Risk Management Options Analysis

Substance Name: Di ethyl hexyl terephtalate (DEHTP)

EC Number: 229-176-9

CAS Number: 6422-86-2

Authority: France, ANSES

Date: January, 2015

Cover Note *The restriction of the use of various phthalates (DEHP, BBP, DBP, DIBP) has challenged the companies to find alternative solutions for product manufacturing. Among the potential replacements, DEHTP has emerged.*

In the framework on the French National Strategy on Endocrine Disruptors in 2014, the French Competent Authority requested ANSES to evaluate the toxicological profile of DEHTP and verify whether risk management measures should be necessary for this substance.

Comments and additional relevant information are invited on this RMOA by DD Month YYYY.

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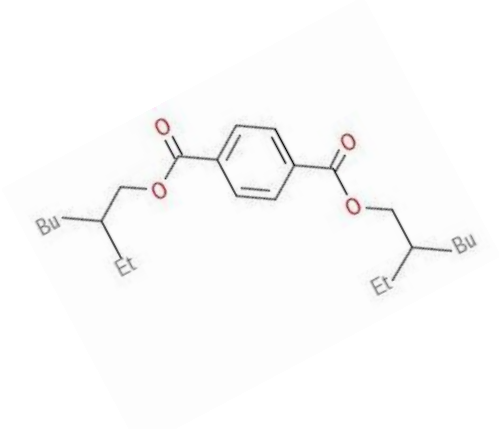
1 IDENTITY OF THE SUBSTANCE

Table 1: Substance identity

EC name:	Bis(2-ethylhexyl)terephthalate
IUPAC name:	Bis(2-ethylhexyl)terephthalate
Index number in Annex VI of the CLP Regulation	none
Molecular formula:	$C_{24}H_{38}O_4$
Molecular weight or molecular weight range:	390 g/mol
Synonyms/Trade names:	Di ethyl hexyl terephtalate (DEHT) Bis(2-ethylhexyl)terephthalate Di octyl terephtalate (DOTP) 1,4-benzene dicarboxylic acid (2-ethylhexyl) ester Eastman 168™ Non-Phthalate Plasticizer

Type of substance ☒ Mono-constituent ☐ Multi-constituent ☐ UVCB

Structural formula



Summary of physico-chemical properties

DEHTP is a clear, viscous liquid. The term phthalates is most often used to refer to ortho(o)-phthalate esters (also called o-phthalic acid esters), where the ester groups are attached ortho to the benzene ring. The chemical name for o-phthalic acid is 1,2-benzenedicarboxylic acid. If the ester groups are attached to the para-positions on the benzene ring, the phthalates are called terephthalates. The chemical name for terephthalates is 1,4-benzenedicarboxylic acid. The DEHTP is a terephthalate ester stoichiometrically equal to DEHP, i.e. phthalate ester bound to two ethylhexyl groups, but with a different spatial structure, because one of the carboxylic groups is placed differently on the benzyl ring ("tere" means tertiary, or third, because the carboxylic group is placed on the third carbon atom counted from the first carboxyl group).

Table 2: Physico-chemical properties of the substance

Property	Value	Method/Reference
Physical state	clear liquid	
Melting point	-48°C	Unknown / Beeler, 1976
Boiling point	383°C at 1015 hPa	Unknown / Beeler, 1976
Relative density	0.984 g/cm^3 at 25 °C	Unknown / Eastman Chemical Co.
Vapour pressure	1013 hPa at 398°C 0.001 Pa at 25 °C 2.85 E-5 hPa at 25°C	Measured / Eastman Chemical Co. Calculated / Eastman Chemical Co. Estimation / EPIWIN
Water solubility	0.0004 mg/l at 22.5°C	"Slow-stir" method; Eastman Chemical Co.
Partition coefficient n-octanol/water (log value)	8.39	EPIWIN Kowwin (v1.66)
Henry's law constant	1.18 E-5 atm-m^3/mol	Estimation / EPIWIN Henry (v3.10, Bond method)
Surface tension	32,7 mN/m (22°C)	EU Method A.5

2 REGULATORY PROCESSES

2.1 Completed/ongoing regulatory processes

Table 3: Completed or ongoing regulatory processes

☐ Compliance check, Final decision	☐ Dangerous substances Directive Directive 67/548/EEC (NONS)
☐ Testing proposal	☐ Existing Substances Regulation - Regulation 793/93/EEC (RAR/RRS)
☐ Annex VI (CLP) (see section 3.1)	☐ Plant Protection Products Regulation - Regulation (EC) No 1107/2009
☐ Annex XV (Candidate List)	☐ Biocidal Product Regulation - Regulation (EU) 528/2012 and amendments
☐ Annex XIV (Authorisation)	☐ CoRAP and Substance Evaluation
☐ Annex XVII (Restriction)	☒ RMO Analysis
☐ (UNEP) Stockholm convention (POPs Protocol)	☐ Other (provide further details below).

2.2 Other Relevant EU legislation for the substance/group of substances

Legal instrument	EU/national	Status of DEHTP
Plastics Regulation EU 10/2011on substances in contact with food	Scientific Panel on food additives, flavourings, processing aids and materials in contact with food (AFC) -18th list of substances for food contact materials	In January 2008 the scientific panel on AFC from EFSA evaluated the safety of DEHTP. Based on EFSA opinion, the substance was then authorized to be used in food contact materials. A TDI of 1 mg/kg bw/day was derived.
European References	Harmonised Standards EN 71-3 (Safety of toys - Part 3: Migration of certain elements); EN 71-5 (Safety of toys - Part 5: Chemical toys (sets) other than experimental sets)	DEHTP is not listed among the banned phthalates reported in the directives 1999/815/CEE and 2005/84.

	and EN 71-9 (Safety of toys – requirements concerning organic chemical compounds)	
Directive 2007/47/EC	**Directive on medical devices**	DEHTP is not listed among the banned substances in accordance with Annex I to Council Directive 67/548/EEC of 27 June 1967

3 HAZARD INFORMATION (INCLUDING CLASSIFICATION)

3.1 Classification

3.1.1 Harmonised Classification in Annex VI of the CLP

There is no existing Harmonised Classification for DEHTP.

3.1.2 Self classification

In the registration dossier there is no proposal for a classification.

3.1.3 CLP Notification Status

There is a notification for "no classification" for a total of 171 notifiers.

There is a notification by only one notifier for a classification as H361 "H361: Suspected of damaging fertility or the unborn child" and H413 "May cause long lasting harmful effects to aquatic life".

3.1.4 Proposal for Harmonised Classification in Annex VI of the CLP

Not relevant.

3.2 Additional hazard information

3.2.1 Existing assessments

Several hazard and/or risk assessments have already been conducted:

- In 2003, SIDS Initial Assessment Report (OECD, SIAM 17, 2003) concluded that DEHTP is currently of low priority for further work because of its low hazard profile.

- In 2008, an evaluation concerning DEHTP was done by the European Food Safety Authority (EFSA) which established a TDI (tolerable daily intake) of 1 mg/kg bw (EFSA, 2008).

- In 2009, RIVM conducted a risk assessment for DEHTP in toys and concluded that DEHTP is not expected to pose any health risk for toy-users at the migrated levels (low migration rate of 0.27-0.48) (RIVM, 2009).

- In 2010, Danish Environmental Protection Agency published a report on identification and assessment of alternatives to selected phthalates (No 1341, 2010). Suitable alternative plasticisers have been identified for most applications of the phthalates including DEHTP and DINCH (DEPA, 2010).

- In 2014, U.S. Consumer Product Safety Commission (CPSC) published a report entitled "Chronic hazard advisory panel on phthalates and phthalate alternatives". According to this report there is no evidence that DEHTP presents a hazard to infants or toddlers from mouthing toys or child care articles containing DEHT. Therefore, CHAP (Chronic Hazard Advisory Panel) recommends no action on DEHTP. However, information on total exposure to DEHTP is not available. The CHAP recommends that the appropriate U.S. agencies obtain the necessary exposure data to estimate total exposure to DEHTP and assess the potential health risks.

3.2.2 Current assessment

Hazards properties presented in this section are based on available data from the CSR of DEHTP, as well as on previous evaluations cited above, scientific literature and the review of the toxicological profile of DEHTP performed by Tox Services (Tox Services, 2012). It can be noticed that a detailed assessment of CSR data were not performed in the context of this RMOA.

3.2.2.1 Human health hazard assessment

Toxicokinetics

The major metabolite is the terephtalic acid (TPA). In an *in vitro* study in rats it was established that a complete hydrolysis of DEHTP occurred which is converted to 2-ethylhexanol and terephthalic acid.

In rat, following absorption the DEHTP was rapidly hydrolyzed to 2-ethylhexanol (2-EH), mono-(2-ethylhexyl) terephthalate, and unlabeled terephthalic acid and these metabolites were absorbed from the gastrointestinal tract. More than 36% of the administered dose was not absorbed and was excreted unchanged in the feces.
The results of this study indicate that about 63% of the administered dose of DEHTP was hydrolyzed to 2-EH, mono-(2-ethylhexyl) terephthalate and terephthalic acid. 2-Ethylhexanol and mono-(2-ethylhexyl) terephthalate were largely metabolized and excreted in urine and feces. The major hydrolytic product was unlabeled terephthalic acid and the major excretory products were TPA and DEHTP, together accounting for 87% of the dose. The excretion of unchanged DEHTP is presumed to be due to limited solubility or the availability of the substance to hydrolytic enzymes. Only a small portion of dose (maximum of 10%) was excreted as mono-(2-ethylhexyl) terephthalate or oxidative metabolites of mono-(2-ethylhexyl) terephthalate. DEHTP is metabolized differently than its isomer, DEHP. While DEHTP is hydrolyzed predominantly to terephthalic acid, DEHP is hydrolyzed largely to mono-(2-ethylhexyl) phthalate.

The rate of percutaneous absorption of DEHTP through sections of human skin was measured in an *in vitro* study. In this study, the absorption rate was measured after an application in excess of the substance.
The rate was 0.103 ± 0.052 µg/cm^2/hr and therefore the test substance would be classified as "extremely slow" with respect to its absorption through human skin, therefore the systemic exposure would be very limited via the dermal route. These data allow the estimation of uptake in man following dermal exposure to the test substance, assuming that skin absorption in man is similar to that observed in this *in vitro* study.

Acute Toxicity

Three studies were available in the registration dossier for the acute oral toxicity. In the key study performed in rats no mortality was observed at a dose of 5000 mg/kg in both males and females. Two additional studies performed in male rats and mice lead to LD_{50} > 3200 mg/kg.
Therefore, no classification is warranted for this endpoint.

In the study performed in guinea pig by dermal route the LD_{50} was > 20.0 mL/kg bw which is equivalent to 19680 mg/kg bw. Therefore, no classification is needed.

No study was available to assess acute toxicity following exposure via the inhalation route.

Additionally, two studies in which male rats and mice were exposed to DEHTP via the intraperitoneal route were provided. For both the LD_{50} was > 3200 mg/kg.

Irritation

In a GLP compliant study available in the CSR conducted according to the OECD guideline 404, male and female New Zealand white rabbits (2 male/1 female) were exposed to 0.5 ml of undiluted DEHTP under occlusive conditions for 4 hours. Followed with a 72 hour observational period. No irritating effects were observed (neither erythema nor edema), and DEHTP was reported as non-irritating under the tested conditions.
In an older study male guinea pigs were exposed to 4920; 9840 or 19680 mg/kg bw for 24 hours followed by a 14-day observation period. Only one animal was exposed per dose. No mortality was observed. After these 2 weeks, no erythema but moderate to severe edema was reported for high dose and low/mid-doses respectively. According to this study the DEHTP should be classified as irritating nevertheless several deviations are observed compared to the guideline. The current guideline specifies that animals should be exposed for 4-hour (not 24), require at least 3 animals per dose and the maximal dose on exposure site should be 0,5 g (but 5-20g were used in this study). Therefore, the reliability for this study is low.
Finally, the substance was evaluated in 18 human subjects (9 men and 9 women). In this study, DEHTP was applied in semi-occlusive patches and the subjects were patched three times over a period of five days (Days 1, 3, and 5). The subjects removed the patches after 24 hours, and scoring of patch sites for irritation was made prior to applications on Days 3 and 5 and on Day 8. Only a minimal irritation was observed, and was then not considered as related to the substance since the effect does not occur in dose-dependant manner.

Concerning potential eye irritation, a GLP and OECD 405-compliant eye irritation/corrosion study was conducted using male and female New Zealand white rabbits (1 male/2 females). Rabbits were exposed to undiluted DEHTP in one eye for 4 hours, with a 72 hours observational period following exposure. No corneal opacity or iritis was observed during the study. Conjunctivitis and redness were reported up to 48 hours after administration. All reported effects were fully reversible within 72 hours and therefore DEHTP is not considered as an eye irritant.
Additionally an older (Teehaar,1975) non-GLP compliant eye irritation/corrosion study was conducted using New Zealand white rabbits (n=6, sex not reported). The rabbits were exposed to undiluted DEHTP in one eye. At 24 hours after exposure one rabbit showed adnexal staining of the nictitating membrane. At 48 hours after exposure all animals appeared normal. Therefore, DEHTP is not warranted to be classified as an eye irritant as all effects were reversible within a 48-hour time period.

DEHTP does not need to be classified for skin or eye irritation/corrosion.

Sensitisation

A dermal sensitization HRIPT study (Human Repeat Insult Patch Test - modified Draize method) was conducted using human volunteers (9/sex) (Lockhart, 2001b). Humans were exposed to nine dermal applications of 0.5% DEHTP in acetone under semi-occlusive conditions over a three-week induction period. Following a two weeks rest period a challenge dose of 0.5% was applied to the skin. DEHTP appeared to be non-irritating and non-sensitizing in all volunteers.
Additionally, a non-GLP compliant dermal sensitization study was conducted using guinea pigs (strain/sex not reported, n=5). The Guinea pigs were exposed to a 1% solution of DEHTP via injection into the footpad followed by a 1% dermal application challenge dose. No signs of sensitization were observed and therefore DEHTP was reported as non-sensitizing under the tested conditions. Nevertheless, due to poor reporting, this study does not have a high reliability.
Overall, DEHTP does not need to be considered as a sensitizer.

Repeated dose studies

In a GLP compliant study by Barber and Topping (1995) conducted in male and female Sprague-Dawley rats, animals received either 0; 54-61 (0.1%); 277-309 (0.5%) or 561-617 (1.0%) mg/kg bw/day of DEHTP in feed during 90 days. This study included a peroxisome study for which 5 male rats were randomly assigned to receive 1000 mg/kg bw/day of a positive control (2-ethylhexanol; known to cause liver enlargement and hepatic peroxisome proliferation). No effects were reported on clinical signs and mortality, body weight and body weight gain, food consumption and compound intake, ophthalmoscopic examination, clinical chemistry, urinalysis, gross pathology and histopathology. Mean hemoglobin, hematocrit, Mean Corpuscular Hemoglobin (MCH) and Mean Corpuscular Volume (MCV) were significantly lower than controls in the top dose male group (4-5% decreases). Mean MCH values were also lower in the mid-dose male rat group (2%). Slight (3%), but statistically significant decreases in MCV and MCH values were observed in mid- and top-dose female rats. Authors concluded that changes in hematology were minimal in severity, and not clearly dose-dependent and were therefore not of biological significance. Absolute liver weight increases (9%) and liver weights relative to body weights ratio increase (11%) were observed in males in the highest-dose group. Only relative liver weight changes reached statistical significance. In females, the absolute liver weight was increased by 7% and the relative liver weight was increased by 9% in the highest-dose groups.

Again, only relative liver weight changes reached statistical significance. Concerning the peroxisome assay, there was no indication of peroxisome induction in animals from the 1.0% dose group. In contrast, the positive control caused a 28% increase in the liver peroxisome fraction and a 33% increase in peroxisome density. Overall, based on the effects observed on hematology and the liver weight changes the study authors established a **NOEL of 277 mg/kg bw/day in males and 309 mg/kg bw/day** (0.5% DEHTP).

In an older supportive study (Teerhar, 1975), three groups of five male Sprague-Dawley rats were exposed through diet to DEHTP at doses of 0; 85 (0.1%) or 885 (1.0%) mg/kg bw/day. No mortality was observed. Only few effects were observed, on clinical chemistry but within the historical values. Moreover, after an histopathology analysis some effects on the lungs in the highest treated group were observed : tracheitis and bronchiolitis but they were not considered of biological relevance since some control animals had the same. **The NOEL was established at 1.0%** (equivalent to 885 mg/kg bw/day)**.**

And finally a 21-day study (Topping *et al*., 1987) was conducted in order to establish a dose-response relationship for the peroxisomal and related effects of DEHTP. Thus five 344 Fisher rats /sex/ doses were exposed to 0; 0.1; 0.5; 1.0; 1.2 or 2.5% (equivalent to 100, 505-487, 1037-1052, 1247-1244, and 2104-1900 mg/kg/day in male and females respectively) of DEHTP through feed. DEHP was used as a positive control.. The exposure to DEHTP induce a substitial reduction of the feed consumption which lead to a significant reduction in body weight in animals exposed to 2.5%. The rats fed with a diet containing 2.5% DEHTP showed slight hepatic peroxisome proliferation. However, since there was such a large reduction in food consumption and body weight gain at this dose, it cannot be concluded that 2.5% DEHTP alone caused this change since feed intake restriction alone has been shown to double the peroxisomal oxidizing activity of liver in rats. **A NOEL of 0.5%** was chosen based on the effects observed on liver weight and on the clinical chemistry

In a supportive study five male albino rats were exposed by inhalation route during 14 days to 0.0718 mg/L of DEHTP 6 hours a day during 14 days. No mortality occured and only minor changes were observed during the study. Therefore, a NOEL of 0.0718 mg/L was derived.

Additionally a study in which five Dunkin-Hartley guinea pig were exposed through dermal route is available. An equivalent of 813 to 1144 mg/kg bw/day of the test substance was applied to the clipped skin of animals once a day for 9 applications over an 11-day period. No mortality was observed. Moreover no signs of skin absorption nor systemic toxicity were evident during the study. Under the conditions of this study, there was no exacerbation of the irritant response with repeated applications of DEHTP since the first application produced moderate erythema in one animal and severe erythema in the other four. Slight edema was observed for all animals but this disappeared by study termination. Necrosis and eschar were not observed in this study.

In conclusion, in the available repeated toxicity studies for DEHTP, few effects were observed on the liver weight, otherwise no toxicity was noticed. Based on the results available, it appeared that DEHTP is not a peroxisome proliferator triggering the biochemical and cellular changes in the liver, contrary to DEHP. The lowest NOEL was 0.5% of DEHTP equivalent to 277 to 309 mg/kg bw/day in males and females.

Genetic Toxicity

Four key *in vitro* studies are available. In two Ames tests (Barber, 1984 and 1994) using a method similar to OECD 471 with Salmonella strains TA1535, TA1537, TA1538, TA98, and TA100, with DEHTP concentrations up to 10,000 µg/plate with or without metabolic activation negative results were observed. An additional mutation assay followed guidelines similar to OECD 476 (HGPRT assay, Barber, 1994), with Chinese hamster ovary (CHO) cells exposed to DEHTP concentrations up to the cytotoxic limit (20 nL/mL) was also negative. In the chromosomal aberration assay (Barber, 1994) which followed guidelines similar to OECD 473 no structural damage was induced in CHO cells exposed to DEHTP at concentrations up to 1,000 nL/mL (the protocol limit of the test).
A supportive Ames test, giving also negative results, is also available (Di Vincenzo *et al*., 1985).

Since all these *in vitro* studies were negative, no *in vivo* study was conducted and DEHTP should not be considered as genotoxic.

Carcinogenicity

A GLP compliant 104-week carcinogenicity study (conducted according to EPA OPPTS 870.4200 guideline) was conducted using male and female Fischer 344 rats (50/sex/dose). Rats were administered doses of 0, 79, 324, and 666 mg/kg and 0, 102, 418, and 901 mg/kg in males and females respectively daily in the diet. There was no evidence of a treatment-related effect on the incidence of any tumor type for any group of rats. There were no statistically significant dose-related differences in incidences of specific tumors between treated and control groups. Toxic effects observed were limited to reduced body weight gain and food conversion efficiency in the two highest-dose groups. Moreover, the DEHTP exposure increased the incidence of eosinophilic inclusions in the nasal turbinates and atrophy of the outer nuclear layer of the retina (in females exposed to 418 mg/kg-day), but the study author regarded these as not toxicologically significant. Therefore a NOEL for tumorigenicity of 666 mg/kg in males and 901 mg/kg in females was established by the study authors.

Since there was no evidence of a tumorigenic response neither in males nor in females rats following a life time exposure to DEHTP, the substance should not be considered as carcinogenic.

Toxicity to reproduction

A GLP-compliant two generation reproductive toxicity study (OECD 416) (Stump, 2001a) is available and was conducted using male and female Sprague-Dawley rats (30/sex/dose). Rats were administered doses of 0, 0.3, 0.6, and 1.0% (0, 133-182, 265-367 and 447-614 mg/kg in males, and 0, 184-478, 372-940, and 595-1349 mg/kg in females for F0 and 0, 159-256, 320-523, and 552-893 mg/kg in males and 0, 206-516, 423-1036, and 697-1549 mg/kg in females in F1) of DEHTP in the diet from 70 days pre-mating to termination in the F0 generation and from PND 22 until termination in the F1 generation. Reproductive parameters (fertility, mating, days between pairing and coitus, gestation, parturition, and estrous cycling) as well as mean litter sizes, numbers of pups born, percentages of males per litter at birth and postnatal survival were unaffected. Female rats displayed systemic toxicity in the 516 and 860 mg/kg groups including decreased food consumption. Slight decreases in organ weights in the top dose F1 group were considered to be secondary to maternal toxicity. Additionally, no dose-response could be established. Based on available data, a NOAEL of 1.0% was

established by study authors for the reproductive toxicity. Additionally, a NOAEL of 0.3% for parental toxicity has also been derived based on the mortality observed both in F0 and F1 parental animals at the highest dose and the effects on the body weight. Moreover, mean weekly body weights were reduced for both males and females in the 1.0% group throughout the F1 generation and for F1 males in the 0.6% group beginning on study week 23. Increases in absolute mean (F0 females) and in relative mean (to final body weight) liver weights (F0 and F1 females) were observed in the 0.6 and 1.0% groups. Finally, a NOAEL of 0.3% was established for neonatal toxicity since mean F1 male and female offspring weights and weight gains in the 0.6 and 1.0% groups were reduced throughout the pre-weaning period. In the F2 offspring, neonatal toxicity was also exhibited by reduced offspring weight gains in the 0.6 and 1.0% groups during lactation.

Three studies are available for developmental toxicity.
A GLP compliant developmental toxicity study (OECD 414, Stump 2001b) with additional uterotrophic evaluations was conducted using female Sprague-Dawley rats (25/group). Rats were administered doses of 0, 0.3, 0.6 and 1.0% (equivalent to 0, 226, 458, and 747 mg/kg bw/day) of DEHTP on days 0 through 20 of gestation. In the uterotrophic examinations sexually immature rats were administered doses of 20, 200, and 2,000 mg/kg via oral gavage on post natal days 19 to 21. Number of viable and non-viable fetuses, resorptions and implantation sites, and corpora lutea did not differ from controls. No visceral or skeletal anomalies and no signs of developmental toxicity were reported. In the uterotrophic assay for estrogenic activity, DEHTP exposure did not affect wet or blotted uterine weight parameters, but effects were observed for the positive control used the 17 alpha Ethinyl Estradiol. A NOAEL of 1.0% for developmental toxicity was established by the study authors since intrauterine growth and survival and fetal malformations were unaffected by test substance administration at any dose level. There was nevertheless an increased occurrence of rudimentary 14th ribs observed in the 1.0% group. This effect was considered as test substance-related, but was not considered as an adverse effect. A NOAEL of 0.6% was also established by the authors for maternal toxicity based on the effects observed on body weights and liver weights at 1.0%.

A GLP compliant developmental toxicity study (OECD 414) was conducted using female CD-1 mice (25/group). Mice were administered doses of 0, 197, 592 and 1,382 mg/kg of DEHTP in the diet on days 0-18 of gestation. No effects were observed on the number of malformations/skeletal variations, litter size, fetal body weights or sex ratios. No evidence of fetotoxicity or teratogenicity was observed even at maternally toxic doses. A NOEL of 1,385 mg/kg was identified for teratogenicity by the authors since the intrauterine growth and survival were unaffected at all dosage levels and a NOAEL 197 mg/kg bw/day for the maternal toxicity was established based on the higher absolute mean for liver weight at the two highest-doses.

Finally, a developmental toxicity limit test (Gray *et al*, 2000) was conducted using female Sprague-Dawley rats. Rats were administered doses of 0 or 750 mg/kg of DEHTP (98% purity) on gestation day 14 through postnatal day 3 via oral gavage. No maternal toxicity, fetotoxicity, or teratogenicity was reported at any dose level. A NOEL of 750 mg/kg was then reported by the study's authors for both maternal and developmental toxicity, but this study is only supportive due to the poor reporting.

Additional supporting data (Tox Services, 2012) is available. In this supportive study five pregnant Sprague-Dawley rats were exposed from gestation day 12 through gestation day 19 (8 days) to DEHTP terephthalate at 500 mg/kg bw/day.

On gestation day 19, all females were euthanized by carbon dioxide inhalation, fetuses were weighed, and anogential distance was obtained. Fetuses were sacrificed, sexed, and the right and left testes were removed. Genes associated with pathways involving lipid, sterol, and cholesterol transport, steroidogenesis, intracellular lipid and cholesterol homeostasis, oxidative stress, insulin signaling, and transcriptional regulation, were evaluated in the present study using Real-time Quantitative Reverse Transcription-Polymerase Chain Reactions. A total of 18 genes were investigated. No statistically significant alterations were noted in any of these genes in animals exposed to DEHTP. Moreover, anogenital distance was not significantly altered in male fetuses exposed to the test substance.

Hence, DEHTP is not considered as toxic for reproduction.

It should be noticed that one of the minor metabolites, i.e. 2-ethylhexanoic acid is classified as Repro. 2 H361d (Suspected of damaging the unborn child). This metabolite is currently evaluated by Spain under the REACH Substance evaluation procedure. The way to take into account this metabolite in the scope of this RMO A has been raised.
One important consideration to be taken into account is that, bis(2-ethylhexyl) phthalate (DEHP) has also 2-ethylhexanoic acid as a minor metabolite. The interspecies variability to DEHP has been documented in the report for its identification as SVHC: in a non-human primate reproductive study, no effects are observed contrary to what is reported for rodents. Therefore, the available data show that laboratory primates are less susceptible than rodents to the reprotoxic effects of DEHP and eventually to its metabolites (ECHA, 2014).
Taken together, it seems that reproductive rodent studies appear to be a reasonable worst case for phthalates and their metabolites covering human intraspecies variability. It has been reaffirmed that the relevance of the rodents studies for humans for DEHP is not questioned (CPSC, 2014).
Additionally, contrary to DEHP, no reproductive effects have been observed in the 2-generation study available for DEHTP in rodents.

Therefore, based on all the information available, DEHTP can be considered having a low toxic potential and appears to be far less toxic than the phthalates it is intended to replace.

3.2.2.2 Environmental fate properties

DEHTP is a highly insoluble substance (water solubility: 0.0004 mg/L) with surfactant properties (surface tension: 32.7 mN/m at 22°C) and little tendancy for volatilization (0.001 Pa at 25°C). It can thus be considered as a "difficult substance" (meaning difficult to test).

Photodegradation

No information is available about photodegradation of DEHTP. However, the half life ($T_{1/2}$) in the atmosphere is 0.487 days based on AOP (v1.90) estimation (EPIWIN v3.10).

Hydrolysis

The test OECD 111 indicates that hydrolysis of DEHTP is unlikely to occur in the pH range of 4 to 9.

Biodegradation

A first ready biodegradability test was performed using ^{14}C-labeled DEHTP. Microorganisms (mixed liquor and raw sewage) were adapted for 2 weeks to DEHTP before the test. Radioanalysis indicated that 40.2% of DEHTP was converted to CO_2 and gas chromatographic measurements showed that 56.2% of the DEHTP was lost from the medium. This study shows that DEHTP is ultimately biodegradable but not ready biodegradable.

A second ready biodegradability test was conducted (Test OECD 301B) with non-adapted sludge using fine silica gel to increase the surface area and the bioavailability of the substance to the microorganisms. The study shows that 73% of DEHTP was converted to CO_2 after 28 days in the "10-days window". This last study shows that DEHTP fulfill the criteria of ready biodegradability.

Simulation tests on ultimate degradation in surface water or sediment were not conducted for DEHTP since DEHTP fulfill the criteria of ready biodegradability according to the previous test. However, an ecotoxicity test performed on sediment organisms (*Chironomus riparius*) at 100, 180, 320, 560 and 1000 mg DEHTP/kg call the ready biodegradability of DEHTP into question. The sediment spiked with DEHTP was composed by 76% w/w industrial quartz sand, 20% w/w kaolinite clay and 4 % w/w sphagnum moss peat (insuring the presence of microorganisms). In this study, DEHTP concentrations were measured in sediment, in overlying and interstitial water. Results indicates that DEHTP were mainly in sediment (the concentrations in overlying and interstitial waters were less than the limit of detection, except for the highest concentration tested). The concentrations measured in sediment were 90-121% of the nominal concentrations at 0 days and 77%-97% of the nominal concentration after 28 days. Thus, these results indicate that the microorganisms in the sediment were not able to degrade DEHTP and that the persistence time of DEHTP once adsorbed on sediment can be long.

With regard to the metabolites, in the literature, Nalli et al. (2002) identified one degradation product of DEHTP produced by the bacteria *Rhodococcus rhodochrous* in aqueous media, namely 2-ethylhexanoic acid. The bacteria grown one week with DEHTP under aerobic conditions at 30°C in mineral salt media supplemented with 0.05 g/L yeast extract and hexadecane as an another carbon source. Indeed, DEHTP was not degraded unless another carbon source was also present. The concentrations of DEHTP and 2-ethylhexanoic acid was monitored during the test. The results showed that half of the DEHTP was degraded after one week and that 2-ethylhexanoic acid concentration increased during the test to represent 3% of the parent compound at the end of the test. 2-ethylhexanoic acid is on CORAP list 2012 for concern relating to suspected toxicity on fertility and is currently evaluated by Spain competent authorities. Thus, PBT or vPvB properties of this substance will be assessed in the near future.

Environmental distribution

Experimental attempt to measure the partitioning of DEHTP to soils and sediment did not allow for adequate measurement of log Koc (possible adsorption of the substance onto the surfaces of the glassware). Thus, log Koc was estimated using KOCWIN QSAR model with a log Kow input of 8.39. A value log Koc of 5.07 was obtained from EPI Suite (v2.00) indicating that DEHTP would adhere strongly to soil and sediment particles.

There is no available information on DEHTP concentrations in the environment. However, Barnabé *et al.* (2008) reported DEHTP and 2-ethylhexanoic acid concentrations in a wastewater treatment plant in Montreal (WWTP based on a physico-chemical process). Mass flow of DEHTP was estimated to 110 kg/d in the influent (i.e. 51 ± 3 µg/L). DEHTP was removed from influent with 72% efficiency (i.e. 14 µg/L of DEHTP measured in effluent) but significant quantities were measured in the sludge. For instance, DEHTP concentration reach 104 ± 5 mg/kg in press-filtered sludge and 45 ± 2 mg/kg in homogenized sludge. Overall, 25% of incoming DEHTP was found in dewatered sludge. Thus, if the sludge is disposed by land application, significant amount could be found in soil. In addition, the metabolite 2-ethylhexanoic acid was observed in all aqueous process streams (36 µg/L in influents and 15 µg/L in effluents). Overall, even though the wastewater treatment plant accomplished the removal of an appreciable fraction of DEHTP in the liquid matrix, the treated effluent and sludge still represent a significant source of DEHTP and 2-ethylhexanoic acid in the environment.

2-ethylhexanoic acid is also found in appreciable amount in the environment despite this metabolite is expected to be easily biodegraded (cf. CSR of 2-ethylhexanoic acid). Horn at al., (2004) measured concentrations of 2-ethylhexanoic acid at 110 µg/kg in sediment, 6.7 µg/L in melted snow and 3.2 µg/L in the St Laurence River water.

It can be noticed that the biodegradation of other plasticizers such as DEHP and DEHA has been shown to result in the production of the same metabolite 2-ethylhexanoic acid (Horn et al., 2004; Nalli et al., 2002). Thus, it is difficult to assess the fraction of 2-ethylhexanoic acid assignable to DEHTP degradation only. It is presumed that significant amount of 2-ethylhexanoic acid are observed in the environment due to the high rate of release of all the plasticizers (not DEHTP only).

Bioaccumulation

The bioaccumulation studies in a saltwater mollusk show no bioaccumulative potential (BCF = 393 L/kg). However, there is no available information on aquatic vertebrate (i.e. fish) to conclude on bioaccumulative potential of DEHTP. Due to the active surface properties of the substance, a BCF-fish can not be extrapolated by QSAR. Therefore, no conclusion can be draw on the bioaccumulation potential in aquatic vertebrate organisms.

3.2.2.3 Environmental hazard assessment

Aquatic toxicity (water and sediment)

No toxicity was observed in fish, invertebrate and algae in any of the short-term or long-term aquatic toxicity studies at exposure concentrations that were often significantly greater than its limit of solubility (0.4 µg/L). Organic solvents were used to obtain DEHTP concentrations higher than its solubility (acetone or DMF). In the majority of the aquatic studies organisms were exposed to DEHTP in flow through conditions and test concentrations were analytically confirmed. The NOECs were at the highest concentrations tested in all studies. Results are reported in the following table.

In sediment, an effect on early larval emergence of *Chironomus riparius* was observed at a nominal sediment concentration of 180 mg/kg but the reported EC_{50} value was in excess of 1000 mg/kg based on development rate.

Regarding the metabolite toxicity (2-ethylhexanoic acid), Horn et al. (2004) show that this transformation product exhibits acute toxicity using Microtox, Daphnia, rainbow trout and fathead minnow toxicity assays (EC_{50} are respectively 43, 23, 150 and 120 mg/L). Moreover, this substance is classified as toxic for reproduction category 2 and thus fufils the toxicity criterion (T) for PBT assessment. 2-ethylhexanoic acid is on CoRAP list 2012 because of suspected toxicity on fertility, wide dispersive use, consumer use, high tonnage and risk >1 for human health. This substance is being evaluated by Spain.

Table 4: Overview of acute and long term toxicity of DEHTP on aquatic organisms (water and sediment)

Organism	**Test**	**Results**	**Reference**
Rainbow trout *Salmo gairdneri*	Acute 7-d, flow-through, Freshwater Acetone added	No mortality or abnormal effects LC50 ≥ 250 µg/L NOEC ≥ 250 µg/L	
Fathead minnow *Pimephales promelas*	Acute 96-h static, freshwater Nominal concentrations	No mortality or abnormal effects LC50 ≥ 984 µg/L 1 NOEC ≥ 984 µg/L1	
Rainbow trout *Salmo gairdneri*	Early Life Stage Toxicity Test, Long-Term 71-d, flowthrough, freshwater Acetone added	No effects on hatchability, survival or growth. NOEC ≥ 280 µg/L	
Water flea *Daphnia magna*	Acute 48-h, static, Freshwater Solvent added (DMF)	No immobility or adverse effects. EC50 ≥ 1.4 µg/L NOEC ≥ 1.4 µg/L	
Planorbid snail *Helisoma trivolvis*	Acute 96-h static, Freshwater Nominal concentration	No mortality or abnormal effects EC50 ≥ 984 µg/L 1 NOEC ≥ 984 µg/L 1	
Eastern Oyster *Crassostrea virginica*	Acute 96-h flow-through, Marine Acetone added	No mortality or inhibition of shell deposition. EC50 ≥ 624 µg/L NOEC ≥ 624 µg/L	
Water flea *Daphnia magna*	Full Life Cycle Toxicity Test, Long-Term 21-day, flow through, freshwater Acetone added	No effects noted on survival, growth, or reproduction. EC50 ≥ 0.76 µg/L NOEC ≥ 0.76 µg/L	
Green alga *Selenastrum capricornutum*	72-h static, growth inhibition,	No inhibition of biomass or growth rate	

	freshwater Solvent added (DMF)	EC50 ≥ 860 µg/L NOEC2 ≥ 860 µg/L	
Midge *Chironomus riparius*	Long-Term Static, 28-Day	No differences in larval growth. An effect on early larval emergence was detected that was significant at concentrations > 180 mg/kg NOEC (emergence) = 180 mg/kg dw EC50>1000 mg/kg	

[1] An oily film was observed on the surface, indicating that the material was not soluble at this concentration

Toxicity to waste water treatment microorganisms

The toxicy of DEHTP on waste water treatment microorganisms was evaluated with a 3h activated sludge respiration inhibition test. Respiration rate of microorganisms exposed to DEHTP was equivalent to negative control rates. Results are reported in Table 3.

Table 5: Toxicity of DEHTP on waste water treatment microorganisms

Organism	Test	Results	Reference
Activated sludge	3-Hour activated sludge respiration inhibition test	No differences in oxygen consumption EC50 > 10 mg/L NOEC ≥ 10.0 mg/L	

Toxicity to terrestrial compartment

Only data on terrestrial plants are available in the CSR. No data on the other relevant terrestrial organisms were submitted.

The acute toxicity of DEHTP was evaluated for 3 plant species under hydroponic system in a seeding growth test for 14 days. The plants (radish, ryegrass and soybeans) were exposed to ^{14}C-DEHTP via the nutriment solution and acetone was added. The results are reported in the following table.

Table 6: Overview of acute toxicity of DEHTP on terrestrial plants

Organism	**Test**	**Results**	**Reference**
Radish *Raphanus sativus*	Acute 14-Day Early Seedling Growth Acetone added Radiolabeled	No apparent effect. EC50 > 1400 µg/L (NOEC = 1400 µg/L)	
Ryegrass	Acute	No apparent	

Lolium perenne	14-Day Early Seedling Growth Acetone added Radiolabeled	effect. EC50 > 1400 µg/L (NOEC = 1400 µg/L)	
Soybeans *Glycine max*	Acute 14-Day Early Seedling Growth Acetone added Radiolabeled	No apparent effect. EC50 > 1500 µg/L (NOEC = 1500 µg/L)	

Environmental endocrine disruption properties

No alert was found in literature on potential environmental endocrine disruption properties of the substance. No activity was demonstrated in the 118 bioassays conducted in the ToxCast framework (on 212 data: 200 inactive, 11 inconclusive and 1 unspecified).

3.2.2.4 Conclusion for environmental fate and hazard

DEHTP is highly insoluble substance with surfactant properties and little tendancy for volatilization. It is thus a "difficult substance" to test. DEHTP has log Koc value of 5.07 indicating that DEHTP would adhere strongly to soil and sediment particles. A biodegradation test showed that DEHTP was ready biodegradable (73% degradation measured via CO_2 evolution, silica gel used to increase its bioavailability). However, an ecotoxicity test performed on sediment organisms (*Chironomus riparius*) call the ready biodegradability of DEHTP into question. Indeed, the concentrations measured in sediment were 90-121% of the nominal concentrations at 0 days and 77%-97% of the nominal concentration after 28 days. Thus, these results indicate that the microorganisms in the sediment were not able to degrade DEHTP and that the persistence time of DEHTP once adsorbed on sediment can be long. A bioconcentration study in oysters indicated that DEHTP has low potential to bioconcentrate in this species (BCF=393 L/kg). However, there is no available information on bioaccumulative potential of DEHTP for aquatic vertebrate (i.e. fish) to conclude. Studies assessing acute and chronic toxicity to fish, invertebrates and algae showed no effects. In sediment, an effect on early larval emergence of *Chironomus riparius* was observed at a nominal sediment concentration of 180 mg/kg but the reported EC_{50} value was in excess of 1000 mg/kg based on development rate. The growth of 3 species of terrestrial plants was not impacted by DEHTP when tested under hydroponic system. No data on other terrestrial organisms are available. No alert was found in literature on potential environmental endocrine disruption properties of the substance. However, the toxicity (Reproductive toxicity Cat. 2 H361d) and the presence in the environment of one degradation product (2-ethylhexanoic acid) has raised concerns. Despite this metabolite is not expected to be persistent in the environment, appreciable amounts are reported in a variety of environmental samples. This metabolite is on CORAP list 2012 and is being evaluating by Spain. It can be noticed that the biodegradation of other plasticizers such as DEHP and

DEHA has been shown to result in the production of the same metabolite 2-ethylhexanoic acid. Thus, it is difficult to assess the fraction of 2-ethylhexanoic acid assignable to DEHTP degradation only.

4 INFORMATION ON (AGGREGATED) TONNAGE AND USES

4.1 Tonnage and registration status

There are 12 registration dossiers and one lead registrant (Eastman).

The substance is registered for a tonnage band 10,000 to 100,000 but according to the lead resgistrant this registration band was selected in anticipation of future sales.

Currently there are some importers from Asia and Turkey as well as domestic suppliers. Eastman continues to increase imports to Europe as phthalate substitution accelerates.

From ECHA dissemination site		
☒ Full registration(s) (Art. 10)	☐ Intermediate registration(s) (Art. 17 and/or 18)	
Tonnage band (as per dissemination site)		
☐ 1 – 10 tpa	☐ 10 – 100 tpa	☐ 100 – 1000 tpa
☐ 1000 – 10,000 tpa	☒ 10,000 – 100,000 tpa	☐ 100,000 – 1,000,000 tpa
☐ 1,000,000 – 10,000,000 tpa	☐ 10,000,000 – 100,000,000 tpa	☐ > 100,000,000 tpa
☐ <1 . . . . . >+ tpa (e.g. 10+ ; 100+ ; 10,000+ tpa)		☐ Confidential

4.2 Overview of uses and exposure information

DEHTP is an important phthalate-free plasticiser, being the diester of terephthalic acid and the branched-chain 2-ethylhexanol. It is used as a general purpose plasticizer for softening PVC plastics. It possesses very good plasticizing properties and may be used as a replacement for ortho-phthalates in many applications.

Applications/Uses according to Eastman (2009b):

- Bottle caps and closures
- Coatings
- Coatings for cloth
- Electric connectors
- Flexible film
- Pavement striping compounds
- Sheet vinyl flooring

- Toys
- Traffic cones
- Vinyl compounding
- Vinyl gloves
- Vinyl products
- Vinyl water stops
- Walk-off mats

Use	
Plasticizer in platics and rubber processing (plastisols and PVC articles). DEHTP is used as a general-purpose plasticizer for polyvinyl chloride (PVC) and polyvinyl chloride/vinyl acetate (PVC/VA) copolymers.	☒ Manufacture ☒ Formulation ☐ Uses at industrial sites ☒ Uses by professional workers ☒ Consumer Uses ☐ Article service life
Medical devices such as • Infusion bags • Tubing • Gloves • Catheters • etc.	☒ Manufacture ☒ Formulation ☐ Uses at industrial sites ☒ Uses by professional workers ☒ Consumer Uses ☐ Article service life
Toys	☒ Manufacture ☒ Formulation ☐ Uses at industrial sites ☒ Uses by professional workers ☒ Consumer Uses ☐ Article service life
Construction formulation additives	☐ Manufacture ☐ Formulation ☐ Uses at industrial sites ☒ Uses by professional workers ☒ Consumer Uses ☐ Article service life
Coatings & Inks (CEPE)	☒ Manufacture ☐ Formulation ☐ Uses at industrial sites ☒ Uses by professional workers ☒ Consumer Uses ☐ Article service life
Laboratory use	☐ Manufacture ☐ Formulation ☐ Uses at industrial sites ☒ Uses by professional workers ☐ Consumer Uses ☐ Article service life
Food contact materials : DEHTP is used as a plasticizer up to approximately 30% in PVC materials, coming into contact with all kinds of foodstuffs under all conditions both for single and repeated use. Typical products can be wraps, tubing, conveyor belts and sealing gaskets.	☒ Manufacture ☒ Formulation ☐ Uses at industrial sites ☐ Uses by professional workers ☒ Consumer Uses ☐ Article service life
Adhesives and Sealants (FEICA)	☒ Manufacture ☐ Formulation

	☐ Uses at industrial sites ☐ Uses by professional workers ☐ Consumer Uses ☐ Article service life

None of the above mentioned uses is advised against.

From the consultation held with Eastman it seems that by far the highest volume use is flooring (75-85% of the total volume of DEHTP).

4.2.1 Occupational Exposure

According to the OECD SIDS, workplace exposure to DEHTP during manufacture is minimized by the use of enclosed equipment, engineering controls, the low volatility of the substance and through the use of good industrial hygiene practices, which include personal protective equipment such as gloves and a dust mask as appropriate.

The primary use of DEHTP is as a plasticizer where it is bound up in a polymer matrix. Although exposure of workers to DEHTP during processing into final products has not been quantified, exposure is likely minimized through the use of enclosed equipment and by good industrial hygiene practices. Processing is done in both closed and open equipment. In both closed and open equipment, exposure is minimized by the use of localized exhaust and subsequent catalytic incineration or aerosol capture of any DEHTP volatilized from the polymer matrix.
Exposure to vapours is unlikely because the vapour pressure for DEHTP is low (estimated to be 2.85 E-5 hPa at 25 °C) unless it is heated where (at the lowest measured temperature of 270 °C the vapour pressure was still only 13.3 hPa). According to the US DEHTP producer, incorporation of DEHTP into products does not require heating to a temperature greater than 149°C (300°F).
Exposure to an aerosol is unlikely during loading for storage and transport, and the likelihood of significant inhalation or dermal exposure is further reduced through the use of good industrial hygiene practices (i.e. personal protective equipment such as gloves and a dust mask if the worker deems it appropriate).

4.2.2 Consumer Exposure

According to the OECD SIDS, exposure by consumers has not been quantified, but is considered to be minimal based on the very limited use of DEHTP in consumer products. Furthermore, exposure is primarily limited to the dermal route. Systemic exposure by the dermal route is significantly attenuated, as shown by the fact that DEHTP has an extremely low percutaneous absorption rate (0.103 ± 0.052 µg/cm2/hr). The only consumer product that creates a potential for direct dermal exposure is "coated fabrics". These fabrics have a flexible vinyl coating applied to them in order to make them waterproof.
Importantly, the coating is applied to only a single side of the fabric (i.e., the outside), thus significantly limiting the amount of dermal contact that may occur. In the case of waterproof fabrics for hospital beds, the vinyl-coated side is located directly against the mattress. This is followed by the placement of a conventional cotton sheet over the top of the non-coated side, further reducing a patient's potential exposure to the vinyl coating. Typically, the vinyl coating on such fabric contains 23-26% DEHTP.

Some human exposure to DEHTP may occur as a result of the presence of this substance in the environment. As discussed above, concentrations of DEHTP in the environment have not been reported, but air and water concentrations are expected to be low based on very limited vapour pressure and water solubility.

5 JUSTIFICATION FOR NO FURTHER ACTION

The presently available information indicates that DEHTP is not expected to pose any health or environmental risks. DEHTP is not considered as toxic for reproduction and no alert was found on potential endocrine disruption properties of the substance.

Nevertheless, some uncertainties remain.

First, further relevant experimental evidence for this compound would strengthen the environmental risk assessment, more specifically the PBT assessment (additional tests for the persistence of DEHTP in sediment and soil, bioaccumulative potential in aquatic vertebrates, toxicity to terrestrial organisms).

Second, the safety for 2-ethylhexanoic acid, a metabolite of DEHTP which is classified as Repro. 2 H361d, should also be judged, specifically for the environmental assessment. The 2-ethylhexanoic acid is being evaluated by Spain under the REACH substance evaluation procedure (CoRAP list 2012). Therefore, no further action is required for DEHTP until Spain states on this metabolite risk for environment. As far as human health is concerned, this metabolite is not expected to pose any risk. Nevertheless, the evaluation of DEHTP may be reconsidered depending on the outcome of the evaluation of this metabolite.

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