Nonylphenols: Human health tier II assessment

08 March 2019

Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
Phenol, 4-nonyl-	104-40-5
Phenol, 2,4-dinonyl-	137-99-5
Phenol, dinonyl-	1323-65-5
Phenol, 2-methyl-4,6-dinonyl-	3011-61-8
Phenol, isononyl-	11066-49-2
Phenol, 4-methyl-2-nonyl-	13207-27-7
Phenol, 4-nonyl-2,6-bis(1-phenylethyl)-	15860-96-5
Phenol, nonyl-	25154-52-3
Phenol, 2-isononyl-4-methyl-	28983-26-8
Phenol, nonyl-, barium salt	28987-17-9
Phenol, nonyl-, sodium salt	54181-64-5
Phenol, nonyl derivs.	68081-86-7
Phenol, 4-methyl-2,6-dinonyl-	63451-44-5
Barium, carbonate 4-nonylphenol complexes	68442-67-1
Barium, carbonate nonylphenol complexes	68515-89-9

Chemical Name in the Inventory	CAS Number
Phenol, nonyl derivatives, barium salts	68515-91-3
Phenol, nonyl derivs., sulfides	68515-93-5
Phenol, nonyl derivs., sulfides, barium salts	68515-94-6
Phenol, nonyl derivs., sulfides, calcium salts	68515-95-7
Phenol, tri-C6-9-alkyl derivatives	68515-97-9
Calcium, nonylphenol sulfides phosphosulfurized polybutene complexes	68910-15-6
Phenol, nonyl-, zinc salt	77194-15-1
Phenol, 2,4-dinonyl-, branched	84852-14-2
Phenol, 4-nonyl-, branched	84852-15-3
Phenol, dinonyl -, branched	84962-08-3
Phenol, nonyl-, branched	90481-04-2
Phenol, nonyl derivatives, sulfides, magnesium salts	91254-25-0
Phenol, nonyl-, barium salt, basic	93028-52-5

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 are various isomers of nonylphenol (including disubstituted nonylphenols) and their sodium, barium or zinc salts. The term 'nonylphenol' corresponds with various isomeric compounds (>200) with a general formula C6H4(OH)(C9H19)x, varying by the degree of branching of the nonyl group and the substitution position on the phenol ring. Whilst the substitution pattern and branching structure of the alkyl chain is not specified in all of the chemical names, in technical nonylphenol mixtures, branched para-nonylphenol isomers constitute at least 90 % of the final product (APERC, 2016).

The CAS numbers for 4-nonylphenol isomers are often incorrectly labelled by the suppliers (US EPA, 2010). The CAS No. 25154-52-3 was routinely used by the Chemistry Abstract Service (CAS) for all nonylphenols, and this was subsequently redefined to cover only linear (straight chain) nonylphenols. The substance represented by CAS RN 84852-15-3 is a mixture of branched-chain isomers of 4-nonylphenol, largely mono-substituted in the para- position but with small amounts of ortho- and di-substituted nonylphenols (US EPA, 2010). This is the most widely produced nonylphenol. This chemical is often incorrectly identified as one of the two straight-chain nonylphenol substances, CAS RN 25154-52-3 or CAS RN 104-40-5 (EC, 2002; US EPA 2010).

Also included in the assessment are nonyl phenol sulfides. These chemicals are oligomeric mixtures of nonylphenol molecules that are likely linked by one or more sulfur atoms through sulfurisation process. These chemicals are similar to nonyl phenols with respect to their low solubility in water, high octoanol-water partition coefficient, and low vapour pressure. Nonyl phenol sulfides are made by reacting the nonyl phenol with sulfur dichloride in the presence of a highly refined lubricant base oil. These may contain unreacted nonyl phenol or its salts in varying amounts as an impurity (OECD, 2009). There is a lack of information on them. In the absence of data, information available from other nonyl phenols can be used for read across, particularly where there is presence of unreacted nonylphenols.

There is a lack of information on dinonylphenols and other substituted alkyl derivatives. In the absence of information to indicate lower toxicity for this group, they will be assumed to be represented by data on nonylphenols. It is also probable that dinonylphenols and other substituted alkyl derivatives are not pure substances and; therefore, contain significant levels of mono nonylphenols. Dinonyl phenols and other substituted alkyl derivatives have; therefore, been included in this assessment.

The sodium cation is considered to be of low systemic toxicity (NICNASa) and zinc and barium cation only produce systemic effects at high doses (NICNASb; ATSDR, 2007). The toxic properties of the chemicals are expected to be mostly due to the nonylphenol constituent and so very similar hazard profiles for human health are expected.

Import, Manufacture and Use

Australian

The chemical, phenol, nonyl-, barium salt (CAS No 28987-17-9, has reported commercial use in printing inks.

The chemical, phenol, 4-nonyl-, branched (CAS No 84852-15-3), has reported commercial use in spray adhesive products.

The chemical, phenol, nonyl- (CAS No 25154-52-3), has reported commercial use in surface coating products.

No specific Australian use, import, or manufacturing information has been identified for the other chemicals in this group.

International

The following international uses have been identified through the European Union (EU) Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) dossiers; the Organisation for Economic Co-operation and Development (OECD) Screening information data set International Assessment Report (SIAR); the European Commission (EC) Risk Assessment Report; Galleria Chemica; the Substances and Preparations in Nordic countries (SPIN) database; the United States (US) National Library of Medicine's Hazardous Substances Data Bank (HSDB), and the US Environmental Protection Agency (EPA) Action Plan.

The chemicals have reported site-limited use including:

- as intermediates in producing nonylphenol ethoxylates, which are then used in a variety of products (industrial detergents, surfactants, cleaners, degreasers, adhesives, paints and coatings and emulsifiers);
- in producing trisnonylphenylphosphite (TNPP), which is a stabiliser in plastics and polymer production; and
- for manufacturing lubricating oil additives and resins.

Phenol, nonyl derivatives, sulfides (CAS No.68515-93-5) has reported use as a lubricant in engine oil.

Barium nonylphenolate has commercial use as a polymer stabiliser.

Branched 4-nonylphenol (CAS No. 84852-15-3) has reported use as an epoxy cure catalyst. The chemical has reported domestic use in a range of domestic products, including home maintenance epoxy products up to a concentration of 60 % (Household Products Database, US Department of Health and Human Services).

The chemicals have reported non-industrial uses in:

- non-agricultural pesticides; and
- wood preservatives.

Both the EC and the US EPA have stated that nonylphenols are mainly used as intermediates in manufacturing other chemicals that are used in consumer or industrial products (EC, 2002; US EPA, 2010). The EC (2002), states that 'nonylphenol is not used directly in products with which the consumer comes into contact'. However, there could be residual levels of unreacted nonylphenols (impurities) in certain consumer products (EC, 2002). In addition, CAS No. 68081-86-7, was identified in two auto treatment products at concentrations up to 6 %.

Restrictions

Australian

Barium salts are listed in the Poisons Standard in Schedule 6 (SUSMP, 2017).

Schedule 6:

'Barium salts except:

(a) when included in Schedule 5;

(b) barium sulfate; or

(c) in paints or tinters containing 5 percent or less of barium calculated on the non-volatile content of the paint or tinter.'

Schedule 6 chemicals are described as 'Substances with a moderate potential for causing harm, the extent of which can be reduced through the use of distinctive packaging with strong warnings and safety directions on the label'. Schedule 6 chemicals are labelled with 'Poison' (SUSMP, 2017).

International

Nonylphenol and 4-nonylphenol, branched (listed as the CAS Nos 25154-52-3 and 84852-15-3) are listed on the following (Galleria Chemica):

- EU Cosmetics Regulation 1223/2009 Annex II—List of substances prohibited in cosmetic products;
- New Zealand Cosmetic Products Group Standard—Schedule 4: Components cosmetic products must not contain; and
- The Association of Southeast Asian Nations (ASEAN) Cosmetic Directive Annex II Part 1: List of substances which must not form part of the composition of cosmetic products; and
- China List of banned substances for use in cosmetics.

Nonylphenols (with the following CAS numbers listed as members 11066-49-2, 84852-15-3, 104-40-5, 25154-52-3, 90481-04-2) are subject to export notification procedures and prior informed consent notification for imports under Regulation No 649/2012 of the European Parliament and of the Council. Importers and exporters of the chemicals must notify relevant authorities before transportation of the substance (European Commission, 2012).

Nonylphenol (CAS RN 25154-52-3) is restricted through Annex XVII to the REACH Regulations. The chemical cannot be used in substances and preparations placed on the market for sale to the general public in individual concentrations ≥0.1 % for uses such as cleaning, textile and leather processing, cosmetic and personal care products. Branched 4-nonylphenol (CAS RN 84852-15-3) is currently listed on the Community Rolling Action Plan for evaluation under the REACH legislation, based on potential for high environmental exposure (ECHA, 2014).

The US EPA is proposing a significant new use rule (SNUR) for 15 nonylphenols (NPs) and nonylphenol ethoxylates (NPEs). 'Persons subject to these SNURs would be required to notify EPA at least 90 days before they manufacture (including import) or process any of these 15 chemical substances for a significant new use. The required notification would provide EPA with the opportunity to evaluate the new uses and protect against unreasonable risks, if any, from potential new exposures to NPs and NPEs, before that activity occurs' (US EPA, 2014).

Nonylphenols are listed on Schedule 1 of the Canadian Environmental Protection Act 1999 (the Toxic Substances List) (Environment Canada, 2013a). Use of nonylphenols has been phased down in Canada since 2004, with most users of nonylphenols required to prepare and implement pollution prevention plans. The majority have met risk management objectives by eliminating the use of these chemicals (Environment Canada, 2014).

Existing Worker Health and Safety Controls

Hazard Classification

Two of the chemicals (CAS Nos. 25154-52-3 and 84852-15-3) are classified as hazardous, with the following hazard category and hazard statement for human health in the Hazardous Chemical Information System (HCIS) (Safe Work Australia):

- Reproductive toxicity—category 2; H361fd (Suspected of damaging fertility. Suspected of damaging the unborn child)
- Acute toxicity—category 4; H302 (Harmful if swallowed)
- Skin corrosion—category 1B; H314 (Causes severe skin burns and eye damage)

The other chemicals are not listed on the HCIS (Safe Work Australia).

Exposure Standards

Australian

No specific exposure standards are available.

International

No specific exposure standards are available.

Health Hazard Information

The test substance used in the majority of toxicity studies was commercially produced nonylphenols. Data are assumed to be representative for all isomers of nonylphenol and dinonylphenols. The term nonylphenol is used as a generic name referring to the chemicals in this assessment.

Toxicokinetics

Following oral administration in animals, nonylphenol was absorbed rapidly via the gastrointestinal tract and underwent extensive first-pass metabolism. The major metabolic pathways are glucuronide and sulphate conjugation. Nonylphenol was distributed throughout the body, with the highest concentration in body fat. Excretion is primarily through the faeces followed by the urinary route (OECD, 2001; EC, 2002).

Female rats that received oral gavage doses of nonylphenol had a higher mean percentage of the administered dose in the liver compared with males, followed by the lungs, spleen, uterus and ovaries. Elimination half-lives after oral administration were calculated to be 12.4 hours in male rats and 8.5 hours in female rats. Metabolic (glucuronide) saturation was observed in female rats administered nonylphenol at 100 mg/kg bw, where unchanged nonylphenols were recovered in the bile and urine. Oestrogen-like effects observed at oral doses of approximately >50 mg/kg bw/day were reported to be a result of bioavailability of nonylphenol following metabolic saturation (REACH).

On the basis of oral absorption studies and the high partition coefficient, absorption via inhalation was predicted to be significant. Furthermore, as the first-pass metabolism would not take place following inhalation exposure, it was assumed that systemic bioavailability from inhalation exposure would likely be greater than oral exposure. Dermal absorption was perceived to be low, although limited skin penetration into the stratum corneum can occur (OECD, 2001; EC, 2002).

Acute Toxicity

Oral

Two of the chemicals (mixed nonylphenol and 4-nonylphenol branched) are classified as hazardous with hazard category 'Acute Toxicity Category 4' and hazard statement 'Harmful if swallowed' (H302) in the Hazardous Chemical Information System (HCIS) (Safe Work Australia). The available data support this classification. The same classification should be apply for the other chemicals in this report with the exception of phenol, nonyl derivatives, sulfides and its calcium and magnesium salts.

The median lethal dose (LD50) for nonylphenol 1200–2462 mg/kg bw in rats and 1231 mg/kg bw in mice. Reported signs of toxicity in rats included lethargy, hunched posture, ataxia with staggering, tremor, salivation, cyanosis, half or fully closed eyes, hypothermia, ruffled fur and partly swollen abdomens (OECD, 2001; METI, 2013; REACH). At necropsy, effects in the forestomach mucosa (reddening, haemorrhage, erosion, strong swelling and chondrification), and discolouration of the kidneys and adrenal glands were observed (REACH).

Phenol, nonyl derivatives, sulfides (CAS No. 68515-93-5) has low acute toxicity based on results from an animal test following oral exposure. The LD50 in rats was >2000 mg/kg bw. Observed sub-lethal effects included depression, rough oily fur, slight salivation and or soft feces from one-hour post dose through day 9 at >5000 mg/kg bw (OECD, 2009). The magnesium (CAS No. 91254-25-0) and calcium (CAS No. 68515-95-7) salts of phenol, nonyl derivatives, sulfides are also expected to have low acute toxicity.

There are no data available for phenol, nonyl derivatives, sulfides, barium salts (CAS No. 68515-94-6). Barium salts, with the exception of barium sulphate, salts of 1-azo-2-hydroxynaphthalenyl aryl sulfonic acid, and other barium salts specified elsewhere are classified as hazardous with hazard category 'Acute Toxicity Category 4' and hazard statement 'Harmful if swallowed' (H302) in the Hazardous Chemical Information System (HCIS) (Safe Work Australia). This hazard classification is supported for all barium compounds in this assessment. Soluble zinc salts have moderate acute toxicity following oral exposure (NICNASb).

Dermal

The chemicals are expected to have low acute dermal toxicity.

The LD50 for nonylphenol was reported as 2031 mg/kg bw in New Zealand White rabbits (EC, 2002).

The LD50 for phenol, nonyl derivatives, sulfides (CAS No. 68515-93-5) was reported to be >5000 mg/kg bw in rats (OECD, 2009).

Inhalation

Limited data are available.

In an acute inhalation study (OECD TG 403) guinea pigs, rats and mice were subjected to 6-hour whole body exposure to vapours of phenol, nonyl derivatives, sulfides (CAS No. 68515-93-5). No deaths or signs of toxicity were observed in any species at concentrations of up to 1.27 mg/L (OECD, 2009).

Corrosion / Irritation

Corrosivity

Two of the chemicals (mixed nonylphenol and 4-nonylphenol branched) are classified as hazardous with hazard category 'Skin corrosion – category 1B' and hazard statement 'H314 (Causes severe skin burns and eye damage)' in the Hazardous Chemical Information System (HCIS) (Safe Work Australia). The available data support this classification. In the absence of data, the same classification should be applicable for the other chemicals in this report, apart from the salts. No data are available on the irritation potential of the salts, but as they do not have the acidity of nonylphenol, it is not possible to read across from the acids (nonylphenols) to the conjugate bases (the salts). This classification also does not apply to the phenol, nonyl derivatives, sulfides and its salts based on the negative findings for the parent substance.

Animal data indicate that the chemicals are corrosive to the skin. However, the degree of corrosion was reported to vary according to the source and exact composition of the chemicals. The chemicals are severe eye irritants and saturated vapour could cause mild sensory irritation in the respiratory tract (EC, 2002).

In a skin irritation study conducted according to OECD Test Guideline (TG) 404, undiluted nonylphenol was applied (occlusively) on the skin of three small White Russian rabbits for four hours, with observation for 14 days. Necrosis was observed after one hour. Other effects observed included bloody fissures, dead and swollen skin, and hardening at application site, eschar formation and scars. The mean erythema and oedema scores over 24, 48 and 72 hours were 4.0 and 3.2. The test substance was considered to be corrosive to the skin (REACH).

In several other dermal irritation studies, corrosivity and irritation were reported. The test substances (named 'nonylphenol S' and 'nonylphenol RNH') were applied on rabbits for one or four hours; severe irritation (necrosis and ulceration) was observed within 24 hours. In two good laboratory practice (GLP)-compliant studies in rabbits, the effects described as erythema grade 2 and oedema grade 1–3 were reported over 24, 48 and 72 hours. Grade 4 eschar formation on day eight was reported in one of the studies. In a rat study, application of 0.1 mL of nonylphenol on rat skin for 24 hours caused sensitivity to touch, severe erythema and thickening, wrinkling and hardening of the skin at the application site (EC, 2002).

In an eye irritation study (OECD TG 405), undiluted nonylphenol (0.1 mL) was instilled into the eyes of small White Russian rabbits (n = 3) for 72 hours, with observations up to 21 days following administration. Severe circumcorneal injection and reddened irises were reported. All animals displayed

strongly crusted eyelids (eyelid inflammation) and one animal developed a necrotic spot on the mucosa. The mean scores for all timepoints (24, 48 and 72 hours) were 1.44 for corneal opacity, 1.0 for iritis, 3.0 for conjunctival redness, and 1.67 for chemosis. Effects were irreversible and the chemical was considered corrosive to the eyes (REACH).

In another eye irritation study, a single application of nonylphenol from two different sources (ICI Oil Works and Rohm & Haas) was tested in groups of rabbits (n = three/group), with observation up to seven days. The chemical from ICI caused effects in all animals, described as grade 2 or 3 for conjunctival redness, grades 1–4 for conjunctival chemosis, grades 1 or 2 for corneal opacity and grade 1 for iritis in two rabbits. Less severe effects were observed for the chemical from the other source. The effects were not reversible within the seven-day observation period (EC, 2002).

Phenol, nonyl derivatives, sulfides (CAS No. 68515-93-5) was reported to be a slight eye irritant when instilled into the unwashed rabbit eye. All conjunctival irritation completely subsided by Day 7. There were no iridal or corneal findings in any animals. The mean irritation score was 2.0/110 according to the Draize methodology (OECD, 2009).

Respiratory Irritation

Nonylphenol vapours could cause mild irritation to the respiratory tract at high exposure concentrations.

In a sensory irritation study, groups of female CD-1 mice (n = five/concentration) were exposed to the vapour of nonylphenol at concentrations of 3636 mg/m^3 (400 ppm) or 267 mg/m^3 (30 ppm). The duration of exposure was not reported. A mean respiratory rate depression of ~25 % was determined at the high concentration whereas, no change in the respiratory rate was observed for the lower concentration. The study indicated that high exposure levels to nonylphenol vapour could cause mild sensory irritation in the respiratory tract of mice (EC, 2002).

Sensitisation

Skin Sensitisation

Based on the available data, the chemicals are not considered to be skin sensitisers.

In a skin sensitisation study (OECD TG 406), guinea pigs (n = 20) were initially exposed to nonylphenol (0.1 mL in corn oil) at 0.9 % or 50 % during the intradermal and topical induction phases, respectively. The skin was later challenged topically with the chemical at concentrations of 10, 30 or 45 % in corn oil. No skin sensitising effects were observed (REACH).

The EC report (2002) concluded that, 'The results of several guinea pig maximisation tests suggest that nonylphenol does not have significant skin sensitising potential'. In a guinea pig maximisation test with limited documentation, nonylphenol from two sources (ICI Oil Works and Rohm & Haas) was tested in ~20 animals. The only skin reactions (details not available) observed were in 2 out of 20 animals using nonylphenol from ICI Oil Works. No skin reactions were observed in guinea pigs exposed to the chemical from the other source (EC, 2002).

In a skin sensitisation study (OECD TG 406), guinea pigs (n = 30) were initially exposed to phenol, nonyl derivatives, sulfides (CAS No. 68515-93-5) (0.5 mL at 100 %) and then at 10 % during the challenge phase. The skin was later re-challenged topically with 0.5 mL of 10 % of the chemical. The treated animals exhibited slight dermal irritation following challenge and rechallenge (OECD, 2009).

Observation in humans

Negative findings were obtained in a human repeated-insult patch test with phenol, nonyl derivatives, sulfides (CAS No. 68515-93-5), where all 182 human subjects did not show any skin sensitisation or irritation on repeated topical applications (OECD, 2009).

Repeated Dose Toxicity

Oral

Based on the available data, the chemicals are not considered to cause serious damage to health (excluding reproductive and developmental effects) following repeated oral exposure in rats. Kidney effects in male rats were observed at 15 mg/kg bw/day in a multigeneration study, but were not present at this dose level in a 90-day dietary rat study.

In a 28-day study, no treatment-related changes were observed in Sprague Dawley (SD) rats administered nonylphenol in the diet at doses ≤100 mg/kg bw/day. Effects were only observed at the highest dose (400 mg/kg bw/day) and included reduced food consumption and body weight gain. Male rats displayed increased urea and cholesterol levels, reduced glucose levels, and increased mean relative kidney, liver and testes weights. At necropsy, hyaline droplet accumulation in the renal proximal tubules (an observation associated with the rat-specific protein a-2µ-globulin and considered not toxicologically relevant to humans) and minor vacuolation in the periportal hepatocytes in the liver were observed in males. No treatment-related changes in organs were observed in females at any dose level. The no observed adverse effect (NOAEL) was established as 100 mg/kg bw/day in this study (EC, 2002).

In a repeated dose oral toxicity study, SD rats (n = 15/sex/dose) were administered nonylphenol in the diet at doses of 0, 15, 50 or 140 mg/kg bw/day for 90 days, with a recovery period of 28 days at the end of exposure. At the highest dose, observations included reduced body weight gain and food consumption in all animals; slight or moderate hepatic cell necrosis in 3 out of 15 female rats (with two of them displaying elevated levels of serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT)); and decreased incidence of renal tubular hyaline droplets in male rats (as opposed to the 28-day study). Oestrous cycle patterns and sperm measurements were not affected at any dose. An NOAEL of 50 mg/kg bw/day was determined (EC, 2002; REACH).

In a short-term study (OECD TG 407), SD rats (n = 10/sex/dose) were administered (gavage) nonylphenol at doses of 0, 10, 50 or 250 mg/kg bw/day for 28–32 days. Three female rats died at the highest dose. Some other effects observed at this dose include salivation, urinary incontinence, a slight decrease in body weight gain and food consumption, and hepatic and renal toxicity (increased relative liver and kidney weights, centrilobular liver cell hypertrophy and a variety of renal tubular lesions, and alteration of serum biochemical parameters). Furthermore, decreased weights of seminal vesicles and ventral prostrate in males; and increased adrenal weights, irregular oestrous cycle and vaginal mucosa hyperplasia in females were observed. At a dose of 50 mg/kg bw/day, small but significant changes in glucose and inorganic phosphate levels and increased serum luteinising hormone (LH) in females, and increased thyroid weights in the males were observed. An NOAEL of 10 mg/kg bw/day was established based on the effects observed at 50 mg/kg bw/day (REACH).

In a multigeneration reproductive and developmental study conducted for up to 20 weeks (with dietary administration equivalent to doses of 0, 15, 50 and 160 mg/kg bw/day) (see **Reproductive and developmental toxicity**), rats administered nonylphenol at 160 mg/kg bw/day (F0: 15 weeks, F1 and F2: birth to 20 weeks, F3: birth to eight weeks) showed reduced body weight gain in adults across all generations (10 % compared with controls); and increased relative kidney weights in F0, F1, F2 adult males and F1 adult females. Effects observed at the 50 mg/kg bw/day dose were reduced body weight gain in the F1 and F3 females, and F2 males, and increased relative kidney weights in the F0, F1, F2 adult males. Necropsy results included an increase in the incidence of renal tubular degeneration and/or dilatation in adult males from all generations at all treated groups, and in adult females (F1, F2 at 160 mg/kg bw/day, and F3 ≥50 mg/kg bw/day). The lowest observed adverse effect level (LOAEL) for repeated dose toxicity was determined as 15 mg/kg bw/day based on histopathological changes in the kidneys (tubular degeneration or dilatation) (EC, 2002).

The European Commission (EC) report (2002) concluded that it was 'difficult to decide for certain whether or not this increased incidence of renal tubular degeneration and/or dilatation is related to treatment because these changes were not seen to the same extent in the 90-day study, which was conducted using the same strain of rats, and because a dose-dependent trend was not apparent in all generations or sexes. The lack of concordance between the studies cannot be explained on the basis of a slightly longer exposure period in the multigeneration study because kidney effects were seen in the F3 generation which was exposed for only 8 weeks, nor on the basis of *in utero* and neonatal exposure because the effect also occurred in the F0 generation. Giving special emphasis to the fact that the increased incidence occurred consistently across all four generations in the multigeneration study, it is considered that this cannot be dismissed as background variation' (EC, 2002). An independent review of the histopathology confirmed renal lesions in all exposed animals, but without a dose response. 'The predominant renal lesions were described as tubular mineralisation at the OSOM/ISOM junction, cystic tubules surrounded by fibrosis, or granular cast formation at the OSOM/ISOM junction'. (EC, 2002).

The available animal data provide strong evidence that the most sensitive adverse effect of barium is renal toxicity. There are some reports of renal effects in case reports of individuals ingesting high doses of barium. Nephropathy has been observed in rats and mice following long-term oral exposure to barium. The no observed effect level in mice exposed to barium chloride for 90 days is 205 mg barium/kg bw/day (ATSDR, 2007).

Dermal

No data are available.

Inhalation

No data are available.

Genotoxicity

Based on the available data, the chemicals are not considered genotoxic.

Nonylphenol gave negative results in several in vitro genotoxicity assays (EC, 2002; REACH):

- negative results in bacterial reverse mutation assays with strains of *Salmonella typhimurium* at concentrations up to 5000 µg/plate, with or without metabolic activation;
- no induction of chromosomal aberrations in Chinese hamster ovary (CHO) cells; and
- no mutations in a mammalian cell gene mutation test in Chinese hamster lung (CHL) fibroblasts, with or without metabolic activation.

Two in vivo micronucleus assays had negative results for nonylphenol (EC, 2002; REACH):

- a bone marrow micronucleus test in rats injected intraperitoneally (i.p.) with the chemical at single doses of 50, 150 or 300 mg/kg bw. The administered doses were considered sufficient to produce toxicity in rats, based on neurological symptoms at ≥150 mg/kg bw;
- a bone marrow micronucleus test in rats orally administered the chemical at 500 mg/kg bw (maximum tolerated dose); and

- immature male SD rats treated (i.p.) once with the chemical at 60 mg/kg bw, showed an increased mitotic index and abnormal mitoses.

Carcinogenicity

No animal carcinogenicity studies are available for the chemicals. Based on the available data for genotoxicity (see **Genotoxicity**), carcinogenicity via a genotoxic mechanism is not expected. Based on the available information, no conclusion can be derived about the carcinogenicity of the chemicals.

Limited data are available giving inconclusive results for the chemicals (EC, 2002; CalEPA, 2009; HSDB):

- Fischer 344 (F344) rats administered nonylphenol in the diet for 28 weeks at concentrations of 25 or 250 ppm had a higher incidence of adenomas and carcinomas than the controls; and
- in a two-stage initiation–promotion transformation assay in BALB/3T3 cells, nonylphenol acted as a pure promoter in cell transformation, indicating that enhancement of carcinogenicity in vivo is possible.

Reproductive and Developmental Toxicity

Two of the chemicals (mixed nonylphenol and 4-nonylphenol branched) are classified as hazardous with hazard category 'Reproductive toxicity – category 2' and hazard statement 'H361fd (Suspected of damaging fertility. Suspected of damaging the unborn child)' in the Hazardous Chemical Information System (HCIS) (Safe Work Australia).

The available data support the fertility classification based on reproductive effects in rats. A neurodevelopmental and behavioural study in rats showed effects on physical maturation and a feminising effect in male pups of dams that received nonylphenol at 50 mg/kg bw/day, supporting the developmental classification. The same classifications should be applicable for the other chemicals in this report.

Many reproductive and developmental toxicity studies in rats and mice have been conducted. Some studies in rats contain reports of reproductive effects in males (decreased epididymal sperm density and testicular spermatid head counts) and females (increased oestrus cycle length and decreased ovarian weights) when exposed to nonylphenol. The NOAEL for reproductive toxicity was established as 15 mg/kg bw/day in female rats in one multi-generation study.

In a 3.5 generation reproductive and developmental toxicity study (OECD TG 416), SD rats (n = 30/sex/dose) were administered nonylphenol in the diet at dose levels of 0, 200, 650 or 2000 ppm (0, 15, 50, 160 mg/kg bw/day). The F0 generation was exposed at about seven weeks old and continued for 15 weeks prior to termination of exposure (when the F3 generation was about eight weeks old). There were no adverse effects on fertility or mating performance. However, reproductive effects in the offspring were observed. In females, increased oestrous cycle length (F1 and F2 females at 2000 ppm), accelerated timing of vaginal opening (F1–F3: by 1.5–7 days at 650 ppm and by 3–6 days at 2000 ppm), and decreased absolute ovarian weights (F2 at 650 ppm, and F1–F3 at 2000 ppm) were observed. Testicular toxicity including reduced epididymal sperm density at the ≥650 ppm dose and reduced testicular spermatid count at the 2000 ppm dose were observed in F2 males. However, the authors mentioned that there were uncertainties with the epididymal sperm density measurements. The study indicated that the observed effects were possibly related to the oestrogenicity of the chemical. The NOAEL for reproductive effects was established as 200 ppm (15 mg/kg bw/day) in females (CalEPA, 2009; REACH). Another i.p. study in neonatal male rats indicated that the critical exposure period for reproductive tract effects was likely to be for the neonatal period (CalEPA, 2009).

Although the study authors stated the testicular effects were possibly due to the oestrogenicity of the chemical, similar effects have been reported for dibutyl phthalate (DBP) in reproductive studies (testicular toxicity including altered testicular and epididymal weights, seminiferous tubule lesions and decreased testosterone levels), due to anti-androgenic properties (NICNAS, 2013).

In another multigeneration reproductive study (US EPA guideline), SD rats (n = 25/sex/dose) were exposed to nonylphenol in two different diets at dose levels of 0, 20, 200, 650 or 2000 ppm (0, 1.5, 15, 50 or 150 mg/kg bw/day) with Purina 5002 diet and 0 or 650 ppm with NIH-07 diet. No treatment-related effects on the reproductive parameters and no effects on the absolute epididymal weights were observed. Reduced ovarian weights were observed at the 2000 ppm (F0), 200 and 650 ppm doses (F1 on Purina 5002 diet), and at the 650 ppm dose (F0 on NIH-07 diet), but this did not affect reproduction. Decreased pup body weight at weaning was observed at the 2000 ppm dose, but not during lactation. An NOAEL of >150 mg/kg bw/day was established for reproductive toxicity (REACH).

Following oral exposure, reproductive studies in male rats have shown atrophy of seminiferous tubule and decreased tubule and lumen diameter at a ≥100 mg/kg bw/day dose, decreased testicular and epididymal weights and significantly decreased testosterone levels at a ≥250 mg/kg bw/day dose, and decreased sperm numbers at a 400 mg/kg bw/day dose. Increased follicle stimulating hormone (FSH) and LH levels in serum were observed at a 125 mg/kg bw/day dose. One brief report on an oral gavage study in SD rats showed testicular toxicity at an exposure level that also caused mortality (100 mg/kg bw/day) (EC, 2002; CalEPA, 2009; METI, 2013).

Reproductive effects in female rats included uterotrophic effects and accelerated vaginal opening (early puberty) at a ≥50 mg/kg bw/day dose. A more significant increase in uterine weights was observed following oral administration, compared with subcutaneous injection (EU, 2002; CalEPA, 2009; METI, 2013). The changes in reproductive organ weights were reported to be associated with the oestrogenic activity of the chemical. Reproductive effects in female rats (20–21 days old) and neonatal male rats were not observed when nonylphenol was administered (i.p.) concomitantly with ICI182780, an oestrogen receptor antagonist (CalEPA, 2009; METI, 2013).

In a developmental toxicity study (OECD TG 414), Wistar rats were orally administered (gavage) nonylphenol in corn oil at dose levels of 0, 75, 150 or 300 mg/kg bw/day on gestation days (GD) 6–15. At the highest dose, maternal toxicity (2/21 deaths, reduced body weight gain and food consumption)

was observed. Effects in the kidneys and spleen were observed at the 150 mg/kg bw/day dose. Post-implantation loss, litter size, foetal weights and abnormalities were not affected. The maternal and developmental NOAELs were established at the 75 mg/kg bw/day and ≥300 mg/kg bw/day doses, respectively (EC, 2002; REACH).

Neurodevelopmental effects were also observed in developmental studies.

In a neurodevelopmental and behavioural study, SD dams were administered nonylphenol (gavage) at doses of 0, 50 or 200 mg/kg bw/day from GD 5 to PND 21. Dams receiving the highest dose had longer gestational periods. 'Physical maturation (incisives and eyes)' of male pups was affected at the 50 mg/kg bw/day dose. The female offspring were observed to be more active than males in open-field tests at the 50 mg/kg bw/day dose. A postulated feminising effect observed as hyperactivity in males was reported as possibly due to brain development impairment from an anti-androgenic action through monoamine systems. In a water maze test (evaluating learning, spatial orientation and memory function), females dosed at 200 mg/kg bw/day showed effects within 30 seconds, which were postulated to be 'masculinisation due to an oestrogenic action' of nonylphenol. This effect was not observed in young adults (Couderc et al., 2014).

Nonylphenols are listed in the EC Endocrine Disruptors Priority List under Category I classification (i.e. Evidence of endocrine disrupting activity in at least one species using intact animals; EC 2015); and the US EPA's Universe of Chemicals list for potential endocrine disruptor screening and testing (US EPA, 2012).

Other Health Effects

Neurotoxicity

There have been reports from animal studies about neurological symptoms following acute oral exposure, including ataxia, tremor and lethargy (see **Acute toxicity: Oral**).

Some in vitro studies with cultured cells and tissues have indicated that nonylphenol could potentially affect brain development and cause neurodegeneration. Nonylphenol inhibited the growth of neural stem cells in 24 hours (CalEPA, 2009).

Effects on the dopamine systems in animals were observed. Nonylphenol at a dose of 10 µM increased hydroxyl radical formation in the rat striatum, and enhanced hydroxyl radical formation induced by the 1-methyl-4-phenylpyridium ion (MPP+, known to cause neurodegeneration leading to Parkinsonism) (CalEPA, 2009).

Nonylphenol was tested on neonatal rats (8.7 mM via intracisternal injection) for spontaneous motor activity and was observed to cause significant hyperactivity in rats aged 4–5 weeks. The same authors observed an increase in spontaneous motor activities in four-week-old rats following intracisternal injection of nonylphenol at the same dose on postnatal day (PND) five. In other studies in rats, nonylphenol was observed to cause a deficit in dopamine neurons and increased activity in the nocturnal phase by 1.3–1.6 fold at a dose of 8.7 mM (CalEPA, 2009).

Collectively, these studies were reported to indicate neonatal treatment-generated animal models of attention-deficit hyperactivity disorder (ADHD). Following oral administration of nonylphenol in F344 rats at doses of 0.1 or 10 mg/kg bw/day from GD three to PND 20, the chemical caused irreversible loss of the capacity to react to fear-provoking stimuli, and altered behavioural characteristics through monoaminergic neural pathways (involving monoamines in neural transmission) in the offspring at both doses (CalEPA, 2009).

Several animal studies have shown that nonylphenol could potentially affect the thyroid system, including biosynthesis, transport and receptor binding. An oral reproductive study in rats showed changes in thyroid hormone levels at 50 mg/kg bw/day (see **Repeat dose toxicity: Oral**). The disruption of thyroid functions could adversely impact neurodevelopment; however, more evidence is required to establish this effect (CalEPA, 2009; METI, 2013; Couderc et al., 2014).

Other effects

The 2002 EC report stated that 'Nonylphenol has been reported to induce cell proliferation in the mammary gland of the Nobel rat following subcutaneous exposure at levels down to 0.05 mg/kg/day, but this finding could not be reproduced in a duplicate study; furthermore, there are doubts about the relevance of this finding to humans and regarding the validity of the original study' (EC, 2002).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include:

- local effects (corrosivity);
- systemic acute effects from oral exposure; and
- systemic long-term effects (reproductive and developmental toxicity).

Public Risk Characterisation

Although the public could come into contact with articles and/or coated surfaces containing the unreacted chemical, it is expected that the residual levels would be very low. While there is the possibility that incidental direct exposure to these chemicals may result from use of some domestic products, such as epoxies and other adhesive products, this would be infrequent. Members of the public may also be exposed to small amounts of sulfides when adding lubricant oil to automotive crankcases, or when changing their own automotive engine oil. Due to the low volatility and low water solubility of the sulfides, and the short period of exposure during finished lubricant oil changes, the risk of adverse health effects to the public is expected to be minimal. Provided that normal precautions are taken to avoid prolonged skin contact, the risk to the public is not considered to be unreasonable for the chemicals in this group.

Given the findings in animal studies and considering the breakdown of nonylphenol ethoxylates to nonylphenols in the environment, there may be potential for human exposure via the environment (NICNASc). However, the risk to humans is considered acceptable if the concentration levels are maintained in accordance with the limitations for nonylphenol set out in the Australian Guidelines for Water Recycling on the 'planned use of recycled water (treated sewage and stormwater) to augment drinking water supplies'. A maximum concentration of 2.9 μg/L has been detected for 4-nonylphenol in secondary treated sewage and a guideline value of 500 μg/L is derived for drinking water augmentation (NRMMC-EPHC-NHMRC, 2008). Therefore, the chemicals are not considered to pose an unreasonable risk to public health.

Occupational Risk Characterisation

During product formulation, 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. The level and route of exposure will vary depending on the method of application and work practices employed.

Given the critical health effects, the chemicals could pose an unreasonable risk to workers unless adequate control measures to minimise dermal and ocular exposure are implemented. The 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.

Based on the available data, the hazard classification in the HCIS (Safe Work Australia) is considered appropriate for two of the chemicals (CAS Nos. 25154-52-3 and 84852-15-3). The same hazard classification should be applicable for the other chemicals in this report (see **Recommendation** section).

NICNAS Recommendation

Assessment of these chemicals 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.

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.

Phenol, nonyl derivatives, sulfides (CAS No. 68515-93-5) and its magnesium (CAS No. 91254-25-0) and calcium (CAS No. 68515-95-7) salts are expected to have low acute toxicity based on available data. Therefore, the acute toxicity classification- 'Harmful if swallowed- Cat 4' should not be applied to these chemicals.

The corrosivity classification- 'Causes severe skin burns and eye damage - Cat. 1B (H314') is the exisiting classification for CAS Nos. 25154-52-3 and 84852-15-3. This classification should not be applied to the salts in this group as no data are available on the irritation potential of these chemicals. If data specific to dinonylphenols becomes available, this classification may be revised and amended. This classfication also does not apply to the phenol, nonyl derivatives, sulfides and its salts (CAS Nos. 68515-93-5, 68515-94-6, 68515-95-7, 91254-25-0).

From 1 January 2017, under the model Work Health and Safety Regulations, chemicals are no longer to be classified under the Approved Criteria for Classifying Hazardous Substances system.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Not Applicable	Harmful if swallowed - Cat. 4 (H302)
Irritation / Corrosivity	Not Applicable	Causes severe skin burns and eye damage - Cat. 1B (H314)

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Reproductive and Developmental Toxicity	Not Applicable	Suspected of damaging fertility or the unborn child - Cat. 2 (H361fd)

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

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

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

Advice for industry

Control measures

Control measures to minimise the risk from oral, dermal 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 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.

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Last Update 08 March 2019

Chemical Identities

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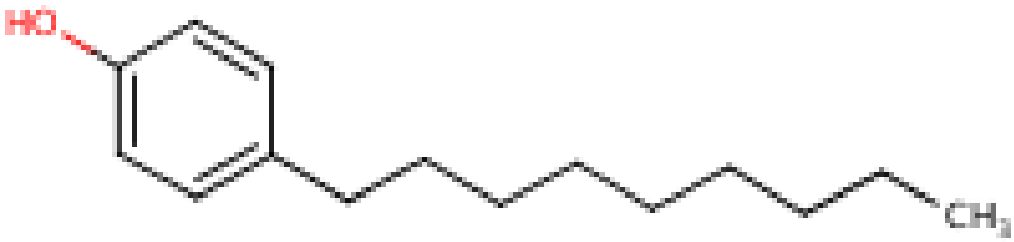
CAS Number	104-40-5
Structural Formula	
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Molecular Weight	220.354

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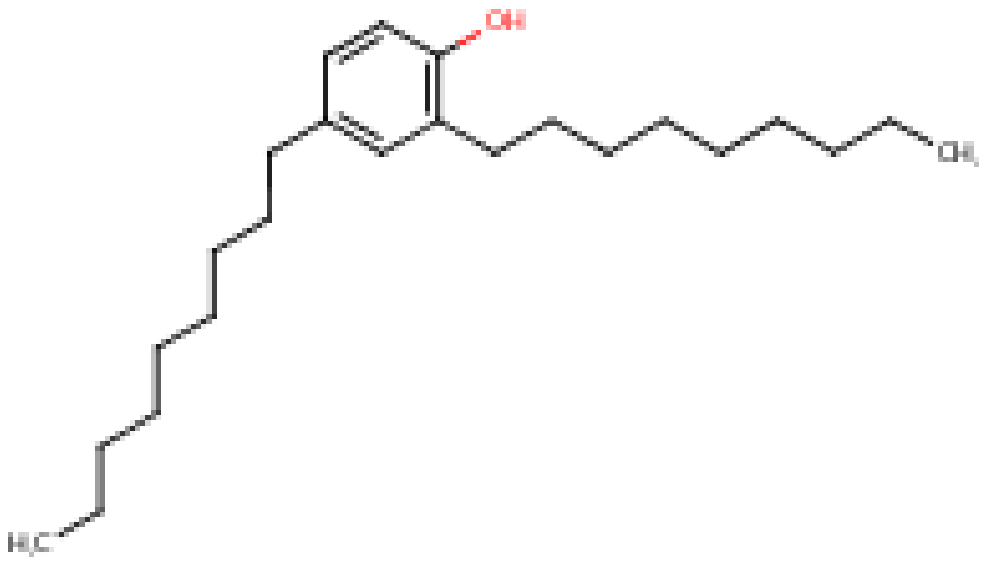
Structural Formula	
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	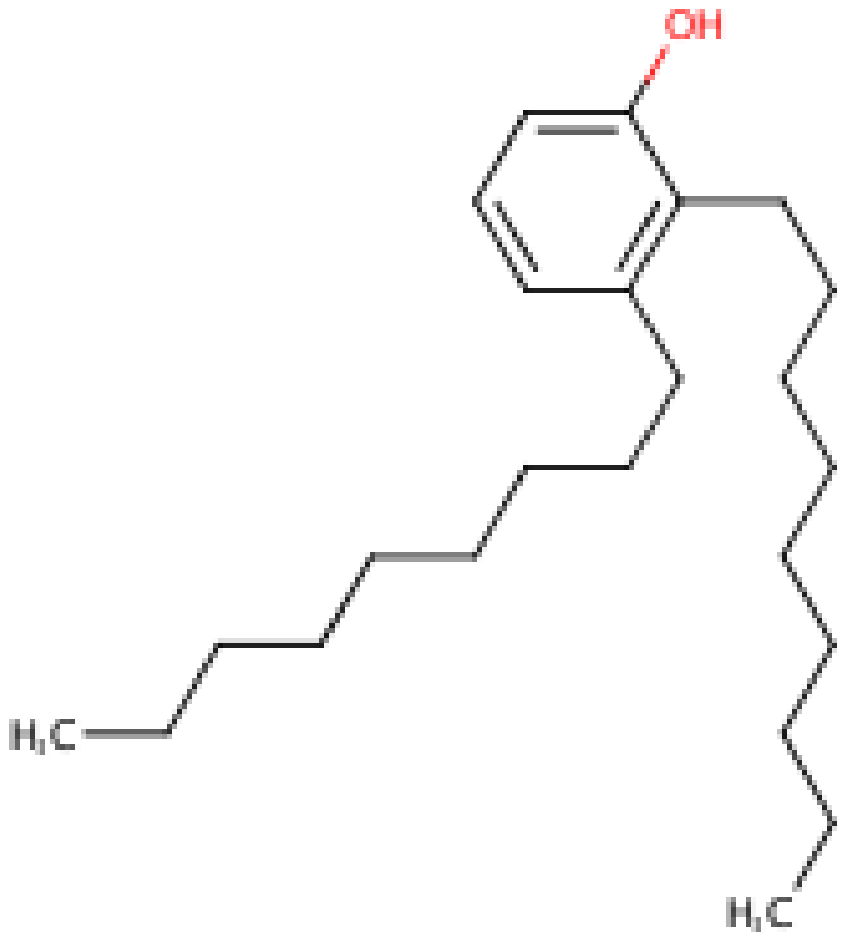
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	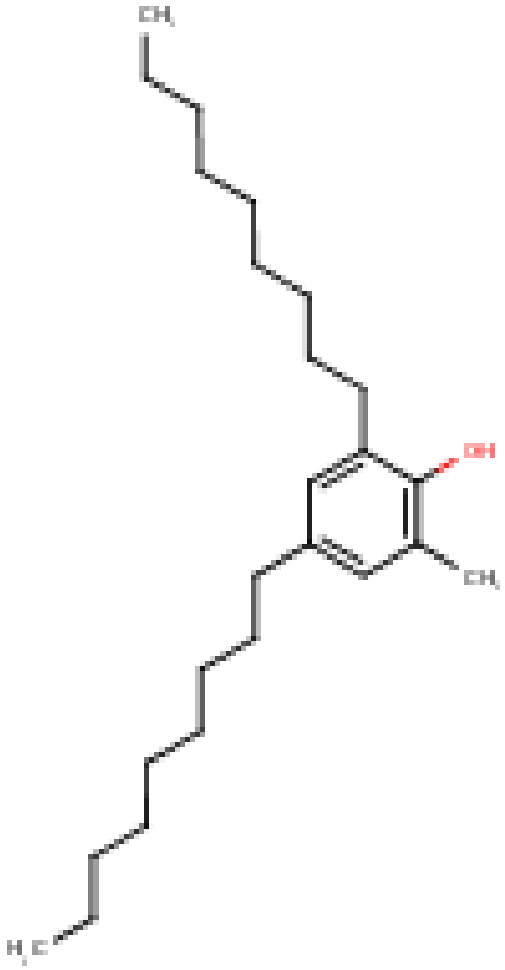
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	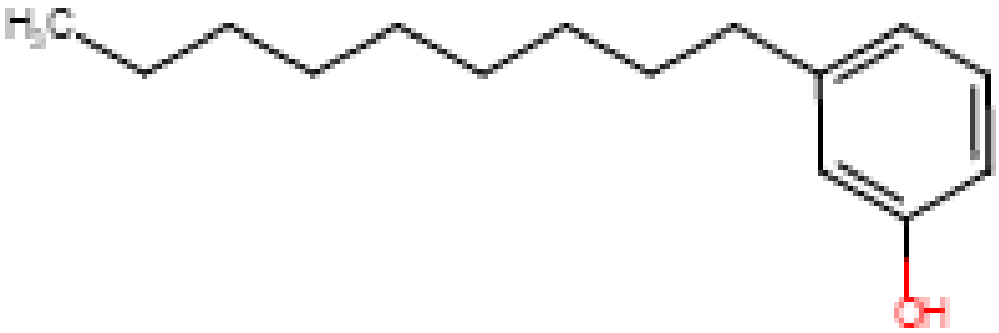
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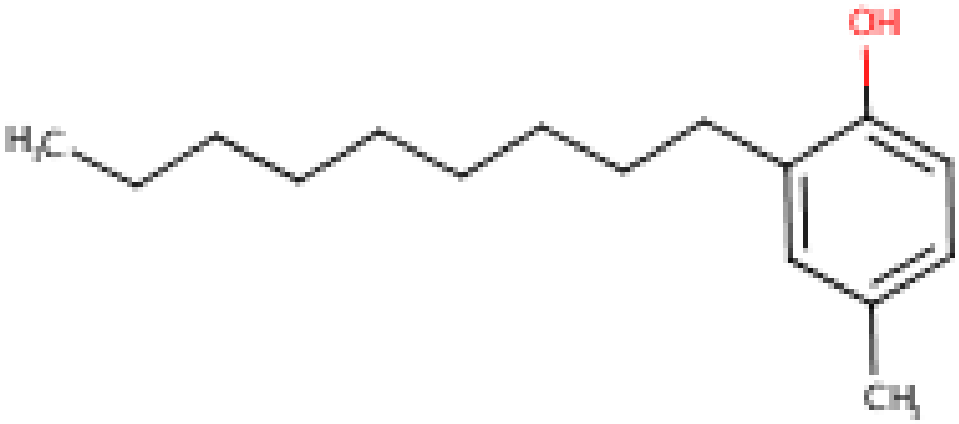

Molecular Formula	C16H26O
Molecular Weight	234.38

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	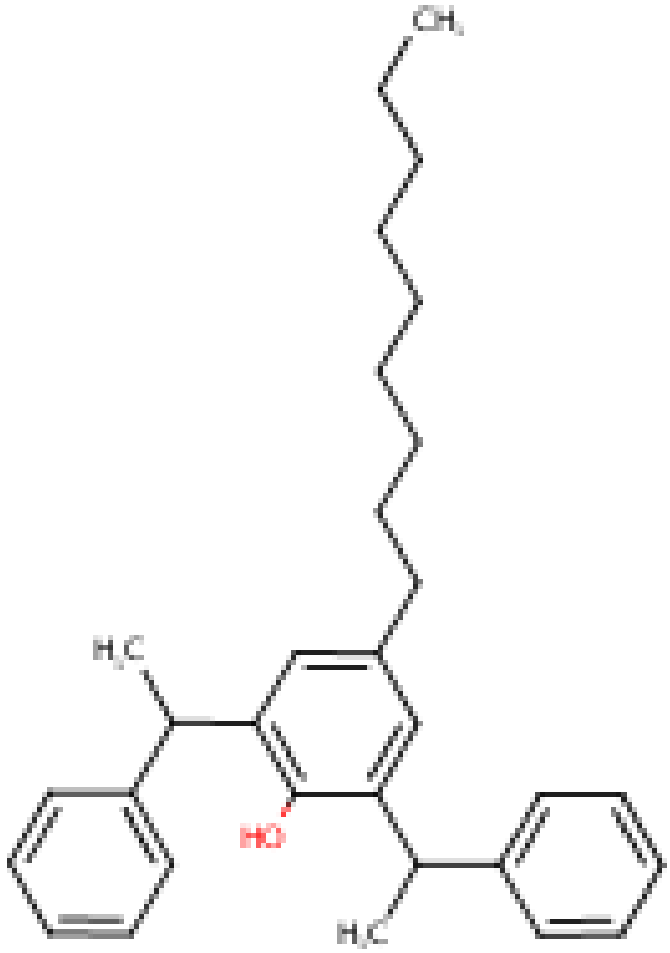
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CAS Number	25154-52-3
Structural Formula	

	
Molecular Formula	C15H24O
Molecular Weight	221.36

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CAS Number	28983-26-8
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	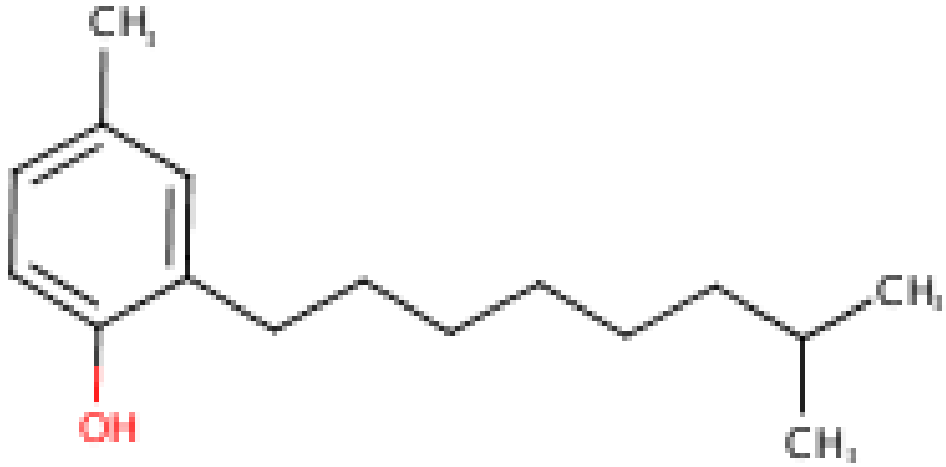
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Molecular Weight	234.38

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	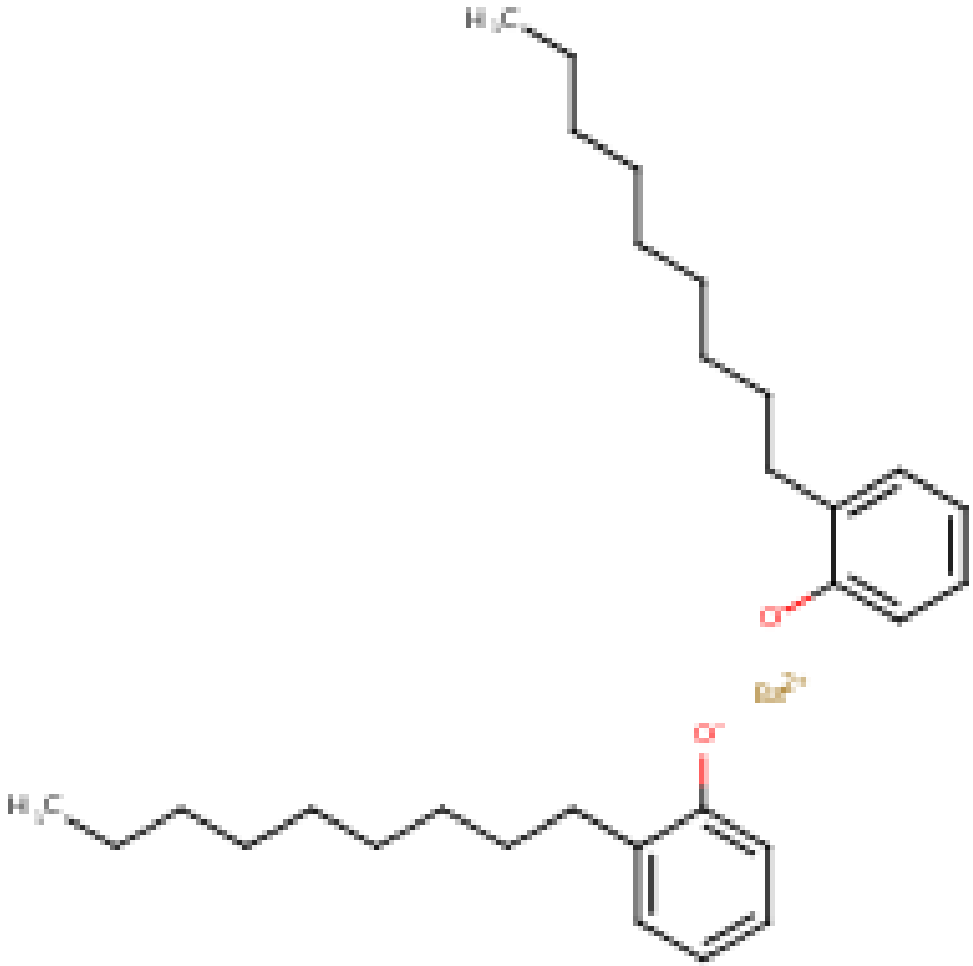
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	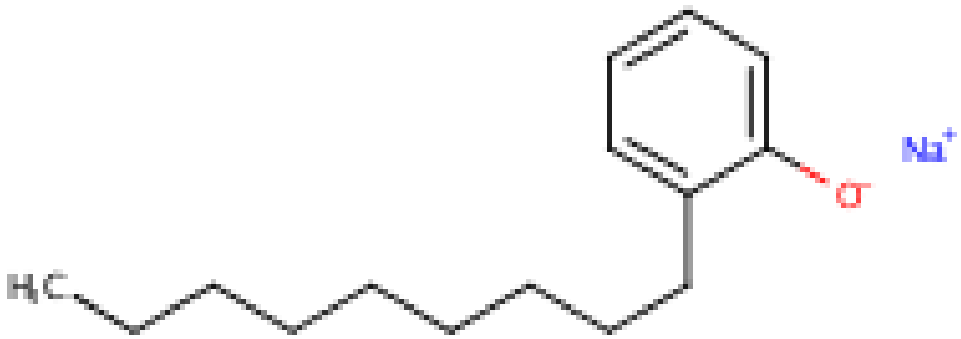
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	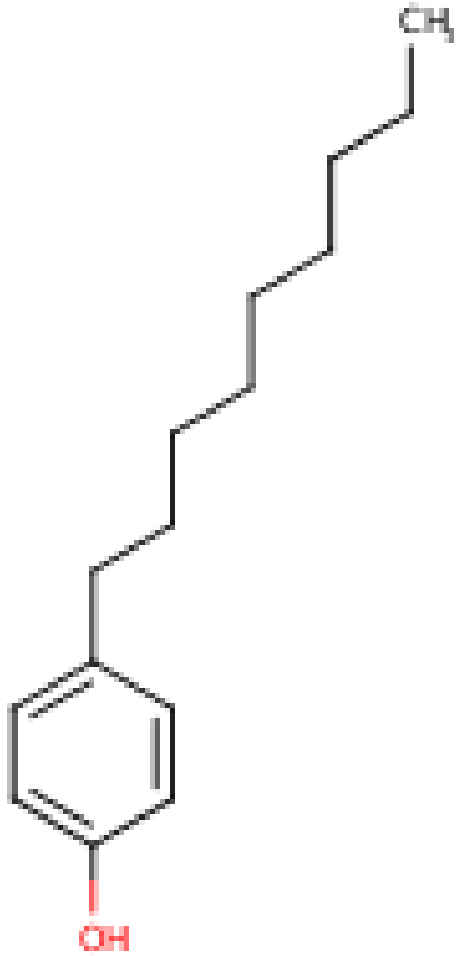
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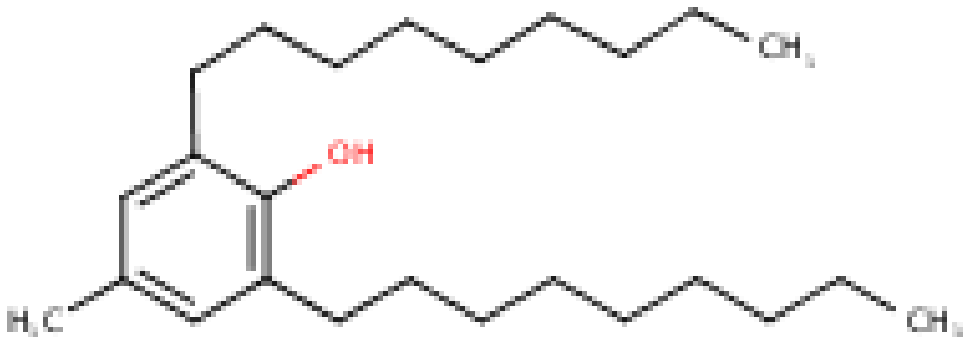
Chemical Name in the Inventory and Synonyms	**Phenol, 4-methyl-2,6-dinonyl-** 4-Methyl-2,6-dinonylphenol dinonyl-p-cresol
CAS Number	63451-44-5
Structural Formula	

	
Molecular Formula	C25H44O
Molecular Weight	360.62

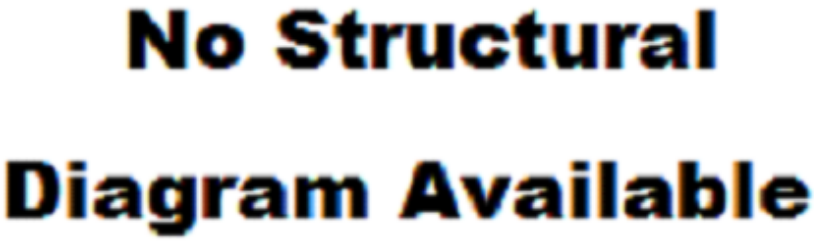
Chemical Name in the Inventory and Synonyms	**Barium, carbonate 4-nonylphenol complexes** Overbased barium nonylphenate
CAS Number	68442-67-1
Structural Formula	**No Structural Diagram Available**

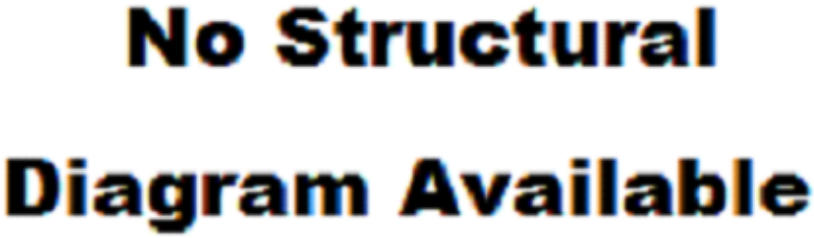
Molecular Formula	Unspecified
Molecular Weight	Unspecified

Chemical Name in the Inventory and Synonyms	**Barium, carbonate nonylphenol complexes** Barium nonylphenolate, carbon dioxide, overbased
CAS Number	68515-89-9
Structural Formula	No Structural Diagram Available
Molecular Formula	Unspecified
Molecular Weight	Unspecified

Chemical Name in the Inventory and Synonyms	**Phenol, nonyl derivatives, barium salts** (C9) Alkylated phenol, barium salt C9-alkylated phenol barium salt
CAS Number	68515-91-3
Structural Formula	No Structural Diagram Available

Molecular Formula	Unspecified
Molecular Weight	289.02

Chemical Name in the Inventory and Synonyms	**Phenol, nonyl derivs., sulfides** C9-alkylated phenol sulfides
CAS Number	68515-93-5
Structural Formula	
Molecular Formula	
Molecular Weight	Unspecified

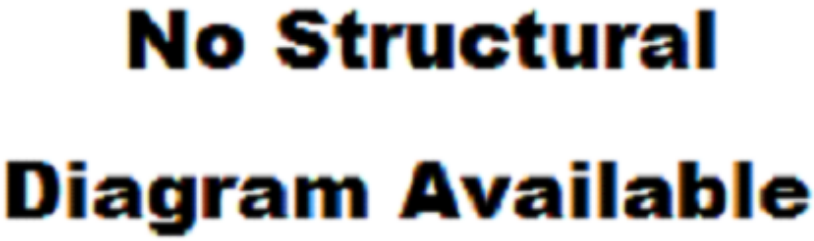
Chemical Name in the Inventory and Synonyms	**Phenol, nonyl derivs., sulfides, barium salts** C9-alkylated phenol sulfides, barium salts
CAS Number	68515-94-6
Structural Formula	

Molecular Formula	
Molecular Weight	Unspecified

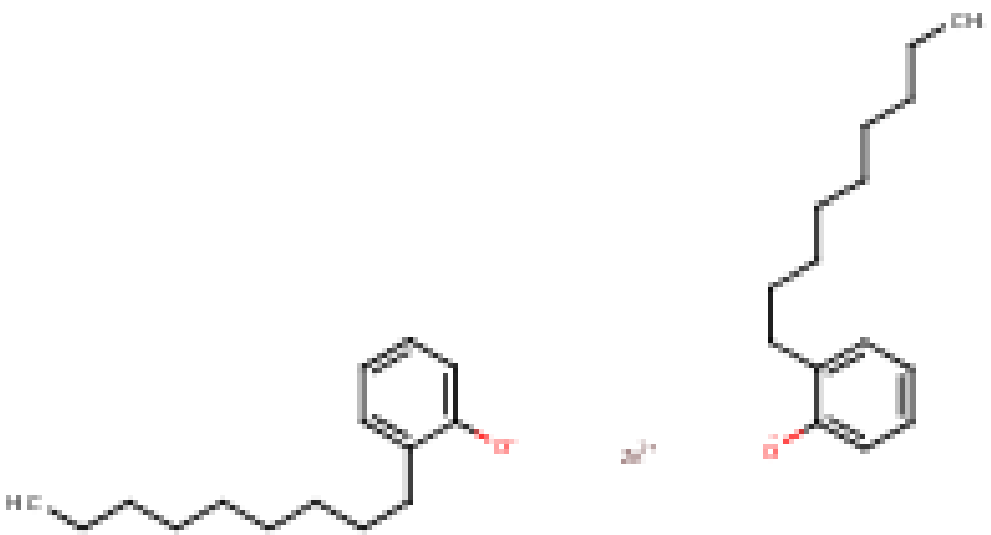
Chemical Name in the Inventory and Synonyms	**Phenol, nonyl derivs., sulfides, calcium salts** C9-Alkylated phenol sulfides, calcium salts
CAS Number	68515-95-7
Structural Formula	No Structural Diagram Available
Molecular Formula	
Molecular Weight	Unspecified

Chemical Name in the Inventory and Synonyms	**Phenol, tri-C6-9-alkyl derivatives** tri-C6-9-alkylphenol
CAS Number	68515-97-9
Structural Formula	No Structural Diagram Available

Molecular Formula	Unspecified
Molecular Weight	Unspecified

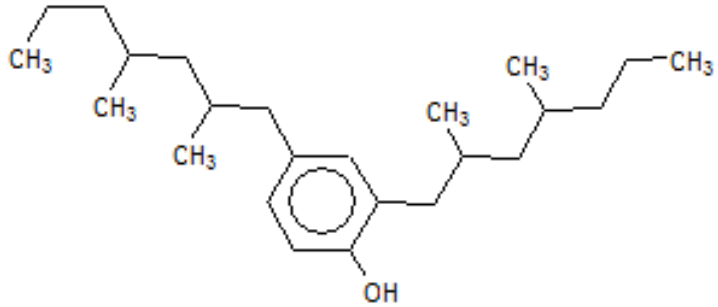
Chemical Name in the Inventory and Synonyms	**Calcium, nonylphenol sulfides phosphosulfurized polybutene complexes**
CAS Number	68910-15-6
Structural Formula	
Molecular Formula	Unspecified
Molecular Weight	Unspecified

Chemical Name in the Inventory and Synonyms	**Phenol, nonyl-, zinc salt** zinc bis(nonylphenolate)
CAS Number	77194-15-1
Structural Formula	

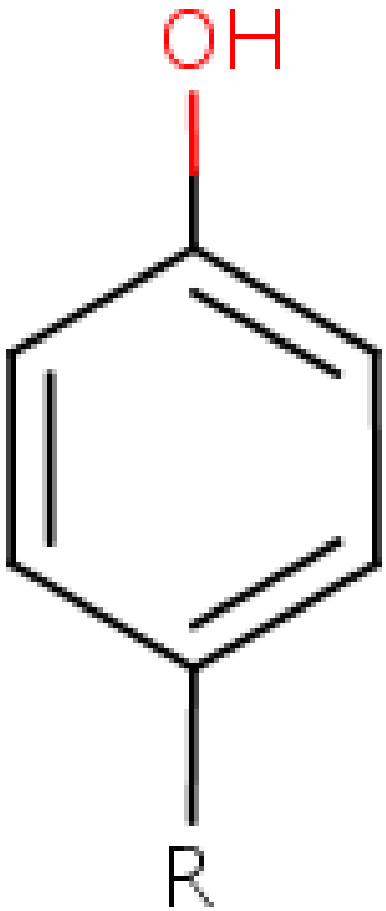


Molecular Formula	C15H24O.1/2Zn
Molecular Weight	252.04

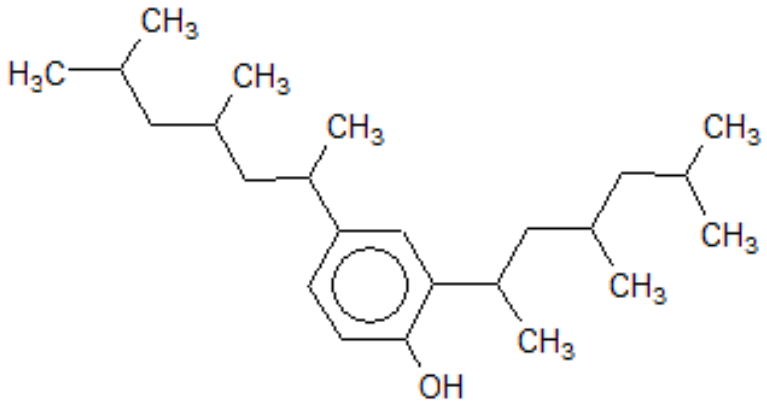
Chemical Name in the Inventory and Synonyms	**Phenol, 2,4-dinonyl-, branched**
CAS Number	84852-14-2
Structural Formula	

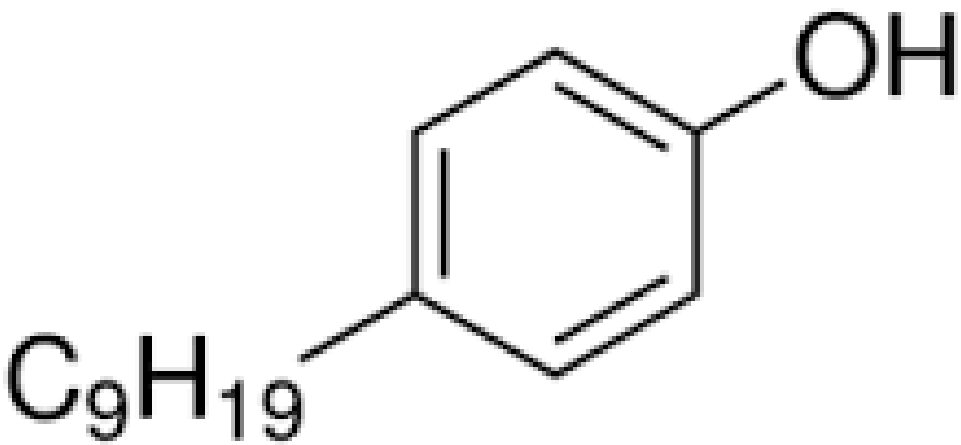
	
Molecular Formula	Unspecified
Molecular Weight	346.59

Chemical Name in the Inventory and Synonyms	**Phenol, 4-nonyl-, branched** branched para-nonylphenol C9 branched alkyl phenol
CAS Number	84852-15-3
Structural Formula	

	
Molecular Formula	Unspecified
Molecular Weight	220.35

Chemical Name in the Inventory and Synonyms	**Phenol, dinonyl -, branched**
CAS Number	84962-08-3
Structural Formula	

	
Molecular Formula	C24H42O
Molecular Weight	346.59

Chemical Name in the Inventory and Synonyms	**Phenol, nonyl-, branched** nonylphenol derivatives
CAS Number	90481-04-2
Structural Formula	
Molecular Formula	Unspecified
Molecular Weight	220.35

Chemical Name in the Inventory and Synonyms	**Phenol, nonyl derivatives, sulfides, magnesium salts** (C9) alkylated phenol sulfides, magnesium salt
CAS Number	91254-25-0
Structural Formula	**No Structural Diagram Available**
Molecular Formula	Unspecified
Molecular Weight	Unspecified

Chemical Name in the Inventory and Synonyms	**Phenol, nonyl-, barium salt, basic**
CAS Number	93028-52-5
Structural Formula	

	
Molecular Formula	Unspecified
Molecular Weight	289.02

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