name in the Inventory and Synonyms	**Phenol, 2-methyl-** o-cresol orthocresol 2-methylphenol 2-cresol 2-hydroxytoluene
CAS Number	95-48-7
Structural Formula	
Molecular Formula	C7H8O
Molecular Weight	108.14

Chemical Name in the Inventory and Synonyms	**Phenol, 4-methyl-** p-cresol 4-methylphenol paracresol 4-cresol 4-hydroxytoluene
CAS Number	106-44-5
Structural Formula	
Molecular Formula	C7H8O
Molecular Weight	108.14

Chemical Name in the Inventory and Synonyms	**Phenol, 3-methyl-** m-cresol 3-methylphenol metacresol 3-cresol 3-hydroxytoluene
CAS Number	108-39-4
Structural Formula	

	
Molecular Formula	C7H8O
Molecular Weight	108.14

Chemical Name in the Inventory and Synonyms	**Phenol, methyl-** cresol mixed cresols cresylic acid hydroxytoluene methylphenol, mixed
CAS Number	1319-77-3
Structural Formula	

	
Molecular Formula	C7H8O
Molecular Weight	108.14

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