2-Ethylhexyl diphenyl phosphate: Human health tier II assessment

25 November 2016

Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
Phosphoric acid, octyl diphenyl ester	115-88-8
Phosphoric acid, 2-ethylhexyl diphenyl ester	1241-94-7

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 chemical being assessed in this report is referred to as 2-ethylhexyl diphenyl phosphate (EHDP) with the CAS RN 1241-94-7. It is reported that this chemical may have also been referred to as diphenyl octyl phosphate, with the CAS RN 115-88-8 (UK Environment Agency, 2009). The term "octyl" is often used to refer to the 2-ethylhexyl group. In the case of n-octyl groups, the prefix is normally used.

While both CAS RNs are listed on the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) Australian Inventory of Chemical Substances (AICS), there is insufficient information to indicate that these represent two uniquely different chemicals. For this reason, this assessment report applies to both CAS RN 1241-94-7 and CAS RN 115-88-8. Due to the known developmental toxicity of the hydrolysis product, 2-ethylhexanol (NICNAS), CAS RN 1241-94-7 is expected to represent the worst case for CAS RN 115-88-8.

Import, Manufacture and Use

Australian

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

The chemical has reported commercial use as a substance for softening materials to improve the feel, to facilitate finishing processes or to impart flexibility or workability. This use includes plasticisers and devulcanising agents.

The total volume introduced into Australia, reported under previous mandatory and/or voluntary calls for information, was <100 tonnes per annum.

International

The following international uses have been identified through the European Union (EU) Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) dossiers; Galleria Chemica; the Substances and Preparations in Nordic countries (SPIN) database; the US Environmental Protection Agency's Aggregated Computer Toxicology Resource (ACToR); the US National Library of Medicine's Hazardous Substances Data Bank (HSDB); and an international assessment (UK Environment Agency 2009).

The chemical has reported domestic uses, including in:

- paints, lacquers and varnishes; and
- adhesives and binding agents.

The chemical has reported commercial uses, including:

- in lubricants and additives;
- as a plasticiser;
- as a fire-retardant; and
- in dyes and textile treatment products.

Restrictions

Australian

No known restrictions have been identified for EHDP.

International

No known restrictions have been identified for EHDP.

Existing Worker Health and Safety Controls

Hazard Classification

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

Exposure Standards

Australian

No specific exposure standards are available.

International

No specific exposure standards are available.

Health Hazard Information

Toxicokinetics

In a toxicokinetics study in Wistar rats, radiolabelled EHDP was administered as a single oral dose, by gavage, at concentrations of 37–50 mg/kg bw, to 22 male animals (Nishimaki-Mogami et al, 1988; REACH a). The chemical was reported to be rapidly absorbed and distributed throughout the body, with highest levels of the chemical detected in blood, liver, kidney and adipose tissue, after the first two hours. Approximately 80 % of the radioactivity was detected in urine and faeces excreted in the first 24 hours. Within seven days, the radioactivity was almost completely excreted (48 % recovered in urine and 52 % in faeces). The major metabolites detected in urine were diphenyl phosphate and phenol, with p-hydroxyphenyl phenyl phosphate and monophenyl phosphate detected as minor metabolites.

Detection of diphenyl phosphate as the major metabolite is considered to indicate that hydrolysis of EHDP commences with rapid cleavage of the 2-ethylhexyl moiety (Testa and Mayar, 2003), therefore resulting in formation of 2-ethylhexanol (2-EH). However, detection of 2-EH, as a metabolite is not discussed in the available toxicokinetics studies.

Acute Toxicity

Oral

The chemical is expected to have low acute toxicity based on results from animal tests following oral exposure.

In a non-guideline acute oral toxicity study, a single oral dose of EHDP was administered by gavage to Sprague Dawley (SD) rats at 2000, 5010 and 7940 mg/kg bw (one female/dose) and 10000, 12600 and 15800 mg/kg bw (5 animals/sex/dose). One female died at each of the two highest doses. The oral median lethal dose (LD50) was reported to be >15800 mg/kg bw. Observed sub-lethal effects included diarrhoea, loss of appetite, lethargy and tremors. Intestinal inflammation and haemorrhagic areas of the liver, lungs and kidneys were also reported at autopsy (REACH a; REACH b).

Dermal

The chemical has low acute toxicity based on results from animal tests following dermal exposure.

In a non-guideline acute oral toxicity study, a single dose of EHDP was applied to the clipped intact skin of New Zealand White (NZW) rabbits at 3160 (one male), 5010 (one female) and 7940 mg/kg bw (one male and one female), for an exposure period of 24 hours, under semiocclusive conditions. No mortalities were reported. The dermal LD50 in rabbits was reported to be >7940 mg/kg bw. Observed sub-lethal effects included reduced appetite and activity (REACH a).

Inhalation

Based on the available data from a high quality animal study, EHDP is not considered to be acutely toxic following inhalation exposure. While another study indicates EHDP vapours may be acutely harmful following inhalation exposure, the available information is insufficient to warrant hazard classification.

In an acute inhalation toxicity study, conducted similarly to OECD Test Guideline (TG) 403, SD rats (five animals/sex) were exposed to 4.8 mg/L (mean measured concentration) of EHDP, as an aerosol, by whole body inhalation exposure, for a period of four hours (REACH a; REACH b). No mortalities were reported. Sublethal effects, observed during the 14-day post-exposure observation period, included reduced activity, respiratory distress and increases in secretory responses. The study concluded a median lethal concentration (LC50) in rats of >4.8 mg/L/4 hours.

In another acute inhalation toxicity study, an LC50 value of 2.1 mg/L (nominal concentration) was reported for rats (five males; species not specified), following exposure to EHDP vapours (REACH a; REACH b). However, this was following a six-hour exposure period (whole body inhalation exposure) to saturated vapours maintained at a temperature of 325°F (163°C). Mortalities (3/5 animals) were observed during the 10-day post-exposure observation period.

Corrosion / Irritation

Skin Irritation

The chemical is considered to be a slight skin irritant based on results from one animal study and observations in humans (see **Skin sensitisation - Observations in humans** section).

In a skin irritation study, conducted similarly to OECD TG 404, 0.5 mL of EHDP was applied to the clipped intact skin of three NZW rabbits, under semiocclusive conditions, for an exposure period of 24 hours, with an observation period of seven days (REACH a; REACH b). After 24 hours, slight skin irritation reactions (erythema and oedema) were reported (mean score of 2.3/8), and these persisted for at least 72 hours. No signs of oedema were observed after five days, and all effects were reversed by the end of the seven-day observation period.

Eye Irritation

The chemical is considered to be a slight eye irritant.

In an eye irritation study, conducted similarly to OECD TG 405, 0.1 mL of undiluted EHDP was instilled in to the right eye only of three NZW rabbits (REACH a; REACH b). The chemical was washed out of the eye after 24 hours, with an observation period of seven days. The left eye served as the control. Moderate erythema, slight oedema and copious discharge from the eye were reported after one hour. After 24 hours, no oedema or discharge were noted; however, slight erythema was still visible. Although detailed irritation scores are not available, all effects were fully reversed after 48 hours.

Sensitisation

Skin Sensitisation

Based on the limited available data from a study in animals, and observations in humans, EHDP is not considered to be a skin sensitiser.

In a skin sensitisation study in rabbits (reported as a patch test, with no further details provided), initial application of undiluted EHDP (volume not specified), followed by a subsequent application (volume not specified) two weeks later, reportedly did not induce skin sensitisation (REACH a; REACH b).

Observation in humans

In a human patch-testing study using 100 male and 100 female volunteers, EHDP (concentration not specified) was applied, as an occlusive patch, to the skin of the upper arm, for an exposure period of 48 hours (induction phase) (REACH a; REACH b). A challenge dose (concentration not specified) was then applied 13 days later. Skin irritation reactions were observed after the induction application in 30/200 volunteers. After the challenge application, skin reactions were observed in 29/200 volunteers; two of the volunteers who had showed skin reactions after the induction application did not show reactions at the challenge application, while one additional volunteer showed reactions to the chemical at the challenge application. All skin reactions were fully reversed within 10 days after removal of the patch, for both induction and challenge applications. Considering the occurance of skin reactions at the challenge phase was comparable to those observed at the induction phase, EHDP was not considered to be a skin sensitiser. However, EDHP was considered to be a moderate skin irritant in this study.

Repeated Dose Toxicity

Oral

In a 90-day dietary study in SD rats (10 animals/sex/dose), conducted according to OECD TG 408, a no observed adverse effect level (NOAEL) of 0.025 % EHDP in diet (equivalent to 17.35 mg/kg bw/day in males and 20.78 mg/kg bw/day in females), was reported (REACH a). Effects observed at higher concentrations, of 0.625 % in diet, (being the highest dose tested, and equivalent to 463.41 and 531.88 mg/kg bw/day, in males and females, respectively) included: reduced body weights; increased relative liver weights and associated changes in histological measures (indicative of centrilobular hypertrophy); increased relative adrenal weights; significant haematological changes in females only; and histopathological changes in the ovaries (hyperplasia and hypertrophy in the interstitial gland cells). An additional group of 10 animals/sex were administered 0.625 % EHDP in diet for 90 days, followed by a 28-day recovery period on control diet. While it was reported that most of the observed effects were considered to be reversed after the 28-day recovery period, hyperplasia and hypertrophy in the interstitial gland cells of the ovaries were still detected in 2/10 females in the recovery group. Minor reversible changes in the liver in males at 0.025 % in males are considered adaptive.

In another 90-day dietary study in SD rats, conducted similarly to the above study, and in accordance with OECD TG 408, a lowest observed adverse effect level (LOAEL) of 0.2 % EHDP was reported (equivalent to 164 and 174 mg/kg bw/day, for males and females, respectively) (REACH a, REACH b). Effects were observed in the liver, adrenals and ovaries, similar to those reported in the above study. No additional recovery group of animals was included in this study.

In a 28-day repeated dose study in SD rats, conducted according to OECD TG 407, six animals/sex/dose were administered EHDP, daily, by oral gavage, at 4, 20, 100 or 500 mg/kg bw/day. A NOAEL of 20 mg/kg bw/day was reported (REACH a). Effects at higher doses were observed in the liver and adrenals, similar to those reported in the above 90-day studies. Histopathological examination of the ovaries was not conducted in this study.

Dermal

No data available.

Inhalation

No data available.

Genotoxicity

Based on the weight of evidence from the available well-conducted in vitro and in vivo genotoxicity studies, EHDP is not considered to be genotoxic.

Negative results were reported for three in vitro point mutation assays (Ames tests), conducted according to OECD TG 471, in *Salmonella typhimurium* strains TA97, 98, 100, 1535, 1537 and 1538, *Escherichia coli* WP2uvra and *Saccharomyces cerivisiae* D4, with or without metabolic activation, at test concentrations up to 1000 µg/plate (REACH a; REACH b).

Negative results were observed in two mammalian cell in vitro assays on EHDP (REACH a; REACH b):

- an in vitro mammalian chromosome aberration test in Chinese hamster lung cells (CHL/IU cell line), conducted according to OECD TG 473, at various incubation periods (6–24 hours) and test concentrations (3–200 µg/mL), in the presence or absence of metabolic activation; and
- an in vitro mammalian cell gene mutation test in mouse lymphoma L5178Y cells, conducted according to OECD TG 476, at test concentrations of 0.002–0.025 µL/ml in the absence of metabolic activation, and 0.006–0.075 µL/mL in the presence of metabolic activation.

An in vivo mammalian bone marrow chromosome aberration test, conducted according to OECD TG 475, gave negative results. SD rats (24 animals/sex/dose) were administered a single oral dose of EHDP at 1500, 5000 or 15000 mg/kg bw. Rat bone marrow was examined for chromosome aberrations at six, 12 and 24 hours after administration. Although clinical signs of toxicity were observed, including reduced activity and decreased body weights, no differences in the mean number and the percentage of aberrant cells in bone marrow was reported. The chemical was not considered to damage chromosomes in rat bone marrow cells at up to the highest dose tested.

Carcinogenicity

While no reliable data are available for EHDP, its hydrolysis product, 2-ethylhexanol (2-EH), was reported to not be carcinogenic in a two-year oral gavage study in rats (NICNAS). The chemical was negative in all available genotoxicity assays.

Reproductive and Developmental Toxicity

The chemical is not considered to be a specific reproductive toxin. However, there is uncertainty regarding its potential as a developmental toxin.

Reproductive Toxicity

In a single generation reproduction study in SD rats, conducted according to OECD TG 415, 16 male and 32 female animals per dose-group received 0.2, 0.4 or 0.8 % EHDP in diet (equivalent to 132, 259, and 490 mg/kg bw/day for males, and 144, 293 and 536 mg/kg bw/day for females). Duration of treatment was 70 days for females and up to 119 days for males (REACH a; REACH b).

Significantly increased testes weights were observed in parental generation (P) males from the 0.4 and 0.8 % dose groups, while significantly increased ovary weights were observed in P females from the 0.8 % dose group only. Histopathological examination of the ovaries revealed significant increases in hypertrophy and hyperplasia of the interstitial gland cells in all females from the 0.8 % dose group. Similar effects were also observed in the 90-day repeated oral dose toxicity studies (see **Repeated Dose Toxicity - Oral** section). However, fertility was not affected by treatment, as no significant differences in mating or gestational indices were reported for any of the treatment groups, compared to the control group.

Signs of general toxicity included significantly reduced body weights in P males and females from the 0.4 and 0.8 % dose groups, and significantly increased relative organ weights (including liver, kidney, adrenal and brain) in P males and females across all treatment groups. Histopathological examinations were only conducted in animals from the 0.8 % (highest) dose group, revealing significant increases in cytoplasmic vacuolisation and hypertrophy of the liver, similar to effects observed in the 90-day repeat oral dose toxicity studies.

Effects observed in offspring (F1) included significantly reduced mean litter weights and body weight gain of pups from the 0.4 and 0.8 % dose groups. Significantly reduced pup survival was also reported for the highest dose (0.8 %) group. Significant differences in relative organ weights were observed in pups across all the dose groups (including liver, heart, spleen, adrenal and brain), while significantly decreased relative ovary weights were reported in female pups from the 0.8 % dose group only.

While effects on offspring of parental animals exposed to EHDP are evident, the effects were non-specific and only seen in the presence of significant parental toxicity.

Developmental Toxicity

In a prenatal development toxicity study, conducted according to OECD TG 414, pregnant female Charles River CD rats were administered EHDP by oral gavage at 300, 1000, or 3000 mg/kg bw/day (25 animals per dose), daily, from gestational day (GD) six to GD 15 (REACH a; REACH b).

While reduced body weight gains were observed in the 1000 (reported as slightly reduced) and 3000 (reported as severely reduced) mg/kg bw/day groups, these were reported to not be statistically different from control group values. Increased hair loss (not significant) was the only reported observation in the 300 mg/kg bw/day group females. No other statistically significant clinical signs of maternal toxicity were reported in this study.

Reported effects in foetuses included an increased number of foetuses with external malformations in the 1000 and 3000 mg/kg bw/day groups, while an increased number of foetuses with skeletal abnormalities (lack of ossification of one or two vertebrae and presence of 14th rudimentary ribs and sternebrae) were

reported across all treatment groups, as compared to the control group. The statistical significance of these effects is not reported. The values were reported to be within the range of the historical control values, and the effects occurred at doses at which toxic effects were seen in repeat dose studies.

Despite lack of statistical significance, NOAELs of 300-1000 mg/kg bw/day for maternal toxicity have been reported for this study (REACH a; REACH b), while a NOAEL of 3000 mg/kg bw/day (the highest dose tested) for developmental toxicity, was reported.

The chemical 2-ethylhexanol (2-EH), an expected hydrolysis product of EHDP (see **Toxicokinetics** section), is classified as a Category 3 hazardous substance toxic to reproduction, with the risk phrase 'Possible risk of harm to the unborn child' (Xn; R63) in HSIS (Safe Work Australia).

The chemical 2-EH was reported to cause developmental toxicity, but not teratogenicity, in rats following treatment via the oral route (NICNAS). These effects were noted in the absence of signs of marked maternal toxicity, and included markedly reduced mean foetal body weights and a higher number of foetuses with skeletal malformations, variations and retardations (similar to the effects noted in the above studies). The NOAEL for developmental toxicity was reported to be 130 mg/kg bw/day.

Based on the proportion of 2-EH formed on hydrolysis of EHDP, the equivalent dose of EHDP required to reach the level of toxicity reported for 2-EH (NOAEL of 130 mg/kg bw/day), is 360 mg/kg bw/day.

Risk Characterisation

Critical Health Effects

While no classifiable hazards have been identified, the possibility of developmental effects related to metabolism to 2-EH needs to be considered in establishing risk for EHDP.

Public Risk Characterisation

Currently, there are no restrictions in Australia on using EHDP in cosmetics or domestic products.

While use of EHDP in cosmetic or domestic products in Australia has not been reported, EHDP is reported to be used in domestic products overseas, that are potentially available for consumer use in Australia.

Considering the range of domestic products that may contain EHDP, the main route of public exposure is expected to be through the skin. Although the public could be exposed to EHDP through potential domestic uses, there is no available information on concentration of use for EHDP in domestic products. However, given the high NOAEL value (360 mg/kg bw/day) derived from studies on 2-EH, EHDP is not expected to pose a 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. Worker exposure to the chemical at lower concentrations could also occur while using formulated products containing the chemical. The level and route of exposure will vary depending on the method of application and work practices employed.

The chemical currently has no hazard classification for worker health and safety; this is considered appropriate based on the available data.

NICNAS Recommendation

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

Regulatory Control

Advice for consumers

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

Advice for industry

Control measures

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

- minimising manual processes and work tasks through automating processes;

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

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

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

Obligations under workplace health and safety legislation

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

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

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

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

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

References

Approved Criteria for Classifying Hazardous Substances [NOHSC:1008(2004)]. Third edition [NOHSC:1008 (2004)]. Accessed at **http://www.safeworkaustralia.gov.au/sites/swa/about/publications/Documents/258/ApprovedCriteria_Classifying_Hazardous_Substances_NOHSC1008-2004_PDF.pdf**

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

Globally Harmonised System of Classification and Labelling of Chemicals (GHS) United Nations, 2009. Third edition. Accessed at **http://www.unece.org/trans/danger/publi/ghs/ghs_rev03/03files_e.html**

National Industrial Chemicals Notification and Assessment Scheme (NICNAS). Inventory Multi-tiered Assessment and Prioritisation (IMAP) Human Health Tier II assessment for 2-ethylhexanol (CAS No. 104-76-7). Sydney, NSW: National Industrial Chemicals Notification and Assessment Scheme. Available: **http://www.nicnas.gov.au/chemical-information/imap-assessments/imap-assessments**

Nishimaki-Mogami, T., Minegishi, K., Tanaka, A. and Sato, M 1988. Isolation and identification of metabolites of 2-ethylhexyl diphenyl phosphate in rats. Archives of Toxicology. Volume 61 (4), pp 259-264

REACH Dossier. REACH b. 2-Ethylhexyl diphenyl phosphate (CAS RN 1241-94-7). Joint submission. Accessed September 2016 at **https://echa.europa.eu/information-on-chemicals/registered-substances**

Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) Dossier. REACH a. 2-Ethylhexyl diphenyl phosphate (CAS RN 1241-94-7). Individual submission. Accessed September 2016 at **https://echa.europa.eu/information-on-chemicals/registered-substances**

Safe Work Australia. Hazardous Substances Information System (HSIS). Accessed September 2016 at **http://hsis.safeworkaustralia.gov.au/HazardousSubstance**

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

Testa, B. and Mayar, J.M. 2003. The cleavage of esters of inorganic acids. In: Hydrolysis in drug and prodrug metabolism. Weinheim, Germany: Wiley, pp. 561-562.

The Poisons Standard (the Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP)) 2016. Accessed October 2016 at **https://www.tga.gov.au/publication/poisons-standard-susmp**

The US Environmental Protection Agency (EPA) Aggregated Computer Toxicology Resource (ACToR) database. Accessed September 2016 at **http://actor.epa.gov/actor/faces/ACToRHome.jsp**

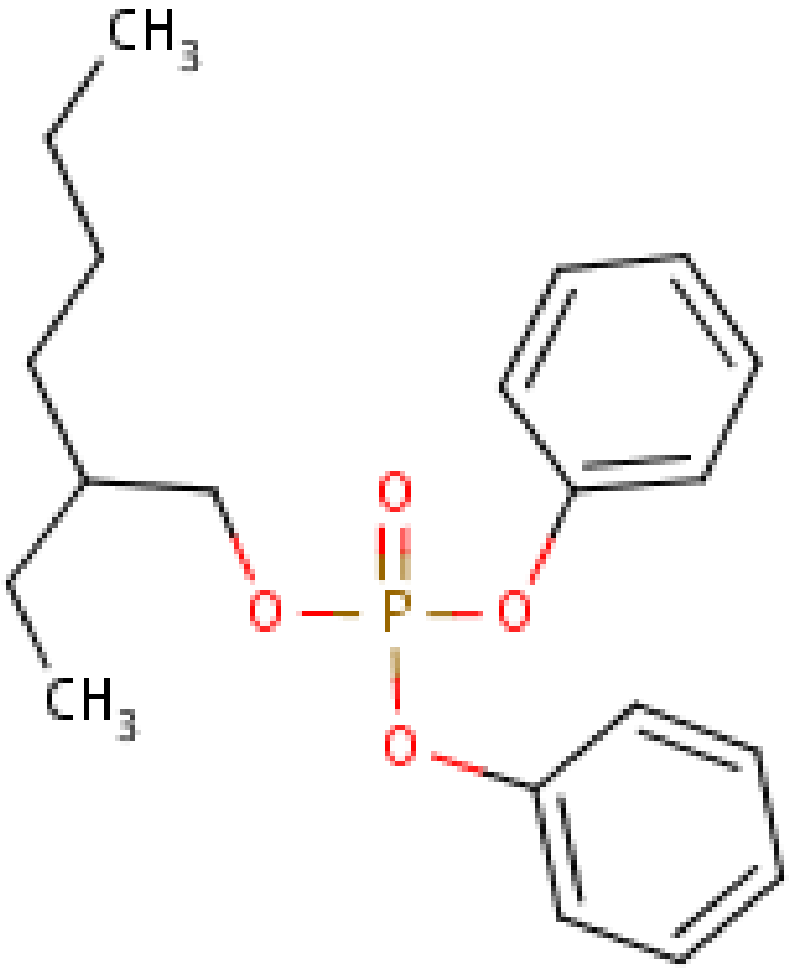
United Kingdom (UK) Environment Agency 2009. Environmental risk evaluation report:2-Ethylhexyl diphenyl phosphate (CAS no. 1241-94-7). Accessed August 2016, at **https://www.gov.uk/government/publications/environmental-risk-evaluation-reports**

US National Library of Medicine Hazardous Substances Data Bank (HSDB). Accessed September 2016 at **http://toxnet.nlm.nih.gov**.

Chemical Identities

Chemical Name in the Inventory and Synonyms	**Phosphoric acid, octyl diphenyl ester** diphenyl octyl phosphate octyl diphenyl phosphate
CAS Number	115-88-8
Structural Formula	
Molecular Formula	C20H27O4P
Molecular Weight	362.21

Chemical Name in the Inventory and Synonyms	**Phosphoric acid, 2-ethylhexyl diphenyl ester** 2-ethylhexyl diphenyl phosphate diphenyl-2-ethylhexyl phosphate EHDP Octicizer phosphoric acid, 2-ethylhexyl diphenyl ester
CAS Number	1241-94-7
Structural Formula	

	
Molecular Formula	C20H27O4P
Molecular Weight	362.4

Share this page