

**ATTACHMENT 2a**

**ENDPOINT SUMMARIES FOR MAMMALIAN AND ENVIRONMENTAL TOXICITY  
SUBMITTED IN THE REACH REGISTRATION DOSSIER FOR  
DIISONONYL PHTHALATE (DIDP)**

**AND**

**TABLE OF CONTENTS FOR DIDP DOSSIER INFORMATION  
ON THE ECHA WEBSITE  
AND PROVIDED TO CPSC ON A DVD**

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## Explanation of Contents

ExxonMobil Chemical Company is submitting the attached information to assist Consumer Product Safety Commission's Chronic Hazard Advisory Panel in its evaluation of diisodecyl phthalate (DIDP). This information consists of extracts from the dossier submitted to the European Chemicals Agency (ECHA) to support registration of DIDP under REACH.<sup>1</sup> The data in that dossier was prepared in accordance with REACH requirements and ECHA guidance.<sup>2</sup>

Our package – consisting of this document and pdfs provided via DVD (Attachment 2b) – includes key information from the registration dossier that has been posted to the Internet by ECHA.<sup>3</sup> This information can be accessed by the public; however, to make it more easily accessible to you, we have printed out each webpage of the DIDP entries.<sup>4</sup> In addition, we are providing endpoint summaries which supplement the toxicity data contained in Sections 5, 6, and 7. The website and our package do not provide the full dossier for DIDP and do not include certain production and analysis information that was claimed as Confidential Business Information in accordance with European Union law and REACH guidance.<sup>5</sup> These sections have been included in Attachment 1b as labeled blank pages just as they are shown on the ECHA website.

The dossier for DIDP has been accepted by ECHA and the substance is registered as of December 1, 2009. Registration indicates that ECHA has undertaken a completeness check and has ascertained that all elements required under REACH have been provided. The completeness check does not include an assessment of the quality or adequacy of the submitted information.<sup>6</sup> It is ExxonMobil's belief, however, that the enclosed data is accurate and provides a useful summary of the database for this product.

Please let us know if you have any questions about the enclosed materials or about their significance within the REACH context. You can contact Angela Rollins, Oxo Americas Regulatory Affairs Advisor, at [angela.rollins@exxonmobil.com](mailto:angela.rollins@exxonmobil.com).

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<sup>1</sup> Regulation (EC) No 1907/2006 of the European Parliament and of the Council of 18 December 2006 concerning the Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH), O.J. L 396, 30.12.2006, pp. 1-849.

<sup>2</sup> See ECHA, Guidance Documents, [http://guidance.echa.europa.eu/guidance\\_en.htm](http://guidance.echa.europa.eu/guidance_en.htm).

<sup>3</sup> ECHA, Search for information on registered substances, <http://apps.echa.europa.eu/registered/registered-sub.aspx>. To access the information, search on the CAS registry number (DINP - 68515-48-0; DIDP - 68515-49-1).

<sup>4</sup> For DINP, we have provided that information submitted under CAS registration number 68515-48-0, which is the number for ExxonMobil's product.

<sup>5</sup> See ECHA (2007), Guidance on data sharing, Chapter 11: Confidential Business Information (CBI), European Chemicals Agency, Guidance for the Implementation of REACH, [http://guidance.echa.europa.eu/docs/guidance\\_document/data\\_sharing\\_en.pdf](http://guidance.echa.europa.eu/docs/guidance_document/data_sharing_en.pdf)

<sup>6</sup> See REACH, Article 20.

## Table of Contents for DIDP Dossier Information on the ECHA Website

Robust summaries of the studies on which the following endpoint summaries are based are available on the ECHA website. The following is the table of contents for the information on the website.

The website can be accessed by the public at <http://apps.echa.europa.eu/registered/registered-sub.aspx> (search on CAS registry number 68515-49-1). However, use of the site requires lots of clicking; therefore, ExxonMobil is providing via DVD a series of pdfs scanned from a printout of the web pages. This table of contents can be used to navigate those pdfs.

Because of the large database for DIDP and the formatting of the ECHA summaries, the hardcopy printout is very large (588 pages). Therefore, we are providing only the pdf form, but would be pleased to provide hard copy upon request.

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## **Endpoint Summaries of Mammalian Toxicity Data Submitted in the REACH Registration for DIDP**

### **Toxicokinetics, Metabolism and Distribution – Section 7.1**

The disposition and metabolism of DIDP was evaluated in Sprague Dawley rats (mean body weights 200g) where radiolabeled DIDP (carboxyl-14C) was administered in corn oil by gavage at doses of 0.1, 11.2, and 1000 mg/kg (General Motors Research Laboratories, 1983). Feces were collected at 24 hour intervals and urine over 12 hour intervals. At termination of the collection periods, the animals were sacrificed and the carcass, brain, lungs, heart, thymus, liver, spleen, kidneys, adrenals, testes, fat, and gastrointestinal tract were weighed and frozen for storage.

Radioactivity was determined by liquid scintillation spectrometry. To assess pre-systemic elimination, biliary cannulation was conducted on anaesthetized animals to determine the percentage of administered radioactivity recoverable in the bile. DIDP and its metabolites in tissues, bile and excreta were separated using high-pressure liquid chromatography. Metabolites of DIDP were identified by co-elution with standards.

#### *Absorption*

The percentage of total administered dose excreted in urine during the 72 hour collection interval decreased with increasing dose from 41.3% to 32.1% and 12.6% after 0.1, 11.2, and 1000 mg/kg doses, respectively. In addition, the percentage of total administered dose excreted in bile during the 72 hour collection interval decreased with increasing dose from 14.3%, 13.8%, and 4.7% for the low, middle and high doses, respectively. The sum of urinary and biliary excretion can be used as an estimate of total administered dose leading to 55.6%, 49.5% and 17.3% after 0.1, 11.2, and 1000 mg/kg doses. Distribution: A limited distribution was observed throughout the body where only 0.5, 0.8 and 0.2% of the administered radioactivity was detected in the carcass of low, middle, and high dose animals, respectively. For doses of 0.1, 11.2, and 1000 mg/kg, radioactivity was detected in the gastrointestinal tract (0.49%, 0.77%, 0.17%, respectively), liver (0.06%, 0.08%, 0.03%, respectively) and kidneys (0.01%, 0.01%, 0.00%, respectively). The content of tissues in  $\mu$ mole equivalents of DIDP increased with increasing dose.

#### *Metabolism*

In urine, phthalic acid and the oxidized monoester derivative were detected but not mono isodecyl phthalate (MIDP) or DIDP at any of the doses. The percentage of radioactivity associated with the monoester derivative group increased with increasing dose from 52% after 0.1 mg/kg to 72% after 1000 mg/kg. Concurrently, the proportion of radioactivity associated with phthalic acid was observed to decrease from 38 to 18% after 0.1 and 1000 mg/kg doses, respectively. In feces, the monoester oxidative derivative, MIDP and DIDP were detected. With increasing dose, the percentage of parent compound recovered in feces increased from 30% with 0.1 mg/kg to 55% with 11.2 mg/kg to 60% with 1000 mg/kg. The percentages of oxidative derivatives of the monoester and of MIDP were 25 and 30%, 14 and 26%, 13 and 13% after the low, middle and high doses, respectively. It was noted that due to potential bacterial degradation of DIDP or its metabolites in feces, quantification of the data was difficult.

#### *Elimination*

The primary route of excretion of radioactivity was in feces accounting for 57.5, 65.6 and 81.7% of the total body burden after 0.1, 11.2, and 1000 mg/kg, respectively. Biliary excretion accounted in part for the content in feces. Excretion in urine was biphasic and there was no apparent dose-effect

relationship on the rates of elimination. The percentage of total administered dose excreted in urine during the 72 hour collection interval decreased with increasing dose from 41.3 to 32.1 to 12.6% after low, middle, and high doses, respectively. Elimination in urine and feces together represented greater than 99% of the administered dose for all doses.

### **Acute Toxicity – Section 7.2**

In a series of LD50 studies reported by Smyth et al. (1962), the LD50 of DIDP in rats is above 62,080 mg/kg. In a 24-hour exposure dermal study (Hazleton Laboratories America, 1978) in rabbits (4 animals, sex not specified), a dose of 3,160 mg/kg was applied on abraded skin and remained in contact with the skin by means of a non-absorbent binding. There was no mortality during the 14-day test period. The dermal LD50 was therefore estimated to be greater than 3,160 mg/kg. (Industrial Bio-Test Laboratories, 1975) was conducted in rats, mice and Guinea pigs (5 males and 5 females) at 0.13 mg/l (nominal concentration). DIDP was reported to have been administered as a vapor, but regarding the test conditions the substance was probably administered as an aerosol. No deaths occurred, no adverse reactions were noticed following the 14-day observation period, and there were no gross tissue changes attributable to effects of the test. The LC50 is >0.13 mg/l.

### **Irritation – Section 7.3**

Six male rabbits were exposed to 0.5 ml of the test material during 4 hours, the patch held in contact with the skin by means of a semi-occlusive dressing. Only very slight erythema in one animal was noted at 60 minutes after removal of the patch. No other signs of irritation at 24, 48 and 72 hours were observed (Exxon Biomedical Sciences, 1996).

In a study reported without any detail, a 24-hour uncovered application of DIDP (mixed isomers) did not produce any irritating effect on rabbit belly (Smyth et al., 1962).

A study using Lawrence's method was conducted in 10 mice; undiluted DIDP produced no signs of irritation when administered by the intraperitoneal route (Lawrence et al., 1973).

Primary irritation potential of 0.2 ml of undiluted DIDP was evaluated during a single 24-hour application (occluded patch) on 14 female subjects and 1 male subject. Examinations at 30 minutes and 24 hours after patch removal did not reveal any sign of irritation (Hill Top Research, 1995b cited in Medeiros et al., 1999).

In a briefly reported study, eye injury in rabbits was assessed considering the degree of corneal necrosis that results from instillation of various volumes and concentrations of chemicals. Grade 1 irritation was obtained for DIDP which indicated, at most, a very small area of necrosis resulting from 0.5 ml of undiluted chemical in the eye (Smyth et al., 1962). In a briefly reported study, undiluted DIDP did not produce any obvious irritation (Lawrence et al., 1973).

In a study conducted in 6 rabbits (Industrial Bio-test Laboratories, 1975), undiluted DIDP produced only slight signs of irritation on the conjunctiva at 1, 4 and 24-hour observation times (redness score 1 or 2 and discharge at 1 hour, redness score 1 at 4 hours and redness score 1 in 1 animal at 24 hours). All the eyes were normal at 48, 72 and 96 hours.

No indication of upper airway irritation was reported following acute inhalation exposure in animals. Local irritant effects were reported in rats exposed repeatedly (5 d/w for 2 weeks) by inhalation to DIDP aerosol at 0.5 mg/l.

### **Sensitization – Sections 7.4 and 7.10**

DIDP did not produce a sensitizing response in two guinea pig studies conducted by the methods of either Buehler (method defined by Directive 92/69/EEC B.6) or Magnusson and Kligman (Huntingdon Research Centre, 1994; Inveresk Research International, 1981, respectively). In both studies, DIDP gave negative results for sensitization with no evidence of irritating effect. Positive controls functioned appropriately.

One guinea pig study conducted according to Buehler gives a clear positive response (Exxon Biomedical Sciences, 1992). The marked response obtained in this study is inconsistent with other information. The strong irritant effect during induction phase, only observed in this assay, is also surprising, considering the minimal evidence for irritancy in other studies. Accordingly, as the results of this study are neither consistent nor plausible. The other two studies (Huntingdon Research Centre, 1994; Inveresk Research International, 1981) are considered more appropriate for assessing the sensitization potential of DIDP.

No positive reactions were reported in patch test studies conducted in humans (Medeiros et al., 1999). Consequently the evidence suggests that DIDP does not cause sensitization in humans.

### **Repeated Dose Toxicity – Section 7.5**

In a Hazelton Laboratories, 1968 study, DIDP was administered to four groups of 10 male and 10 female rats in dietary levels of 0.05%, 0.3% and 1% (approximately 25, 150 and 500 mg/kg/d, respectively). A group of untreated rats served as control. No compound-related effects were observed at any dietary levels with regard to physical appearance, behavior or survival. Growth of the test rats was not significantly affected. Body weight gains for the two highest levels in males were lower than controls (but not significantly different) and the two test groups were comparable through the ninth week. Overall, weight gains at 13 weeks for the male test groups showed a dose-related, although slight, decrease. Body weight gains for the high dose females were only slightly lower than the controls. Food consumption values were comparable to the controls. The clinical laboratory values for the test groups showed no significant compound-related differences from control values.

Observations at necropsy revealed the livers of the high dose group animals, particularly the males, to be markedly larger than those of the control rats. Statistical analysis revealed the liver weights and liver/body weight ratios for the high dose group males and females to be significantly higher than those for the corresponding controls. No other consistent gross changes were noted in the liver. Histologically, the liver showed no compound-induced alterations. The kidney/body weight ratios but not the absolute weights in the high and intermediate dose group males were significantly higher than those for the corresponding controls. Histologically, the kidneys showed no compound-induced alterations.

A minimal increase in thyroid activity was observed at the highest dose. It can be concluded from this study that the NOAEL is 0.3% (about 150 mg/kg/d) based on the fact that the highest dose leads to liver and thyroid effects. Relative (but not absolute) kidney weights were affected at this intermediate dose, probably due to a lower body weight.

In a 2-week study (General Motors Research Laboratories, 1981) designed to evaluate the fate of DIDP (see Section 5.1), toxicity was assessed. DIDP was administered to 8 male rats (6 for control) by inhalation (aerosol) at analytical concentration of  $505 \pm 7$  mg/m<sup>3</sup> (MMAD: 0.98  $\mu$ m) 6 hours a day, 5 times per week. Rats were observed daily for body weight gain, appearance and gross behavior. Animals were sacrificed at the end of the observation period (3 weeks) and tissue samples taken for histopathology. There were no marked outward signs of toxicity during exposure. The rate of body weight gain was not different between control and exposed animals. Effects in the lungs were: moderate increase in the width of alveolar septa with slight interstitial mixed inflammatory reactions, alveolar macrophages and type II pneumocytes were increased in number, peribronchial lymphoid tissue appeared slightly more prominent. In liver, spleen and kidneys, no obvious histologic alterations were noted except for a slight hepatic fatty metamorphosis. No systemic toxicity, but local irritant effects were observed at the concentration tested, thus a NOAEL of 0.5 mg/l (500 mg/m<sup>3</sup>) can be assumed.

### **Mutagenicity/Genotoxicity – Section 7.6**

DIDP is not mutagenic in vitro in bacterial mutation assays (with and without metabolic activation) and is negative in a mouse lymphoma assay (Zeiger et al., 1982; 1985; Hazleton Biotechnologies Company, 1986; Barber et al., 2000). DIDP is not clastogenic in a mouse micronucleus assay in vivo (Hazleton Washington, 1994; McKee et al., 2000).

### **Carcinogenicity – Section 7.7**

A two year bioassay was conducted in which DIDP was administered in the diet at concentrations of 400, 2000, and 8000 ppm to F344 rats (Cho et al., 2008). The average daily doses of DIDP were reported to be calculated from the body weights and feed consumption data using the concentrations of DIDP in the diet. For doses of 400, 2000, and 8000 ppm, the calculated average daily doses of DIDP over 2 years for male rats reported in the paper are incorrect. Actual exposures for male rats were 21.9, 110.3 and 479.2 mg/kg-bw/day and for female rats 22.9, 128.2 and 619.6 mg/kg-bw/day (personal communication with Wan-Seob Cho). Rats of both sexes exhibited significant decreases in overall survival and body weights, and increases in the relative weights of kidneys and liver with 8000 ppm DIDP. No treatment related neoplastic lesions were observed in the internal organs, including the liver. In addition, measurement of catalase enzyme activity, a marker for cell peroxisome proliferating activity, suggests that DIDP can induce peroxisome proliferation at an early stage (12 weeks of treatment) but fails to maintain the catalase-inducing potential by 32 weeks of treatment. An increased incidence of mononuclear cell leukemia (MNCL) was observed in this study, but MNCL is a common neoplasm in F344 rats, and the observed increased incidence is likely to be a species-specific effect with little or no relevance to humans. Therefore, DIDP was not considered to be carcinogenic at doses up to 8000 ppm in rats.

### **Reproductive and Developmental Toxicity – Section 7.8**

#### *Fertility*

Reproductive toxicity of DIDP was evaluated in two 2-generation reproductive toxicity tests (Exxon Biomedical Sciences, 1997b and 2000; key data published in Hushka et al., 2001). Rats were exposed by dietary administration to levels of DIDP ranging from 0.0 to 0.8% (or approximately 15 to 600 mg/kg/day). There were no statistically significant differences in male mating, male fertility, female fertility, female fecundity, or female gestational indices between treated and control animals in

the P1 or P2 generation (Table 14, Hushka et al., 2001). Mean days of gestation and mean litter size and of the treated and control groups were similar. There were no statistically significant differences in the mean sex ratio of the treated offspring compared with controls. It was concluded that fertility was unaffected by DIDP treatment at levels up to 600 mg/kg/day.

In both studies, liver and kidney effects were observed in the P1 generation. Increased liver weights and associated hepatocellular hypertrophy were observed at dietary concentrations of 0.4% and greater in both studies. These dietary concentrations also produced kidney effects that were associated with alpha 2u microglobulin toxicity, a male rat specific effect and thus not relevant to humans. In the first study, minor effects on the liver were observed at 0.2% (103 – 203 mg/kg/day). In the second study, no hepatic effects were recorded at this concentration (114 – 225 mg/kg/day). As there is a range in intake levels, it is likely this dietary concentration results in ingestion of DIDP at or near the NOAEL for systemic effects from repeated dosing. Up to the highest dose tested no overt signs of reproductive toxicity were reported, and no effects were observed on fertility parameters. In the P2 generation liver and kidney changes were observed in the P2 males. A NOAEL of approximately 33 to 76 mg/kg bw/d (0.06%) was derived with the range being due to the fact received doses differed depending on the period considered. Up to the highest dose tested no overt signs of reproductive toxicity were reported, and no effects were observed on fertility parameters.

#### *Development*

Developmental toxicity studies of DIDP conducted at doses of 100, 500, and 1000 mg/kg provided evidence of slight and transient signs of maternal toxicity at 1,000 mg/kg/d (significant reversible decrease of body weight gain and food consumption) suggesting a conservative NOAEL of 500 mg/kg/d for maternal toxicity. The only statistically significant changes were skeletal variations (supernumerary cervical and rudimentary lumbar ribs) on a per litter basis at the high dose. Rudimentary ribs are a common finding in rat fetuses and should not be regarded as associated with malformations, but may only be related to transient maternal stress. It should be noted that supernumerary ribs were located in the cervical region which is less common (Waterman et al., 1999), but the biological significance of cervical supernumerary ribs remains uncertain. A NOAEL of 500 mg/kg/d may be assumed for skeletal variations.

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## **Endpoint Summaries of Environmental Toxicity Data Submitted in the REACH Registration for DIDP**

### **Stability – Section 5.1**

Indirect photochemical degradation of di-isodecyl phthalate (DIDP) as mediated by OH<sup>-</sup> attack is estimated to have a half-life of 0.41 days or 4.9 hours based on a 12 -hour sunlight day, a rate of 2.62E-11 cm<sup>3</sup>/molecule\*sec, and an average OH<sup>-</sup> concentration of 1.5E6 OH<sup>-</sup>/cm<sup>3</sup>. A 12-hour day half-life value normalizes degradation to a standard day light period during which hydroxyl radicals needed for photolysis are generated in the atmosphere. Although DIDP has the potential to degrade rapidly by OH<sup>-</sup> attack, multimedia distribution modeling indicates DIDP is predicted to partition negligibly (0.1%) to the air compartment because it has a low vapor pressure (0.000051 Pa). Although DIDP has a relatively short atmospheric oxidation half-life (4.9 hours), this process is unlikely to contribute significantly to the loss of DIDP from the environment.

Results from the EQC (Equilibrium Criterion) Levels I and III distribution models (Mackay, 2001) show that because of its low water solubility, DIDP is not expected to partition to the water compartment (see Section 4.2.3). However, abiotic degradation of any trace amounts of DIDP which may be present in aquatic environments is unlikely to occur at a significant rate based on modeled data. The HYDROWIN model, a subroutine within the USEPA (2000) computer program, estimates a hydrolysis half-life for DIDP of 3.4 years at pH 7 (25°C) and 125.2 days at pH 8 (25°C). Direct photochemical degradation in water occurs through the absorbance of solar radiation by a chemical substance. If the absorbed energy is high enough, then, in the resultant excited state, the chemical may undergo a transformation. A prerequisite for direct photodegradation is the ability of one or more bonds within a molecule to absorb ultraviolet (UV)/visible light in the 290 to 750 nm range. Light wavelengths longer than 750 nm do not contain sufficient energy to break chemical bonds, and wavelengths below 290 nm are shielded from the earth by the stratospheric ozone layer. An approach to assessing the potential for DIDP to undergo direct photochemical degradation is to assume that degradation will occur in proportion to the amount of light wavelengths >290 nm absorbed by DIDP molecules. DIDP does not absorb light within a range of 290 to 750 nm. Therefore, direct photolysis will not contribute to the degradation of DIDP in the aquatic environment because it does not absorb light at wavelengths in the range that contributes to this process.

### **Biodegradation – Section 5.2**

DIDP is readily biodegradable (74% biodegradation in 28 days), and does meet the 10-day window based on results from a standard OECD ready biodegradation test guideline (EMBSI, 2009). DIDP also biodegraded to 67.0% in 28 days and was readily biodegradable but did not meet the 10 -day window (EBSI, 1995). The principle transformation products would be mono-isodecyl phthalate (MIDP) and isodecyl alcohol (IDA) (Staples et al., 1997b). Data are available to assess the potential biodegradability of these two transformation products. Biodegradation data are available for a mono ester that is a mixture containing approximately equal amounts of monoesters with normal octyl (n-C8) and normal decyl (n-C10) side chains. Results show that show that this mixture is readily biodegradable (Scholz, 2003) and data also show that IDA is readily biodegradable, but does not meet the 10-day window (Exxon Biomedical Sciences, Inc., 1997a). The n-C8/n-C10 mono ester biodegraded to 94% after 28 days, while IDA biodegraded to 71% after 28 days.

Studies are not available to assess the biodegradability of DIDP under simulated conditions (i.e., wastewater treatment). However, there are data for di-n-decyl phthalate (CAS No. 84-77-5; DnDP), an

analog to DIDP, using treated wastewater that suggest DIDP would demonstrate a high extent of biodegradation under wastewater conditions (Furtmann, 1993). DnDP biodegraded 82% after 7 days based on the disappearance of the parent compound from the test system. The initial DnDP concentration was 7.8 µg/l and the DT50 was 1 day.

The elimination of DIDP in a sewage treatment plant (STP) through biodegradation and distribution, as reported by the European Commission (2003), was estimated using the SIMPLETREAT model. The model calculated that 91.9% of DIDP would be eliminated in a STP, which is consistent with the high loss reported by Furtmann (1993). The measured data for DnDP and the modeled data suggest that DIDP will be largely eliminated in a STP.

Studies are not available to assess the biodegradability of DIDP in sediment. Although there are no data specifically for the diester, there are biodegradation data for the monoester of DIDP (monoisodecyl phthalate, MIDP) that showed an average half-life of 25 hours in marine sediments under aerobic conditions based on results from two studies (Otton et al., 2008). Research suggests that the formation of the monoester occurs as the first step in the biotic degradation of DIDP (Staples et al., 1997b). Because this step does not appear to be rate limiting, as evidenced by the high extent of biodegradation demonstrated by DIDP in a ready test, the aerobic degradation of the diester in sediment is expected to occur at a similar rate.

Studies are not available to assess the biodegradability of DIDP specifically in soil. However, data for an analog substance di-isononyl phthalate (CAS No. 68515-48-0; DINP) can be used to estimate the loss rate of DIDP in soil. DINP exhibited a half-life in soil of approximately 51 days, based on the loss of parent substance (ExxonMobil Biomedical Sciences, Inc., 2009). These data were developed in an earthworm toxicity test conducted in soil in which the concentration of DINP was monitored over a 56-day period. During that period DINP concentration decreased from 982 to 441 mg/kg soil (wet weight). Because DIDP and DINP exhibited similar extents of biodegradation in ready biodegradability tests, 67 and 71% respectively, they would be expected to biodegrade in soil at similar rates and to similar extents.

### **Bioaccumulation – Section 5.3**

DIDP has a low potential to bioaccumulate in the environment based on results from a biomagnification food-web field study, a bioaccumulation dietary lab study with a fish, a calculated bioconcentration factor value, and a measured biota-soil accumulation factor (BSAF) for a soil-dwelling invertebrate.

**Biomagnification:** High molecular weight phthalate esters (di-C8 PEs to di-C10 PEs) have been shown not to biomagnify through the food web, but rather decrease in tissue concentration with increasing trophic position. The results of a study to assess the bioaccumulation of high molecular weight phthalate diesters in an aquatic food-web that included 18 marine species, showed that DIDP did not biomagnify, but rather decreased in tissue concentration in organisms of increasing trophic position. Decreasing concentrations, also referred to as biodilution, can be quantified by food-web magnification factors (FWMFs). A FWMF that is greater than 1.0 is an indication of chemical biomagnification within a food-web, whereas a value of less than 1.0 indicates biodilution or dilution from lower to higher trophic levels. Study results showed that lipid equivalent concentrations of the high molecular weight phthalate diesters significantly declined with increasing trophic level and that the FWMF for diisodecyl phthalate was 0.44.

Bioaccumulation: The finding above is consistent with a laboratory fish bioaccumulation study in which rainbow trout were fed a DIDP spiked diet for 14 days. The low water solubility and high  $K_{ow}$  of DIDP prevent conducting an aqueous exposure BCF (bioconcentration factor) study. At the end of the exposure period, fish were sampled after different depuration times (0, 0.6, 1, 3 days). Results demonstrated limited bioaccumulation with a lipid normalized biomagnification factor (BMF, concentration ratio in tissue to that in diet) of 0.1 and rapid subsequent depuration with a tissue elimination half-life of 1 day. The half-life of 1 day was used to calculate a BCF in fish of 1 L/kg for DIDP.

Bioconcentration: Calculated BCF data for di-isodecyl phthalate (DIDP) suggest that it has a low potential to bioconcentrate in the aquatic environment.

BSAF: Data to assess the potential for terrestrial bioaccumulation of DIDP were reported in a 14-day earthworm (*Eisenia fetida*) toxicity study. The biota-soil accumulation factor (BSAF) as measured in a natural soil was 0.015 based on a DIDP concentration in the earthworm of 120 mg/kg (wet weight) and in soil of 7829 mg/kg (dry weight). A BSAF value of <1 indicates a lack of bioaccumulation.

### **Aquatic Toxicity – Section 6.1**

The data used to characterize the acute and chronic aquatic toxicity of di-isodecyl phthalate ester (DIDP; CAS #68515-49-1) are consistent with the data for several high molecular weight phthalate diesters summarized by Staples et al. (1997). These data show that high molecular weight phthalate diesters do not produce acute or chronic aquatic toxicity at or below their maximum attainable water solubility to fish, invertebrates, and algae.

#### **Short-term Toxicity to Fish – Section 6.1.1**

The data used to characterize the fish acute toxicity of di-isodecyl phthalate ester (DIDP; CAS #68515-49-1) ester are consistent with the data for several high molecular weight phthalate diesters summarized by Staples et al. (1997). These data show that high molecular weight phthalate diesters do not produce acute toxicity to fish at or below their maximum attainable water solubility. The acute fish dataset includes results for various species of freshwater fish including *Oncorhynchus mykiss*, *Pimephales promelas*, and *Lepomis macrochirus*, and a marine fish, *Cyprinodon variegatus*.

#### **Long-term Toxicity to Fish – Section 6.1.2**

The data used to characterize the fish chronic toxicity of di-isodecyl phthalate ester (DIDP; CAS #68515-49-1) are consistent with the data for several high molecular weight phthalate diesters summarized by Staples et al. (1997). These data show that high molecular weight phthalate diesters do not produce chronic toxicity to fish at or below their maximum attainable water solubility. The chronic fish dataset includes a result for the freshwater fish, *Oryzias latipes*.

#### **Short-term Toxicity to Invertebrates – Section 6.1.3**

The data used to characterize the invertebrate acute toxicity of di-isodecyl phthalate ester (DIDP; CAS #68515-49-1) are consistent with the data for several high molecular weight phthalate diesters summarized by Staples et al. (1997). These data show that high molecular weight phthalate diesters do not produce acute toxicity to invertebrates at or below their maximum attainable water solubility. The acute invertebrate dataset includes several results for three species, *Daphnia magna*, *Paratanytarsus parthenogenetica*, and *Mysidopsis bahia* (new name: *Americamysis bahia*).

#### **Long-term Toxicity to Invertebrates – Section 6.1.4**

The data used to characterize the invertebrate chronic toxicity of di-isodecyl phthalate ester (DIDP; CAS #68515-49-1) ester are consistent with the data for several high molecular weight phthalate diesters summarized by Staples et al. (1997). These data show that high molecular weight phthalate diesters do not produce chronic toxicity to invertebrates at or below their maximum attainable water solubility. The acute invertebrate dataset includes results for one species, *Daphnia magna*.

#### **Toxicity to Aquatic Algae and Cyanobacteria – Section 6.1.5**

The data used to characterize the algal toxicity of di-isodecyl phthalate ester (DIDP; CAS #68515-49-1) ester are consistent with the data for several high molecular weight phthalate diesters summarized by Staples et al. (1997). These data show that high molecular weight phthalate diesters do not produce toxicity to algae at or below their maximum attainable water solubility. The algal dataset includes results for one species, *Selenastrum capricornutum* (new name: *Pseudokirchnerella subcapitata*).

#### **Toxicity to Microorganisms – Section 6.1.7**

The data used to characterize the microbial toxicity of di-isodecyl phthalate ester (DIDP) are consistent with the data for other high molecular weight phthalate diesters summarized by Staples et al. (1997). These data show that high molecular weight phthalate diesters do not produce toxicity to microorganisms at or below their maximum attainable water solubility. The microbial dataset for DIDP includes results for one species, *Photobacterium phosphoreum*, and a population of bacteria from a wastewater treatment plant.

#### **Sediment Toxicity – Section 6.2**

DINP has been tested in sediment toxicity test in concentrations up to 3000 mg/kg. No adverse effects had been observed with two sediment dwelling organisms.

#### **Terrestrial Toxicity – Section 6.3**

The data used to characterize the sediment toxicity of di-isodecyl phthalate ester (DIDP; CAS #68515-49-1) are consistent with the data for several high molecular weight phthalate diesters summarized by Call et al. (2001). These data show that high molecular weight phthalate diesters do not produce toxicity to sediment dwellers at high sediment loading rates. The sediment invertebrate dataset includes results for three species, *Chironomus tentans*, *Hyalella azteca*, and *Rana arvalis*.

#### **Toxicity to Soil Macroorganisms Except Arthropods – Section 6.3.1**

The toxicity of di-isodecyl phthalate ester (DIDP) as measured by mortality to the earthworm (*Eisenia fetida*) was evaluated in a 14-day study using natural and artificial soils. The toxicity of di-isononyl phthalate ester (DINP), an analog to DIDP, as measured by reproduction to the earthworm (*Eisenia fetida*) was evaluated in a 56 day study using an artificial soil. No significant mortality was observed in natural and artificial soils dosed with DIDP after 14 days at a nominal loading rate of 10,000 mg/kg soil (dw), which measured 7,664 mg/kg natural soil (dw) and 8,435 mg/kg artificial soil (dw) (concentrations are from analyses of soils at test initiation). DINP did not effect earthworm (*Eisenia fetida*) reproduction, based on a 56-day limit study in artificial soil at a high measured concentration of

982.4 mg/kg soil (dw). Because DINP and DIDP are structurally similar and exhibit the same biological activity, these data support the conclusion that DIDP would also not effect earthworm reproduction.

### **Toxicity to Terrestrial Arthropods– Section 6.3.2**

In accordance with REACH Chapter R.7C Endpoint Specific Guidance, specifically R.7.11.6.3 Testing Strategy (Table R.7.11-2), data to characterize toxicity to terrestrial arthropods is waived for the following reasons. Di-isodecyl phthalate ester (DIDP) is readily biodegradable, consequently it is considered to degrade rapidly in the environment and not persist. DIDP does not cause acute or chronic aquatic toxicity at its maximum water solubility, consequently it does not pose an acute or chronic aquatic hazard, and it is not possible to derive NOEC or PNEC values needed for quantitative risk assessment. However, it is possible to qualitatively conclude based on low solubility and available effects test data that DIDP is not harmful to aquatic organisms. Acute and chronic toxicity data for soil macroorganisms, earthworms, also show that DIDP does not cause effects at high soil loading rates. Therefore, based on these considerations, additional short and long-term toxicity testing for soil organisms is not needed.

### **Toxicity to Terrestrial Plants – Section 6.3.3**

The data used to characterize the terrestrial plant toxicity of di-isodecyl phthalate ester (DIDP; CAS #68515-49-1) ester show that DIDP does not cause toxicity to terrestrial plants at a high soil loading level, 8630 mg/kg, based on germination. The terrestrial plant toxicity dataset includes results for two species, Lolium species and Lactuca sativa.

Studies are not available to assess the chronic toxicity of DIDP to plants. However, DIDP has been shown to be readily biodegradable, which suggests it will be rapidly degraded in the environment, and applications do not directly apply DIDP to soil. As acute studies in plants show no effects at high soil loading levels and acute and chronic studies with other terrestrial organisms also show no effects at the highest soil loading levels tested, chronic effects to terrestrial plants are not expected. Additionally, guidelines on information requirements state that testing for soil organisms is not needed if a substance is readily biodegradable and is not directly applied to soil.

### **Toxicity to Soil Microorganisms – Section 6.3.4**

In accordance with REACH Chapter R.7C Endpoint Specific Guidance, specifically R.7.11.6.3 Testing Strategy (Table R.7.11-2), data to characterize toxicity to soil microorganisms is waived for the following reasons. Di-isodecyl phthalate ester (DIDP) is readily biodegradable, consequently it is considered to degrade rapidly in the environment and not persist. DIDP does not cause acute or chronic aquatic toxicity at its maximum water solubility, consequently it does not pose an acute or chronic aquatic hazard, and it is not possible to derive NOEC or PNEC values needed for quantitative risk assessment. However, it is possible to qualitatively conclude based on low solubility and available effects test data that DIDP is not harmful to aquatic organisms. Acute and chronic toxicity data for soil macroorganisms, earthworms, also show that DIDP does not cause effects at high soil loading rates. Data also show that DIDP does not inhibit respiration in wastewater treatment microorganisms at levels above its water solubility. Therefore, based on these considerations, additional short and long-term toxicity testing for soil microorganisms is not needed.

### **Toxicity to Birds – Section 6.3.5**

In accordance with ECHA (2008) Guidance on information requirements and chemical safety assessment, Chapter R.7c: Endpoint Specific Guidance, the long-term or reproductive toxicity to birds study does not need to be conducted as this substance is unlikely to pose a secondary poisoning risk and has been shown to be readily biodegradable and to have a low bioaccumulative potential (fish BCF below 100 and no indications of bioaccumulation in mammalian tests). The extensive mammalian toxicity database for di-isodecyl phthalate ester (DIDP) demonstrates a lack of acute and chronic effects to vertebrates. Further, DIDP is not persistent in the environment, demonstrates a low bioaccumulative potential, and is not toxic to aquatic and non vertebrate terrestrial organisms and plants. The sum total of these data establish a weight of evidence that strongly supports the conclusion that DIDP will not produce acute or chronic effects in birds, thereby eliminating the need to conduct testing for this endpoint.