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Flexible PVC Recycling Workplace Exposure and Environmental Emissions

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Version 1.0

EXECUTIVE SUMMARY

This report evaluates occupational exposure and environmental emissions associated with flexible PVC recycling operations across two European recycling facilities. The study was commissioned in response to concerns raised by the European Chemicals Agency (ECHA) regarding potential worker exposure to organotin stabilisers and plasticisers, as well as the environmental release of PVC particulates during recycling operations. The assessment was conducted by Plastics Recyclers Europe (PRE) and VinylPlus through on-site exposure monitoring, elemental dust analysis, and review of plant emission data.

The methodology included personal air sampling of workers in representative "worst-case" exposure roles, measuring inhalable and respirable dust in accordance with recognised occupational hygiene standards. Dust samples were further analysed for elemental content including tin (Sn), lead (Pb), cadmium (Cd), antimony (Sn), zinc (Zn), calcium (Ca), and titanium (Ti). For the quantification of plasticiser exposure urinary biomonitoring was performed.

The results show that across all monitored facilities, worker exposure to inhalable and respirable dust remained below limits set in this study at 5 mg/m^3 and 1 mg/m^3 respectively based on an overview of National Occupational Exposure Limits (OELs), indicating general dust exposure is well controlled. Similarly, lead and cadmium exposures remained well below the EU binding OELs and Antimony and Zinc, exposure remains below limits based on national OELs. Furthermore, based on an extreme worst-case calculation assuming all measured tin is caused by a single organotin substance, it can be concluded that there is no unacceptable risk for human health in relation to organotins.

Table 1 Summary of measured airborne concentrations in the breathing zone of 19 workers across 4 EU Recycling facilities and the risk assessment based thereupon.

Substance	Inhalable Dust (mg/m^3)	Respirable Dust (mg/m^3)	Pb (µg/m^3)	Cd (µg/m^3)	Sb (µg/m^3)	Zn (µg/m^3)	Sn (µg/m^3)	Max DOTE (µg/m^3)
Median	0.4	0.09	0.7	0.02	0.03	2.62	0.12	0.8
90th Percentile	1.3	0.15	1.1	0.23	0.27	10.74	0.46	2.9
Reference max exposure level	5	1	30	1	250	500	-	25
RCR	0.26	0.15	0.037	0.230	0.001	0.021	-	0.116

Estimated Daily Intakes of individual plasticiser, calculated based on metabolites found in urine samples of workers before and after their shift, were below tolerable daily intakes values determined by a review of their toxicology. Even in a very conservative mixture risk assessment approach for anti-androgenic effect, the risk characterisation ratios remained below 1 indicating that workers operate safely.

All activities in participating plants occurred under channelled extraction ventilation, which has been showed in rigid PVC recycling to limit release of particulate matter including microplastics to level of around 0.0007-0.0021%. As such the release of microplastics is strictly limited.

Overall, the report concludes that modern rigid PVC recycling facilities operating under current European best practices present a low occupational and environmental risk profile. Existing engineering controls, ventilation systems, material containment procedures, and operator awareness measures appear highly effective in controlling worker exposure and limiting environmental emissions.

CONTENTS

INTRODUCTION

The European Chemicals Agency's (ECHA) Investigation Report on PVC and its Additives (Appendix A+B, C, D, E, F) raises concerns about 1) worker exposure to organotin substances in PVC recycling facilities, 2) environmental release of PVC particles containing additives from recycling facilities, and 3) worker exposure to plasticisers in flexible PVC recycling facilities (ECHA 2023).

Plastics Recyclers Europe (PRE) and VinylPlus (V+) have agreed on a project to address these concerns. The core idea is to 1) perform workplace exposure measurements for inhalable/respirable dust and subsequent analysis of dust for tin and other elements, 2) collect existing emission measurement data, and 3) perform biomonitoring for plasticiser exposure (flexible PVC only). The outcome of the project will be two reports: this one focussing on findings in flexible PVC recycling and another one focussing on rigid PVC recycling.

METHODS

PLANT SELECTION AND RECRUITMENT

Potential volunteer plants were contacted and provided a two-page briefing describing the project and what would be expected of them. It was expected of the plants to provide currently existing information on workplace exposure, operational conditions and existing risk management measures, and to provide emission measurement reports. Subsequently a virtual meeting was held with plants that expressed an interest to agree on a sampling plan for the plant.

During the virtual meetings, the target was to include 4 workers per plant in the measurement campaign and select the most representative worst case exposure positions. While this may appear to be relatively low number of workers, there were several instances where this proved to be the complete workforce operating the plant per shift. This number of 4 workers was occasionally deviated from in the event that there was a good reason for inclusion of more workers based on the activities performed by the workers.

EXPOSURE MEASUREMENTS

All workers were informed by plant management prior to the execution of the measurement campaign and were explicitly informed that they were under no obligation to participate. Not participating would also not have any negative consequences for their employment.

Qualified PRE Staff travelled to the participating plants to carry out the sampling. The calibrated equipment was rented from RPS Analytics which also carried out the analysis of the collected samples.

Inhalable & Respirable Dust

Each worker included in the study was equipped with two sampling pumps. One fitted with an IOM filter for the collection of inhalable dust, and another fitted with a cyclone filter for respirable dust. These measurements were carried out according to MDHS 14/4 *General methods for sampling and gravimetric analysis of respirable, thoracic and inhalable aerosols*.

Elemental Analysis

The following elements in the inhalable dust fraction Sn, Pb, Cd, Ti, Sb, Zn, Ca were quantified by ICP-MS performed according to ISO 30011 *Workplace air — Determination of metals and metalloids in airborne particulate matter by inductively coupled plasma mass spectrometry*.

Urine Sample Collection

Workers were asked to provide a urine sample in a urine cup with transfer unit before and after their shift. On site each cup sample was used to fill two 9 ml vacuum tubes, one was sent for analysis and the other one was stored refrigerated until the receipt of the samples was confirmed. All samples were sent for plasticiser analysis to the Institute for Prevention and Occupational Medicine of the German Social Accident Insurance is an institute of the Ruhr University Bochum (IPA).

COLLECTION OF PLANT CONTEXTUAL INFORMATION

PRE Staff collected information on the plants' operational conditions and risk management measures during the visit. Photographs and video were taken during the visit for inclusion in this report. Before inclusion into the report photos were edited in the following ways:

1. Individuals were made unrecognisable in order to protect their identity
2. Identifying markings (e.g. logos) including text in the local language were blurred
3. Any proprietary machinery or processes were cropped or blurred.

Plants were given a period to evaluate the processing and highlight additional aspects that they would not like to have included in the report that would reveal trade secrets.

RISK ASSESSMENT HUMAN HEALTH

Dust

At the moment there is no EU Binding Occupational Exposure Limit (OEL) for dust. However, there is a patchwork of national limit values and practices[1] that places limits on dust exposure in the occupational health and safety sphere. The GESTIS International Limit Value Database was consulted to see what kind of limits have been adopted at national level.

For inhalable dust, several countries (AT, BE, DK, DE (AGS), HU, IE, PL, ES, SE, CH) maintain a time weighted average limit of 10 mg/m^3, while a more limited number of countries (FR, DE (DFG)) maintain a limit of 4 mg/m^3.

For respirable dust, HU has a limit of 6 mg/m^3, AT and US (OSHA) maintains a limit of 5 mg/m^3, IE limits exposure to 4 mg/m^3, BE, ES, and CH set the limit at 3 mg/m^3, DE has a limit of 1.25 mg/m^3 (AGS) and a limit of 0.3 mg/m^3 (DFG), and finally FR has a 0.9 mg/m^3 limit.

For the purpose of this report a level of 5 mg/m^3 for inhalable dust and a level of 1 mg/m^3 for respirable dust will be considered safe. This is somewhat an arbitrary choice roughly based on the lower side of the spectrum of what Member States have adopted.

[1] For example, NL does not have a limit value on dust, but does maintain that in a workplace risk assessment employers must for those substances without a national limit look abroad to find a limit value.

Lead and Cadmium

There are EU Binding Occupational Exposure Limit values for lead and cadmium in the Carcinogens, Mutagens and Reprotoxic substances Directive (CMRD), which are 30 and 1 µg/m³, respectively. These are considered appropriate for this report and airborne concentrations of these elements will be compared with these limit values.

Organotins

ECHA in its investigation report indicated that 10 organotin substances were possible to be used as heat stabilisers in PVC; of which 5 or 6 may be in use in the EU (Table 2, with presumably used substances in bold).

In reality, due to the production process of organotins, it is not normally the case that commercial substances are purely one or the other molecule (Graf 2000). For example, DOTE is produced by first reacting tin tetrachloride and certain organometallic compounds to produce tetraoctyltin. Tetraoctyltin can then be reacted with an equal part tin tetrachloride to produce **mainly** dioctyltin dichloride, with an impurity of monooctyltin trichloride and trioctyltin chloride. This isomeric mixture is then reacted with isooctyl mercaptoacetate to produce **mainly** dioctyltin bis(2-ethylhexyl thioglycolate), with some monooctyltin tris(2-ethylhexyl thioglycolate) (MOTE) and trioctyltin 2-ethylhexyl thioglycolate.

Given the adverse properties of DOTE, years ago a switch has been made to MOTE. Unfortunately, while the chemical reaction to produce trioctyltin chloride and dioctyltin dichloride from tetraoxtyltin and tin tetrachloride proceeds smoothly, the production of monooctyltin trichloride is more challenging and results in somewhat more substantial impurity of dioctyltin dichloride. As such in the subsequent step with isooctyl mercaptoacetate, DOTE becomes a substantial impurity in MOTE. A serious effort has been made over the past years that has resulted in the possibility of to supply MOTE with ever lesser concentrations of DOTE.

For those substances with human health hazard classifications (DOTE, DMTE and MMTE) ECHA performed a quantitative risk assessment comparing exposure highly conservative exposure modelling results with Derived No Effect Levels (DNELs) for these substances.

The quantification of DOTE, DMTE and MMTE in inhalable/respirable dust is difficult[2] and considerably more laborious and thus expensive than the quantification of the elemental tin content. Furthermore, while it is possible to first weigh the inhalable fraction and subsequently analyse it with ICP-MS to determine the elemental composition, it is not possible to weigh the inhalable fraction **and** perform ICP-MS **and** quantify DOTE, DMTE, and MMTE. Direct analysis of these substances would require an additional pump and sample on the worker that is already having to tolerate the presence of two such pieces of equipment.

A more efficient approach would be to calculate extreme worst-case concentrations of DOTE, DMTE and MMTE, assuming that all airborne tin is caused by a single one of these substances. For example, if 1 µg/m³ of tin is measured in the breathing zone of a worker in a recycling facility and it is assumed that this 1 µg/m³ is caused fully by DOTE with a tin content of 15.8% then the worst-case maximum concentration of DOTE is 1 [µg Sn/m³] / 0.158 [Sn/DOTE] = 6.3 µg DOTE/m³.

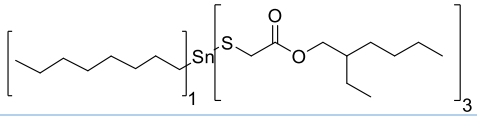
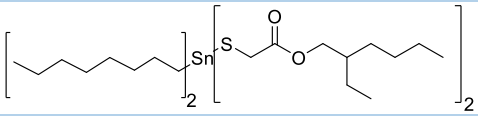
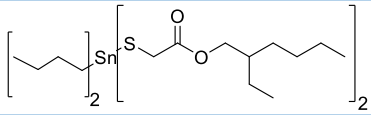
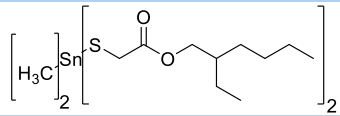
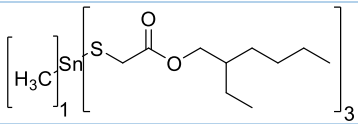
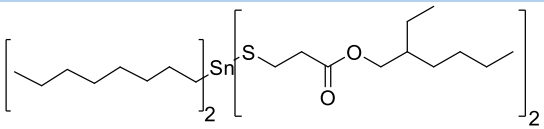
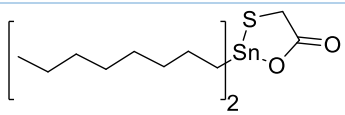
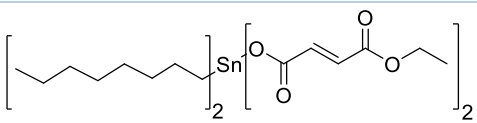
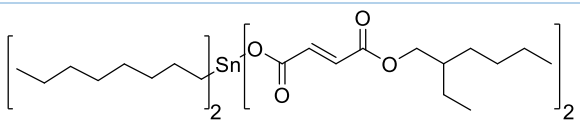
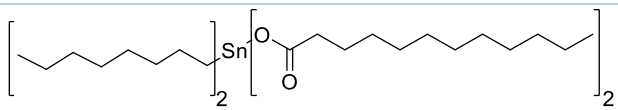
[2] Typically, the direct carbon-Sn bond is very much covalent, while the O-Sn or S-Sn is somewhat ionic. When these kinds of molecules are used in polymers, the carbon-Sn bond maintains cohesion while the O-Sn bonds may dissociate resulting in an organotin ion such as dibutyltin^{2+} and an anion such as in this example two dodecanoate $CH3–[CH2]_{10}–COO^{1-}$. One would have to quantify the organotin ion and the anions within the material separately and make assumptions on their association.

If these extreme worst-case maximum concentrations are below the limit values for these substances, then the worker is operating safely. If the worst-case maximum concentrations exceeds the limit value, it is not proof that the worker is operating in concentrations above the limit value, but would require further follow-up specific measurements to determine the exact concentration of the substance in question. In the example above the DNEL for worker inhalation exposure is 25 µg DOTE/m³ and it can be concluded that the worker is operating safely.

The ECHA PVC Investigation Report indicates that a limit value of 25, 180, and 5750 µg/m³ are applicable to DOTE, DMTE, and MMTE. These limit values will be used to evaluate the maximum DOTE, DMTE, and MMTE concentrations to calculate maximum RCRs for risk assessment.

The conservativeness of this approach should not be underestimated. In Europe around 8600 tons of organotins are used, with the majority of this (5900 tons) in short life cycle applications such as packaging (films). The remaining volume is used in building and construction (B&C), automotive, and medical applications (blister packs). The use of organotins in construction profiles was/is a practice that is mainly restricted to the US, while in the EU cadmium, then lead, and now calcium/zinc stabilisers are used. The use of DOTE itself has been largely phased out in the EU over the past years and the impurity of DOTE in MOTE has seen a decline over the same period as well. Furthermore, what little organotin was used in B&C was mainly methyltin- rather than octyltin-based. As such RCRs calculated based on the maximum airborne DOTE concentration will be result in a large overestimation of risk.

Table 2 Structures, name, abbreviation, EC/CAS number, registered tonnage, and uses of organotin molecules.

Structure	Name	Abbreviation	EC / CAS	Aggregated Tonnage (active registrants)	
	**Monooctyltin bis(2-ethylhexyl thioglycolate)**	**MOTE**	**248-227-6 27107-89-7**	**1000-10000 (4)**	**Pipe fittings, Packaging (food and non-food), Automotive parts, Medical packaging (blister packs)**
	**Dioctyltin (2-ethylhexyl thioglycolate)**	**DOTE**	**239-622-4 15571-58-1**	**1000-10000 (5)**	**Pipe fittings, Packaging (food and non-food), Automotive parts, Medical packaging (blister packs)**
	Dibutyltin bis(2-ethylhexyl thioglycolate)	DBTE	234-186-1 10584-98-2	10-100 (4)	No identified uses
	**Dimethyl bis(2-ethylhexyl thioglycolate)**	**DMTE**	**260-829-0 57583-35-4**	**1000-10000 (4)**	**Pipe fittings, Packaging (food and non-food), Automotive parts, Medical packaging (blister packs)**
	**Monomethyl tris(2-ethylhexyl thioglycolate)**	**MMTE**	**260-828-5 57583-34-3**	**1000-10000 (12)**	**Pipe fittings, Packaging (food and non-food), Automotive parts, Medical packaging (blister packs)**
	**Dioctyltin bis(2-ethylhexyl mercaptopropionate)**	**DOT-MalEt**	**261-645-3 59185-95-4**	**10-100 (1)**	**No identified uses**
	**2,2-dioctyl-1,3,2-Oxathiastannolan-5-one**	**DOTTG**	**239-581-2 15535-79-2**	**Ceased Manufacture**	**Pipe fittings**
	Dioctyltin bis(ethyl maleate)		268-500-3 68109-88-6	100-1000 (3)	Pipe fittings
	Dioctyltin bis(ethylhexyl maleate)		233-117-2 10039-33-5	10-100 (2)	No identified uses
	Dioctyltin dilaurate	DODL	222-883-3 3648-18-8	100-1000 (6)	No identified uses

Antimony, Calcium, Zinc, and Titanium

Antimony trioxide if added to PVC formulations would potentiate the inherent flame retardancy of the PVC material (Schiller 2015; Weil and Levchik 2009). This is normally not needed and thus not done in rigid PVC formulations. However, in flexible PVC formulations, where the addition of plasticiser reduces the flame retardancy of the material, antimony trioxide may be used. As the marginal cost of inclusion of antimony under the ICP-MS method was zero, it was decided to quantify antimony in inhalable dust also for rigid PVC.

The GESTIS International Limit Database reveals that only Finland adopted a limit value specific to Antimony dioxide of 0.5 mg/m³. Two European countries adopted a limit value for "Antimony and compounds, except antimony trisulphide, antimony trioxide and antimony hydride": AT and NL at 0.5 mg/m³. Finally, 16 countries have set a limit for "Antimony and its Antimony compounds (except stibine)" as Sb at: 0.5 mg/m³ in AT, BE, DK, FI, FR, HU, IE, NO, PL, ES, CH, NL, and UK; 0.25 mg/m³ in SE; 0.2 mg/m³ in LV and RO. For the purpose of this report, it is assumed that a limit of 0.25 mg Sb/m³ would be protective of human health.

Calcium in PVC formulations can be present because of two different types of additives used: calcium(-zinc) stabiliser packs and calcium carbonate (Schiller 2015). Calcium carbonate (i.e. limestone) is a filler additive used in a wide variety of plastics (and other materials). The GESTIS International Limit Database, reveals that limits have been adopted for calcium carbonate in 7 European countries: 10 mg/m³ in FR, HU, IE (inhalable fraction), PL, and UK (inhalable fraction); 6 mg/m³ in LV; 4 mg/m³ in IE (respirable fraction) and UK (respirable fraction), and 3 mg/m³ in CH. Given the similarity of these limits with the general dust limits, it seems that these limits for calcium carbonate might be set to control dust exposure and not because of inherent toxicological properties of the substance. Next to this Calcium stabilisers tend to be calcium fatty acid salts which are not of any toxicological relevance. As such no quantitative risk assessment for calcium exposure is performed.

Zinc exposure can be due to the use of zinc stabilisers or zinc oxide in PVC (Schwab et al. 2015; Schiller 2015). Zinc oxide is a UV stabiliser additive used in thermoplastics and zinc stabilisers are normally zinc fatty acid salts used in PVC formulations exclusively. However, the main use of zinc oxide, in general, is in the rubber industry where it plays a role in the vulcanisation process and exposure to zinc in the mechanical processing of PVC wastes containing rubber may contribute to zinc exposure.

The GESTIS International Limit Database indicates that there are limits in several European countries for either: "Zinc oxide, dust", "Zinc oxide", or "Zinc and its compounds, inorganic, inhalable aerosol" set at: 10 mg/m³ in FR, NO (inhalable), and ES; 5 mg/m³ in HU, NO (respirable), PL, and SE; 3 mg/m³ in CH, 2 mg/m³ in BE (respirable), FI, DE (DFG); and 0.5 mg/m³ in LV. Again, similar to, but perhaps slightly lower than, for regular dust. For the purpose of this report, it is assumed a limit of 0.5 mg Zn/m³ is protective of human health.

Finally, titanium is an indicator of the presence of titanium dioxide (Auer et al. 2017; Schiller 2015). Titanium dioxide reflects electromagnetic radiation that has roughly double the wavelength of size of the particles. As such TiO2 particles with a size of 190 – 390 nm reflect visible light and pigmentary TiO2 tends to have a particle size distribution that is designed to match. There are also grades of nano-TiO2, with much smaller particle size distributions, however this is primarily used in sunscreens to protect against UV radiation (which has a smaller wavelength than visible light). In PVC formulations only pigmentary TiO2 has been used and while such pigmentary TiO2 will have a particle size distribution that has a tail extending in the sub 100 nm range, this is but a very small fraction of the pigmentary grade. As such specific recommendations regarding the nanoform of titanium dioxide such as the limit of 0.8 µg/m³ proposed by ANSES do not apply here (ANSES 2020).

The GESTIS International Limit Database states that there are limits in European Countries for "Titanium dioxide": 11 mg/m³ in FR; 10 mg/m³ in BE, IE (inhalable), LV, PL, RO, ES, and UK (inhalable); 6 mg/m³ in DK; 5 mg/m³ in AT (respirable), NO, and SE; 4 mg/m³ in IE (respirable) and UK (respirable); and 3 mg/m³ in CH (respirable). Germany (DFG) has a limit of 0.3 mg/m³ for the respirable fraction, that must be multiplied by the material density. In general, the toxicity of TiO2 does not seem to be very much related to the substance, but rather to the particulate nature of the material (RAC 2017; Heinrich et al. 1995). As such, any risk is covered by the risk assessment for dust and no specific risk assessments for titanium dioxide exposure is performed.

For antimony and zinc there will be a risk characterisation included in the plant reports. The data for calcium and titanium will however be available along with all other raw data in the annex I.

Plasticisers

41 plasticiser metabolites from 20 plasticisers were quantified with a multimethod developed by IPA in previous work (Kasper-Sonnenberg et al. 2025) for general population samples. IPA also performed standard creatinine quantification. Raw data can be found in Annex I Table 28.

Estimations of the daily intake (EDI) in µg per kg (b.w.) per day were performed for each plasticizer based on the respective urinary metabolite concentrations (UC), the product with a standardised urinary excretion rate for male and females (Koch et al. 2003) and multiplied by the ratio between the molar mass of the parent compound and the metabolites, then divided by the product of the urinary excretion factor (Fue) and urinary creatinine concentration (see equations (1) and (2)). Collated experimentally derived and estimated Fue were used as described elsewhere (Kasper-Sonnenberg et al. 2025).

$$DI = \frac{UC_m \times CE \times M_p}{F_{UE} \times UC_{cr} \times M_m} \quad (1)$$

	Name	Value	Unit	Source
DI	Estimated Daily Intake		µg/kg bw/d	Calculated
UC_m	Urinary Concentration of Metabolite		µg/L	Measured
CE	Creatinine Excretion Rate	Male: 23 Female: 18	mg CR/kg bw/d	(Koch et al. 2003)
M_m	Molecular Weight of Metabolite		Da	
M_p	Molecular Weight of Parent Compound		Da	
F_{UE}	Urinary Excretion Factor		-	(Kasper-Sonnenberg et al. 2025)
UC_{cr}	Urinary Concentration of Creatinine		mg CR/L	Measured

Where more than one biomarker with known and appropriate Fue's was available, the EDIs were calculated for the sum of their respective metabolites (DEHP, DiNP, DiDP, DINCH) as shown in equation (2).

$$DI = \left(\frac{UC_{m1}}{M_{m1}} + \frac{UC_{m2}}{M_{m2}} + \frac{UC_{m3}}{M_{m3}} + \frac{UC_{m4}}{M_{m4}}\right) \times \frac{CE \times M_p}{UC_{cr} \times \sum F_{UE}} \quad (2)$$

For Dicyclohexyl phthalate (DCHP) and Dioctyl phthalate (DnOP) no Fue's were available and no EDI's were calculated[3]. For all others the EDI values can be found in annex I Table 29. The EDI values were divided by toxicological benchmarks obtained as described below to obtain risk characterisation ratios (RCRs). The calculated RCRs can be found in annex I Table 30.

For Dipropyl phthalate (DnPrP; cas: 131-16-8), Bis(2-methoxyethyl) phthalate (DMoxyEP; cas: 117–82-8), Diisopentyl phthalate (DiPeP; cas: 605–50-5), Dipentyl phthalate (DnPeP; cas: 131–18-0), Dihexyl phthalate (DnHexP, cas: 84–75-3), Diisohexyl phthalate (DiHepP; 71888–89-6), and Diheptyl phthalate (DnHepP; cas: 3648–21-3) no toxicological benchmark was obtained and thus no risk characterisation ratio was calculated. None of these substances are REACH Registered meaning that they are not produced or imported into the EU by any single operator in volumes exceeding 1 ton per year. Also, outside of the EU, there is little to no indication that these substances are actually produced and used in any relevant quantities beyond the production of analytical standards and volumes of scientific (toxicological) research.

The lack of commercial relevance of these substances is also reflected in the biomonitoring results. No detectable metabolite concentrations present for DnPrP and DMoxyEP. For DiPeP there was only one sample with a detectable concentration of its metabolite MiPeP that only slightly exceeded the limit of quantification. Only one of the three metabolites of DnPeP was detected (4cx-MnBP), this is however also postulated to be a secondary metabolite resulting of beta-oxidation of higher molecular weight phthalate metabolites (Kasper-Sonnenberg et al. 2025). Similarly, metabolites of DnHexP were detected in urine, however the main metabolite MnHexP was only detected in two samples at concentrations only slightly exceeding the limit of detection (0.3 and 0.8 µg/L) and the alternative metabolite 5cx-MnPeP was detected at a higher frequency and concentrations (n=17/ 22; range: 0.3 – 2.8 µg/L). The latter metabolite is however also postulated to be a secondary beta-oxidation product of higher molecular weight plasticisers.

Support for this hypothesis can be found in the data collected in this study by performing regression analysis of the DiNP metabolite concentrations with the postulated secondary beta-oxidation metabolites. This regression shows a very clear association (Figure 1). This is relevant from a practical perspective as it makes linear carboxylate metabolites less suitable for the estimation of daily intake.

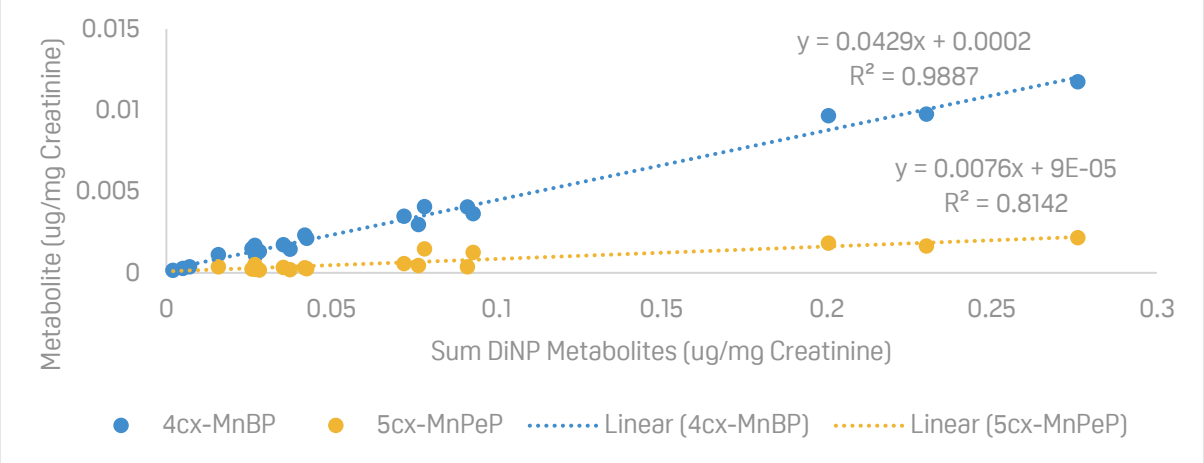


Figure 1 regression analysis of sum DiNP Metabolite concentrations in µg/mg creatinine vs the postulated secondary beta oxidation metabolites 4cx-MnBP and 5cx-MnPeP. Datapoints below limit of detection were excluded from this analysis.

[3] This is not necessarily an issue as the metabolites of these were not detected in urine with the rather low LoD of 0.2 µg/L; likely due to their very limited use. DnOP is not REACH Registered and DCHP only has 2 active registrants.

Dimethyl Phthalate (DMP)

The substance DMP (cas: 131–11-3) is normally not used as a plasticiser in flexible PVC formulations, but it is a solvent used in many industries[4] and can be found in cosmetics and fragrances. Should the study demonstrate elevated levels of this substance in workers, this is more likely due to exposure outside of the workplace (e.g., due to use of cosmetics products (e.g. hand lotion) in the workplace).

A brief search on the ECHA website and in the EFSA journal did not reveal any official authority reports on DMP in which a toxicological benchmark was established. The lead registration dossier however did contain sufficient information to conclude on its toxicity.

The substance does not appear to be mutagenic/genotoxic based on a number of in vitro and in vivo studies. In addition, a 1-year dermal carcinogenicity study in mice did not result in tumorigenesis. Reproductive toxicity was extensively evaluated based on studies on DMP and with the aid of read across from the close structural analogue DEP, based on which it is fair to conclude that DMP does not display antiandrogenic effect, no effect on fertility and development.

In terms of repeated dose toxicity, a recent (2023) study report of an OECD 422: Combined Repeated Dose Toxicity Study with the Reproduction/Developmental Toxicity Screening Test is available in which Wistar Hannover rats received in their diet 1500, 5000, and 15000 ppm of DMP. This revealed no treatment related changes in any of the dosed groups establishing a NOAEL of 15000 ppm (1007 mg/kg bw/day) for males and 1595 mg/kg/day for females). This lines up relatively well with other studies such as a 1955 study where female rates were fed with 2, 4, and 8% DMP for two years, which revealed slight effects on growth at the 8 and 4% dosing rate along with kidney damage at the 8% rate. This study established a NOAEL at 2% which is about 1000 mg/kg bw/d. Two sub chronic toxicity studies in rats with nonstandard dosing levels (single exposure group of 500 mg/kg bw/d) hinted at increased liver size and metabolism. This latter finding might not be unusual when ~1% of feed is substituted by a test compound.

The registrants derived a general population oral Derived No Effect Level (DNEL) based on a repeated dose study with the structural analogue DEP which established a NOAEL of 750 mg/kg bw/d. No factor for dose response or exposure duration is proposed. They applied an interspecies factor for allometric scaling of 4 but argued that no factor would be needed for other differences since the only effect observed was unspecified reduction in body weight (likely due to diet palatability). The default intraspecies factor of 10 for the general population is used and an additional factor 2 for the quality of the whole database was used. This resulted in a general population oral DNEL of 9.4 mg/kg bw/d.

All in all, a NOAEL of 1000 mg/kg bw/day based on the new sub-chronic repeated dose study and supported by the old chronic dose study seems to be appropriate. In this case no assessment factor for dose response and exposure duration would be needed. The standard interspecies factor for allometric scaling would be 4 and for remaining differences would be 2.5. The intraspecies factor for the working population would be 5. Given the relative abundance of information and the low criticality of the toxicological effect no assessment factors for database quality or additional uncertainty would be needed. This would result in an oral DNEL for workers of 20 mg/kg bw/d.

Diethyl Phthalate (DEP)

The use pattern of DEP (cas: 84-66-2) is similar to DMP and should not be expected to be present in flexible PVC containing waste nor in recycled product. It is however even more common component in cosmetics and

[4] For example, it can be used to dissolve solid additives for use in flexible polyurethane foam production.

fragrances than DMP and exposure can be expected from these sources is predominantly result of private non-occupational uses, but may be caused by occupationally used/provided hand creams.

A brief search on the ECHA website and in the EFSA journal did not reveal any official authority reports on DEP in which a toxicological benchmark was established. The lead registration dossier however did contain sufficient information to conclude on its toxicity.

Genotoxicity can be ruled out based on several in vitro tests. A chronic dermal carcinogenicity study in mice and rats which concluded that: "*There was no evidence of carcinogenic activity of diethyl phthalate in male or female F344/N rats. There was equivocal evidence of carcinogenic activity in male and female B6C3F1 mice based on increased incidences of hepatocellular neoplasms, primarily adenomas.*". With regards to the findings in mice it should be noted that mice end to be quite sensitive to liver tumours with control group incidence being relatively high already. Furthermore, the NOAEL for this effect was above the limit dose of 1000 mg/kg bw/day: >=1050 mg/kg bw/day for males and >=1100 mg/kg bw/day for females. All in all the registrants conclude that this is insufficient evidence for a carcinogenicity classification.

A two-generation reproductive toxicity study in rats with successive exposure of 2 generations, the NOAEL for general toxicity and reproductive performance was set at the highest dosed group of 15000 ppm (equivalent to a mean intake of 1150 -1375 mg/kg/day). A NOAEL for development and growth of pups was established at 3000 ppm (equivalent to a mean intake of 222 -267 mg/kg/day) due to decreased body weight gain in the 15000 ppm dosed group. The developmental toxicity was further elucidated in two Prenatal Developmental Toxicity Studies, one in rats and one in rabbits. The latter was of slightly lower quality but showed no effect on development. In rats maternal toxicity occurred in the 2.5% dose group and developmental toxicity occurred in the 5% dose group. This resulted in a NOAEL for maternal toxicity at 0.25% (around 200 mg/kg bw/d) and a NOAEL for developmental toxicity of 2.5% (around 2000 mg/kg bw/d). In these cases it is possible or even likely that developmental effects are seen as a result of the maternal toxicity and therefore should not be regarded as true developmental effects. All in all, the substances need not be classified for reproductive toxicity.

Finally repeated dose toxicity was investigated in rats with a sub-chronic oral study which concluded that: *Overall food consumption was significantly lower in rats of both sexes given 5% and females given 1%. On necropsy the absolute weights of the brain, heart, spleen and kidneys were statistically significantly lower than controls in both sexes given 5% in the diet for 16 weeks. Histopathology revealed some fatty degeneration and slight vacuolation of the liver, pyleonephritis and lymphocytic infiltration of the kidney. The incidence was not dose related and all are common findings in the strain of rat used. The NOAEL for the study was 0.2% in the diet (equivalent to 150 mg/kg bw/day) based on the observed changes in body weight/food consumption and organ weights.*

The registrants identified the 150 mg/kg bw/d as a point of departure to derive inhalation and dermal worker DNELs as well as general population inhalation, dermal, and oral DNELs. For the general population DNEL they applied no assessment factor for the does response relationship, a factor of 2 for the exposure duration, intraspecies factors of 4 for allometric scaling and 2.5 for other differences, and an intraspecies factor of 10 for the general population. All in line assessment factors were in line with ECHA guidance R8.

For the purpose of this study a PoD of 150 mg/kg bw/d would be supported by the repeated dose toxicity study, but also cover any residual uncertainty of other effects for other end points (e.g. carcinogenicity in mice is only seen at far greater levels). The same default factors as the registrants can be applied with exception of to the intraspecies factor which is set to 5 since this is the default factor for the working population. This results in a combined worker DNEL of 1.5 mg/kg bw/d.

Diisobutyl Phthalate (DiBP)

DiBP is a low molecular weight plasticiser that has been used in flexible PVC in the EU (and still is being used outside of the EU). While it will be very functional as a plasticiser, it also has a relatively high volatility and was(/is) thus often used in combination with other somewhat less volatile plasticisers. The substance can also be used as a solvent for other substances and therefore has been (/is) used in applications like lacquers, printing inks, sealants, and adhesives.

DiBP has been evaluated by the US Consumer Product Safety Commission (CPSC 2011), ECHA in a REACH Restriction Proposal (ECHA 2016a), and by EFSA (EFSA 2019). The CPSC evaluation derived for a point of departure (PoD) a $BMDL_{10}$ of 9.8 mg/kg bw/d based on developmental effects during a gestational exposure study (Howdeshell et al. 2008) where pregnant female Sprague-Dawley rats were gavage dosed with DiBP during GD8-18. ECHA was of the opinion that a previously used LOAEL PoD of 125 mg/kg bw/d based on a study showing histological effect of DiBP on adult tested and epididymides (Saillenfait, Sabaté, and Gallissot 2008), was associated with a high degree of uncertainty[5]. Instead, ECHA performs read-across with DnBP where it argues that there is a 25% potency difference and that the LOAEL of 2 mg/kg bw/d of DnBP would translate to a LOAEL of 2.5 mg/kg bw/d for DiBP. EFSA did not evaluate DiBP explicitly since it is not approved for use in food contact materials.

The European Human Biomonitoring initiative (HBM4EU), a (partially) EU funded collaboration between academia and Member State Competent Authorities, published a derivation of biomonitoring guidance values (Lange et al. 2021). These values are expressed as mg of a specific metabolite per Liter of urine and the one for DiBP in workers is 3.5 mg MiBP/L urine while the one for the general population is 0.16 and 0.23 mg MiBP/L urine for children and adults incl. adolescents. The supplementary information reveals that for general population limit the same logic is applied as in the ECHA REACH Restriction Proposal, whereby a study into DnBP with exposure from GD 15 – PND 21 demonstrating delayed germ cell development and male mammary gland changes at doses as low as 2 mg/kg bw/d (Lee et al. 2004) is taken to be a LOAEL and the 25% potency difference results in a LOAEL for DiBP of 2.5 mg/kg bw/d. The same assessment factors (10 for intraspecies, 10 for interspecies, and 3 for LOAEL→NOAEL extrapolation) are used to obtain a DNEL of 0.0083 mg/kg bw/d. However, for the worker limit a study into the toxicity of DnBP where rats were exposed from GD13-19 was used showing that at a dose greater than 10 mg/kg bw/d there is a reduction of foetal testosterone and reduction in expression of key genes encoding proteins involved in cholesterol transport and steroidogenesis (Lehmann et al. 2004). This NOAEL for DnBP was converted to a NOAEL for DiBP assuming a 25% potency difference to 12.5 mg/kg bw/d. Subsequently an interspecies assessment factor of 10 and an interspecies assessment factor of 10[6] is used to obtain a worker DNEL of 0.125 mg/kg bw/d.

The logic for using another study as a point of departure for the worker exposure limit is because the GD12-19 is an *in utero period of exposure, when extrapolated to humans, corresponds to the critical window of exposure for the male reproductive system, that is during the 1st trimester of pregnancy*. Which makes more sense for a worker toxicological benchmark derivation, since normally female workers are on maternity leave during the period following birth (i.e. the PND when translated to animal study terms).

For the purpose of this report, we note that the 12.5 mg/kg bw/d NOAEL of the HBM4EU PoD is not very different from the 9.8 mg/kg bw/d $BDML_{10}$ derived by the CPSC based on data for DiBP itself. As such the 12.5 mg/kg bw/d value is taken forward as a PoD in this report as well. However, here strictly the ECHA Guidance

[5] A point of view not shared by the REACH registrants which maintain the 125 mg/kg bw/d LOAEL as a PoD for DNEL derivation.

[6] A factor of 10 is however in contradiction to ECHA Guidance which specifies that a value of 5 should be used for interspecies differences.

R12 (ECHA 2012a) is followed for selection of assessment factors meaning a factor of 4 for interspecies allometric scaling and 2.5 for remaining interspecies differences and a factor of 5 for worker intraspecies differences to arrive to a DNEL of 0.25 mg/kg bw/d.

Dibutyl Phthalate (DnBP)

DnBP has similar properties and thus uses as DiBP, but is slightly more expensive to produce normally. As such it will be less prevalent in the waste materials and the technosphere in general.

DnBP has been evaluated by ECHA in a REACH Restriction Proposal (ECHA 2016a) and by EFSA (EFSA 2019). ECHA concluded that the point of departure should be a LOAEL of 2 mg/kg bw/d based on a study with exposure from GD 15 – PND 21 demonstrating delayed germ cell development and male mammary gland changes (Lee et al., 2004). The agency subsequently applied the following assessment factors: 4 and 2.5 for allometric scaling and remaining interspecies differences, 10 for intraspecies differences, and 3 for LOAEL→NOAEL extrapolation; to arrive to a DNEL of 0.0067 mg/kg bw/d. EFSA concurred with ECHA on the PoD of 2 mg/kg bw/d as a PoD and applied the following assessment factors: 100 for inter- and intra-species differences and a factor of 2 for LOAEL→NOAEL conversion; to arrive at a TDI of 0.01 mg/kg bw/d.

The HBM4EU limit value for DnBP in urine is 3 mg MnBP/L for workers and 0.12 and 0.19 mg MnBP/L for children and adults incl. adolescents in the general population (Lange et al. 2021). For the derivation of the general population limits, the study (Lee et al. 2004) with exposure from GD 15 to PND 21 demonstrating delayed germ cell development and male mammary gland changes with a LOAEL of 2 mg/kg bw/d is taken as the PoD. The following assessment factors are used: 10 for interspecies differences, 10 for intraspecies differences, and 3 for LOAEL→NOAEL conversion. Taking into account the difference in relevant exposure period for workers (see more detailed explanation under the DiBP section), a study with exposure during GD 12-19 showing reduction of foetal testosterone and reduction in expression of key genes encoding proteins involved in cholesterol transport and steroidogenesis with a NOAEL of 10 mg/kg bw/d is taken forwards as a PoD. Assessment factors of 10 for interspecies and 10 for intraspecies differences are used.

For the purpose of this report the PoD of 10 mg/kg bw/d identified for workers under the HBM4EU project will be used. However, strictly the assessment factors of ECHA Guidance R12 will be used for DNEL derivation, namely 2.5 and 4 for allometric scaling and remaining interspecies differences and 5 for worker intraspecies difference. Resulting in a DNEL of 0.2 mg/kg bw/d.

Butylbenzyl Phthalate (BBzP)

BBzP is a low molecular weight plasticiser with a better ability to plasticise PVC in low temperature applications than DEHP. It was used often in combination with other plasticisers in flexible PVC applications (and is likely still used to some degree outside of the EU). There may have been (/be) some use as a solvent for other substances in lacquers, printing inks, sealants, and adhesives.

BBzP has been evaluated by ECHA in a REACH Restriction Proposal (ECHA 2016a) and by EFSA (EFSA 2019). ECHA observed that in a previous EU RAR evaluation in 2007 a NOAEL of 50 mg/kg bw/d for developmental effect was identified based on a relevant study (Tyl et al. 2004) and a NOAEL for fertility effect was determined to be 100 mg/kg bw/d (Nagao et al. 2000). It noted that since then a two generation study showing decreasing anogenital distance (AGD) in male offspring with a LOAEL of 100 mg/kg bw/d as well as a study by Ahmad et al. (2014), showing reduced reproductive organ weight and altered sperm count at 100 mg/kg bw/d with a NOAEL at 20 mg/kg bw/d. Based on this, ECHA came to the position that the NOAEL should be considered 50 mg/kg bw/d. EFSA came to the same conclusion as ECHA. Both ECHA and EFSA used the same total assessment factor of 100, for ECHA split out by 4 and 2.5 for allometric scaling and remaining interspecies differences and 10 for intraspecies differences. Both conclude on a toxicological benchmark for the general population of 0.5 mg/kg bw/d.

The HBM4EU limit for BBzP is 3.0 MBzP/L for workers and adults including adolescents and 2.0 MBzP/L for children (Lange et al. 2021). These limits are based on rection in foetal testicular (Furr et al. 2014) and adult serum testosterone and additional changes in sperm parameters (Ahmad et al. 2014) which both give a LOAEL of 100 mg/kg bw/d. This is explicitly in contradiction to the ECHA and EFSA opinions. To the LOAEL the following assessment factors are applied: 10 for interspecies, 10 for intraspecies, 3 for LOAEL→NOAEL extrapolation, and 3 for data quality. This would translate into a DNEL of 0.11 mg/kg bw/d.

Since the consideration of the Furr et al. (2014) study seems to be the main difference underpinning the difference in point of departure, this study was further assessed. Pregnant rats were exposed during GD14-18 to varying concentrations of a test substance and sacrificed at GD18 whereafter three testis from three foetuses were excised and subjected to an *ex vivo* testicular testosterone production assay. The experiments were performed in blocks where for each block 15 pregnant female rats divided over 5 groups of 3 animal. In the first set of blocks 750 mg/kg bw/d of a test compound was given to 4 of the groups (the 5th serving as a control), with the aim to determining whether the test compounds could elicit a positive or negative response. In subsequent blocks differing concentrations of the same test compound were given to the groups of 3 animals to establish dose responses.

BBzP was established to be positive in the initial experiments and included in two dose response blocks (see Figure 2). In the first dose response block BBzP showed a dose response producing significant reduction in testosterone production at the lowest tested dose of 100 mg/kg bw/d which further reduced slightly with increasing maternal dose up to 900 mg/kg bw/d. Based on these observations alone one could establish that the LOAEL for the testicular testosterone production is 100 mg/kg bw/d. However, BBzP was included in a second dose response block that investigated the maternal doses of 11, 33, and 100 mg/kg bw/d, which showed that there was no statistically significant reduction in testicular testosterone production up to 100 mg/kg bw/d. Should this data be taken in isolation, one could establish a NOAEL at 100 mg/kg bw/d.

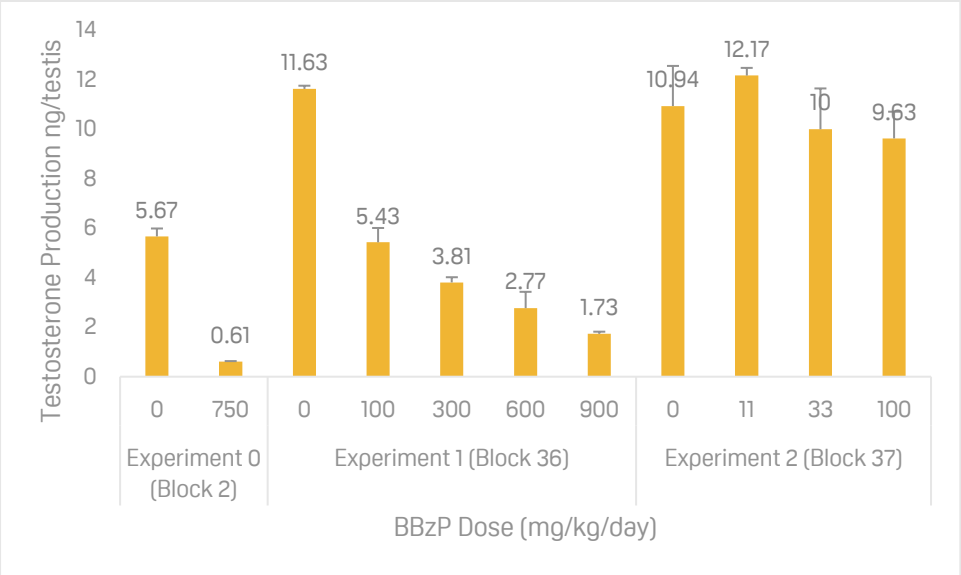


Figure 2 ex vivo testis testosterone production of foetuses (ng/testis) of dams sacrificed at GD18 following administration of varying doses of BBzP to dams during GD14-18. Error bars indicate the standard error. Adapted from: Furr et al. (2014)

In light of this ambiguity, and the whole body of evidence showing NOAELs at 33-100 (Furr et al. 2014), 20 (Ahmad et al. 2014), 50 (Tyl et al. 2004), and 100 (Nagao et al. 2000) mg/kg bw/d, as well as LOAELs at 100 (Furr et al. 2014; Ahmad et al. 2014); the ECHA position that the NOAEL should be 50 mg/kg bw/d is not an unreasonable one and will be taken forward as a point of departure. The default assessment factors of 2.5

and 4 for allometric and other interspecies differences and the worker intraspecies differences factor of 5 are applied to arrive to a DNEL of 1 mg/kg bw/d. The factor for LOAEL→NOAEL is not used since the 50 mg/kg bw/d is considered a NOAEL. The factor of 3 for data quality used by the HBM4EU project, is not applied since the database supporting the 50 mg/kg bw/d is extensive and robust.

Di(2-ethylhexyl) Phthalate (DEHP)

DEHP was the plasticiser of choice in the EU for decades because of its excellent miscibility with PVC, balanced low and high temperature resistance, and strong plasticising effect. The use within the EU has been largely phased out in favour of DINP an DEHTP. The use of DEHP outside of the EU should be considered to be substantial, although there is an increasing move towards DINP and DEHTP as well as other regulatory regions enact restrictions on the use of DEHP (and low molecular weight plasticisers) and companies may voluntarily switch as well. Those regions/companies making the switch today, will benefit from the reformulation expertise that was developed in the EU over the past decade or so.

DEHP has been evaluated by the Risk Assessment Committee (RAC) in a specific DNEL derivation for application for authorisation (ECHA 2013a), ECHA in a REACH Restriction Proposal (ECHA 2016a) [7], and by EFSA (EFSA 2019). RAC took as a point of departure a NOAEL of 4.8 mg/kg bw/d from a three-generation study with dietary exposure of DEHP to rats (Layton and Wolfe 2004) as a starting point, but calculated a corrected NOAEL of 3.4 mg/kg bw/d based on evidence suggesting that oral absorption in rats is 70% and in humans would be 100%. Oral DNELs for the general population were calculated with default assessment factors of 2.5 and 4 for allometric and other interspecies differences and the general population intraspecies differences factor of 10; resulting in a 0.034 mg/kg bw/d oral DNEL for the general population. No worker oral DNELs were derived, however for the dermal DNEL a factor of 5/7 was used to correct for exposure duration (5 days per workweek vs 7 days per week exposure duration in oral feed study). In the context of the REACH restriction proposal ECHA used the same rationale to set an internal DNEL of 0.034 mg/kg bw/d. EFSA used the same point of departure of 4.8 mg/kg bw/d, but did not correct for species specific absorption and simply applied an assessment factor of 100 to arrive at a TDI of 50 µg/kg bw/d.

Under the HBM4EU initiative, the EFSA TDI was favoured over the RAC/ECHA approach as a starting point for general population urinary metabolite health based guidance values (Apel and Ougier 2017). However, for workers the authors consider the use of a multigenerational study inappropriate and identified a chronic oral diet study with male and female rats as a key study. The most sensitive end-point representative of anti-androgenic effect was bilateral aspermatogenesis observed in the 500, 2500, 12500 ppm in feed dose groups (corresponding to 36.1, 181.7, and 938.5 mg/kg bw/d); with a NOAEL established by the 100 ppm dose group (5.8 mg/kg bw/d). The assessment factors used in this study are roughly[8] 2.5 and 4 for allometric and other interspecies differences and a factor of 5 for worker interspecies difference. This would result in a DNEL of 0.116 mg/kg bw/d.

For the purpose of evaluating worker DEHP exposure on its own in this report the worker DNEL of 0.116 mg/kg bw/d is used. Since this may be somewhat controversial as it deviates from the RAC/ECHA approach, it is worthwhile to note that the corrected NOAEL for worker oral exposure taking into account the 5 days exposure per workweek factor would be 4.7 mg/kg bw/d and the resulting worker oral DNEL would be 0.094 mg/kg bw/d. Furthermore in the derivation document it was note that: "*RAC considers the NOAEL of 4.8*

[7] Note the distinction between RAC and ECHA. RAC is a committee of experts delegated by the Member States on personal title and it derived the DNEL in the 2013 document. ECHA staff drafted the REACH Restriction Proposal.

[8] The description hereof in the source publication is relatively convoluted.

mg/kg bw/day to be "conservative", given the low incidences at the LOAEL". Combined we do not see an issue with the chosen DNEL.

Diisononyl Phthalate (DiNP)

DiNP and DEHTP are the alternatives to DEHP (and other lower molecular weight ortho-phthalates), they have gained dominance in the plasticiser application in the EU and prominence elsewhere. Given their slightly higher viscosity, they are less likely candidates to be substitutes for the solvent use of DiBP and DnBP in applications like lacquers, printing inks, sealants, and adhesives.

DiNP has been evaluated twice by EFSA (EFSA 2005, 2019). In the first evaluation a chronic toxicity oncogenicity study in F-334 Rats (Exxon Biomedical Sciences 1986) was identified as a key study with a NOAEL of 15 mg/kg bw/d based on *increased incidence of spongiosis hepatis, accompanied by increased serum levels of liver enzymes and increases in absolute and relative liver and kidney weights in both sexes*. Using their default interspecies factor of 10 and intraspecies factor of 10 a TDI was established at 0.15 mg/kg bw/d. The subsequent group evaluation of DiNP together with DnBP, BBzP, DEHP, and DiDP the non-peroxisomal proliferation-related chronic hepatic and renal effects in rats were again identified as the most critical endpoint and the TDI for of 0.15 mg/kg bw/d was reaffirmed.

Under the HBM4EU project it seems no HBM-GV_{GenPop} was established for DiNP, however a **ρ**HBM-GV_{GenPop} was derived for **ρ**hthalate syndrome mixture risk assessment purposes (Kortenkamp and Koch 2020; Lange et al. 2022). In the key study identified for antiandrogenic activity pregnant rats were dosed with 50, 250, and 750 mg/kg bw/d of DiNP during GD12-19 and sacrificed either after 2 hours or 24 hours . Foetal testicular testosterone levels were quantified, and histological examination of testis was performed. In the 250 and 750 mg/kg bw/d dose groups testosterone production was reduced 2 hours after the last dose was administered (but not after 24 hours) and the incidence of multinucleated germ cells (MNG) increased (Table 3). A NOAEL was established at 50 mg/kg bw/d by the study authors (Clewell et al. 2013).

*Table 3 Effects on markers of sexual development at 2 and 24 h following the final dose of 50, 250, or 750 mg/kg/day DiNP from GD 12 to 19. [a] Time following the final dose of 50, 250, or 750 mg/kg/day from GD 12 to 19. [b] Measured in all pups and averaged by litter. The litter was used as the statistical unit. [c] Histopathology was performed on one randomly assigned male pup per litter. Morphological changes were scored using a semi-quantitative, 5-step grading system ranging from minimal (grade 1) to severe (grade 5). Value shown represents the number of animals with increased severity of MNGs or LC aggregates. [d] Measurements performed on one randomly assigned male pup per litter. [e] Testosterone was measured separately for the testes pairs of two male pups per litter and averaged by litter. The litter was used as the statistical unit. * $p < 0.05$, 1-way ANOVA with Dunnett's post-test. ** $p < 0.01$, 1-way ANOVA with Dunnett's post-test. *** $p < 0.001$, 1-way ANOVA with Dunnett's post-test.*

	Time point[a]	Control	50 mg/kg	250 mg/kg	750 mg/kg
Number of litters examined	2	25	8	8	7
	24	27	8	8	8
Absolute AGD (mm)[b]	24	2.48(0.03)	2.43(0.06)	2.43(0.05)	2.52(0.04)
Scaled AGD (AGD/BW1/3)[b]	24	15.53(0.17)	15.14(0.40)	15.29(0.29)	15.64(0.24)
# Animals with MNGs[c]	24	0	0	2	6*
# MNGs per testis section[d]	24	0(0)	0(0)	0.75(0.31)**	1.25(0.45)***
MNGs per ST cross-section[d]	24	0(0)	0(0)	0.02(0.01)*	0.03(0.01)***
Increased # of germ cells[c]	24	0	0	0	2
# Animals with large LC aggregates	24	2	3	1	7*
ST diameter (um)	24	58(1)	56(1)	58(2)	60(1)
Testis testosterone (% control)[e]	2	100(9)	104(19)	50(11)**	35(5)***
Testis testosterone (% control)[e]	24	100(12)	84(19)	161(33)	122(25)

Kortenkamp and Koch (2020) calculated a benchmark dose with a response of 5% (BDM_5) of 80 mg/kg bw/d and a Benchmark dose (lower bound) (BDML) of 5.9 mg/kg bw/d. The BDML was used as a point of departure to derive a Reference Dose for Anti-Androgenic Activity (RfD AA) of 0.059 mg/kg bw/d using a "standard uncertainty factor" of 100. The use of a BDML that of 5.9 mg/kg bw/d as a point of departure, while the study data (see Table 3) show such a clear NOAEL at 50 mg/kg bw/d is debatable. The study authors indicate that this is in line with EFSA Guidance (EFSA 2017) and is *in line with analysis of 246 developmental and reproductive toxicity studies (Allen et al. 1994) and of experiments from the US NTP (Bokkers and Slob 2007) which established that the BDML for a BMR of 5% was on average close to the respective NOAELs*.

For the current evaluation we note that Allen et al. (Allen et al. 1994) demonstrated that the BDM_5 tends to be in agreement with NOAELs that are of the continuous data variable type (e.g. testis testosterone as a percentage of control). The statistically determined lower bounds of a 95% confidence interval represent an additional factor taking into account uncertainty on top of the normally used assessment factors. Indeed, in this case the BDM_5 is 80 mg/kg bw/d and thus very much in agreement with the very clear NOAEL of 50 mg/kg bw/d that is observed in the study.

As such the most sensitive point of departure remains the 15 mg/kg bw/d based on non-peroxisomal proliferation-related chronic hepatic and renal effects in rats. To obtain a worker DNEL, an assessment factor of 2.5 and 4 is used for allometric scaling and other interspecies factors and a worker intraspecies factor of 5 is used to arrive to a combined worker DNEL of 0.3 mg/kg bw/d.

Diisodecyl phthalate (DiDP)

DiDP is an alternative to DEHP that tends to be slightly more expensive than DiNP and DEHTP, has a slightly lower plasticising efficacy, but slightly greater high temperature resistance. As such it is a somewhat less used alternative to DEHP.

Both ECHA (2013b) and EFSA (2019) evaluated the toxicity of DiDP. In terms of repeated dose toxicity, ECHA identified a chronic study in rats demonstrating a LOAEL for spongiosis hepatis at 22 mg/kg bw/d (Cho et al. 2008, 2010), a 90 day study in dogs with a NOAEL of 15 mg/kg bw/d based on hepatic effects, and a 90 day study in rats establishing a NOAEL of 60 mg/kg bw/d based on increased relative liver weight. ECHA applied a factor 2 to derive corrected L-/N-OAELs as animals seem to absorb 50% of the dose administered orally, while humans absorb 100% of the dose. The derivation is subsequently performed with assessment factors as described in Table 4. For a not explained reason, ECHA subsequently averaged the outcome of the derivations to set the general population DNEL at 0.75 mg/kg bw/d.

*Table 4 DNEL Derivation for Repeated Dose Toxicity by ECHA. *The default assessment factor for sub-chronic to chronic extrapolation is 2 for a rat 90 day study. The lifespan of a Beagle dog is around 13 year; thus, a study duration of 90 days covers roughly 2% of its lifespan. As a comparison between dog and rat, a 28 day study (subacute) covers 4% of the lifespan and a 90 day study (sub-chronic) covers 12% of a rat's life. Thus, a 90 day dog study covers about half of the length of a subacute study in rats. This justifies a default assessment factor of 6 for subacute to chronic extrapolation for the 90 day dog study (see Table R. 8-5 in ECHA guidance R.8)*

	90 d Dog Study	90 d Rat Study	2 year Rat Study
Critical Effect	Liver Toxicity	Increased Liver Weight	Spongiosis hepatis
PoD Type	NOAEL	NOAEL	LOAEL
PoD (mg/kg bw/d)	15	60	22
PoD Abs Corrected (mg/kg bw/d)	7.5	30	11
Interspecies Allometric Scaling	1.4	4	4
Interspecies Remaining Differences	2.5	2.5	2.5
Intraspecies	10	10	10
Exposure Duration	6*	2	1
LOAEL→NOAEL	1	1	3
DNEL (mg/kg bw/d)	**0.036**	**0.15**	**0.037**

While ECHA did conclude that DiDP does not have antiandrogenic effect a NOAEL of 33 mg/kg bw/d was identified based on neonatal survival rates. To this NOAEL the same factor of 2 is used to correct for absorption in rats vs humans, and default assessment factors 4 for allometric scaling and 2.5 for remaining interspecies differences and 10 for intraspecies differences were applied. The resulting DNEL is 0.08 mg/kg bw/d.

EFSA (2019) also concluded that the DiDP does not exhibit antiandrogenic effect and identified the NOAEL of 15 mg/kg bw/d established by the 90 d dog study as the most sensitive end-point. Contrary to ECHA, EFSA did not correct for absorption or extensively discuss assessment factors and just used a total factor of 100 (based on 10 for intraspecies and 10 for interspecies differences). This resulted in a TDI of 0.15 mg/kg bw/d

The original 90 day dog and rat study reports, while summarised in the REACH Registration dossier, are not available to the author of this report and thus it is difficult to judge the underlying source data. However, they both highlight an effect in the liver and the 2-year carcinogenicity study did include an examination of liver weight and histopathology (Cho et al. 2008, 2010). Changes in relative liver weight (and other changes in organ/body weight) only occur at the highest dose group (see Table 5). Histopathological changes that were

identified in the liver of the low dose group were microgranuloma[9], "Spongiosis hepatis" in males and inflammation in females. The microgranulomas and inflammation are of low incidence and there is not following a dose response pattern, thus of questionable relevance, and unlikely to qualify as adverse. Spongiosis hepatis also known as cystic degeneration in the liver is seen by some as a pre-neoplastic lesion but not by others (Karbe and Kerlin 2002; Bannasch 2003; Kerlin and Karbe 2004). However, even if it were a pre-neoplastic lesion, it should be noted that observing a pre-neoplastic lesion in non-chronic (e.g. 90-day studies) gives rise to a substantially different level of concern than when it is observed in studies with chronic exposure. Furthermore, the paper by Cho et al. (2008, 2010) does not discuss historical control incidence for this histopathological finding. A cursory look at a US NTP study performed at roughly the same time revealed that the control group had an incidence of Cystic Degeneration in the liver of 2/48 (2%) in female F-344 rats and 11/48 (11%) in males (US NTP 2012). Considering the other relative organ weight changes and histopathological evaluations of other tissues, the substance seems to be well tolerated up to and including the mid dose group.

For the purpose of this report, the NOAEL of 33 mg/kg bw/d based on neonatal survival rates is taken as a point of departure. The ECHA correction for absorption is tentatively taken forward and assessment factors of 2.5 allometric scaling and 4 for remaining interspecies differences as well as a factor of 5 for intraspecies differences is used to arrive to a DNEL of 0.33 mg/kg bw/d.

*Table 5 Final body weights and relative organ weights for F344 rats exposed to DIDP for 2 years (n=52 for all groups). Relative organ weights are given as mg organ weight/g body weight (mean ± S.D.) * Significantly different (P < 0.01) from the vehicle control group by Williams' or Dunnett's test. Taken from: Cho et al. (2008, 2010)*

Males	**Dose (mg/kg bw/d)**	**0**	**21.86**	**110.25**	**479.2**
	Body weight	350.43±46.72	344.19±56.97	357.31±60.80	301.49±68.25*
	Kidney	7.94±1.60	8.63±2.79	8.12±1.53	10.46±2.96*
	Liver	31.81±11.26	36.29±20.31	35.04±21.24	44.41±16.02*
	Spleen	6.89±10.26	10.81±17.01	10.31±17.41	13.19±15.00
	Testis	14.19±5.38	13.53±5.91	14.36±5.16	14.87±8.10
Females	**Dose (mg/kg bw/d)**	**0**	**22.92**	**128.18**	**619.56**
	Body weight	269.68±36.04	272.68±69.07	268.24±66.46	221.29±50.15*
	Kidney	8.07±1.43	8.51±3.05	8.94±3.72	10.18±2.77*
	Liver	31.02±8.33	31.75±11.00	32.14±7.66	46.17±13.89*
	Spleen	5.45±9.67	6.88±9.43	7.48±12.84	11.71±16.28
	Ovary	0.71±2.47	0.36±0.27	0.57±0.86	0.49±0.53

[9] A granuloma itself is a small localised cluster of immune cells (macrophages) that forms in response to chronic inflammation, infections, or foreign substances. Microgranuloma's would be smaller instances of the same phenomenon.

*Table 6 Incidence of non-neoplastic liver lesions in rats exposed to DIDP for 2 years. Displayed as: number of animals with lesion (percentage). *, **Significantly different (P < 0.05 and P < 0.01) from the vehicle control group by the poly-3 test. Adapted from: Cho et al. (2008, 2010)*

Males	**Dose (mg/kg bw/d)**	**0**	**21.86**	**110.25**	**479.2**
	n	49	48	49	39
	Fatty change	4(8.2)	6(12.5)	1(2.0)	0*(0.0)
	Altered cell foci	27(55.1)	19(39.6)	18*(36.7)	3**(7.7)
	Oval cell hyperplasia	1(2.0)	3(6.3)	2(4.1)	6*(15.4)
	Hypertrophy	0(0.0)	0(0.0)	1(2.0)	4*(10.3)
	Microgranuloma	1(2.0)	5*(10.2)	6*(12.2)	4*(10.3)
	Necrosis	3(6.1)	7(14.6)	5(10.2)	8*(20.5)
	Peliosis	1(2.0)	0(0.0)	2(4.1)	4*(10.3)
	Spongiosis hepatis	0(0.0)	3*(6.3)	3*(6.1)	5**(12.8)
Females	**Dose (mg/kg bw/d)**	**0**	**22.92**	**128.18**	**619.56**
	n	49	47	47	40
	Altered cell foci	31(63.3)	26(55.3)	27(57.4)	17*(42.5)
	Inflammation	2(4.1)	8*(17.0)	11**(23.4)	3(7.5)
	Microgranuloma	10(20.4)	6(12.8)	12(25.5)	3*(7.5)
	Necrosis	2(4.1)	4(8.5)	6(12.8)	9**(20.9)

Di(2-ethylhexyl) Terephthalate (DEHTP)

DiNP and DEHTP are the alternatives to DEHP (and other lower molecular weight ortho-phthalates), they have gained dominance in the plasticiser application in the EU and prominence elsewhere. Given their slightly higher viscosity, they are less likely candidates to be substitutes for the solvent use of DiBP and DnBP in applications like lacquers, printing inks, sealants, and adhesives.

DEHTP was evaluated by EFSA (2008) following a petition that included a mutagenicity tests, repeated dose toxicity data including a chronic carcinogenicity study in rats, and reproduction/teratogenicity studies. The substance was concluded to be negative for mutagenicity and reproductive toxicity. The NOEAL identified was 79 mg/kg bw/d, based on effects on the retina and the nasal turbinates observed in the chronic carcinogenicity study. A TDI of 1 mg/kg bw/d *is derived by rounding up*[10].

For the purpose of this report, same point of departure is used of the NOAEL of 79 mg/kg bw/d. However, the assessment factors are 4 for allometric scalling and 2.5 for remaining interspecies differences and 5 for worker intraspecies differences to arrive to a DNEL of 1.58 mg/kg bw/d.

Diisononyl 1,2-cyclohexane dicarboxylic acid (DINCH)

DINCH is a more expensive alternative plasticiser with inferior plasticising efficacy to DiNP and DEHTP. As such it has failed to reach dominance in the plasticiser application in the EU, however it does serve a prominent role in niche applications (e.g. medical devices) where it is perceived as inherently safer than other phthalate alternatives.

[10] It is implied that a total assessment factor of 100 consisting of 10 for interspecies and 10 for intraspecies is used.

EFSA (2005) evaluated the toxicity of DINCH following a petition that included a mutagenicity tests, repeated dose toxicity data including a chronic carcinogenicity study in rats, and reproduction/teratogenicity studies. The substance was concluded to be negative for mutagenicity and reproductive toxicity. The NOEAL identified was 100 mg/kg bw/d, based on adverse effects observed in the kidney in the sub-chronic repeated dose study. A TDI of 1 mg/kg bw/d was established with the "application of the default uncertainty factor of 100", presumably consisting of 10 for intraspecies and 10 for interspecies differences.

For the purpose of this report the 100 mg/kg bw/d is used as a point of departure, and an assessment factor of 2 for exposure duration (sub-chronic → chronic), 4 for allometric scaling and 2.5 for remaining interspecies differences, and 5 for intraspecies differences is use to arrive to a DNEL of 1 mg/kg bw/d for workers.

Mixture Risk Assessment

Certain phthalates have a similar toxicological impact on rats causing reduced testosterone production and a spectrum of related effects on the male reproductive system, commonly referred to a phthalate syndrome. According to Kortenkamp & Koch (2020), ortho-phthalates with too short or too long linear side chains are of very low potency, while linear side chain lengths of C4-C7 have a higher potency. The authors indicate that branched side chain ortho-phthalates the C4-C9 ortho-phthalates are also potent. Thus, they conclude that the ortho-phthalates that can contribute to phthalate syndrome are: DIBP, DBP, DiPP, DPP, DNHP, DCHP, BBP, DIHP, DHP, DEHP and DINP. Some of these are substances that are not produced or used in industry and consequently have not been the subject of much toxicological studies/research/assessment and are relatively irrelevant in terms of human exposure. They do derive toxicological benchmarks for the general population for the more relevant/studied: DBP, DIBP, BBP, DEHP, and DINP. They proceed to perform a mixture risk assessment based on urinary sample from the general population where they calculate RCRs for individual substances and add these together to create a sum-RCR, which they argue should be below 0.2 given that there may be other substances that are causing anti-androgenic effects.

The inclusion of DINP in the scope of such assessments is controversial. The ECHA Risk Assessment Committee has concluded that the substance should not be classified for reproductive toxicity (ECHA 2018). However, the EFSA, when mandated to reevaluate DnBP, BBzP, DEHP, DiNP, and DiDP, concluded that DiNP should be included in the group-TDI for DnBP, BBzP, DEHP, and DiNP; albeit with a much lower weighting than the other ortho-phthalates (EFSA 2019). The EFSA however acknowledged that DiNP affected foetal testosterone at levels (50 mg/kg bw/d) that were 3 times higher than the effect on the liver (NOAEL of 15 mg/kg bw/d). [Following this logic, a health based limit value based on the liver effect would also be protective against the anti-androgenic effect of DiNP. As such, exposure to DiNP alone should not give cause for concern of anti-androgenic effect as long as it remains below the threshold established for liver effects. Only if the ortho-phthalates that do have as a primary action the anti-androgenic effect are demonstrating relatively high exposure, could DiNP push the endocrine system over an edge.]

This is also reflected in the current version of Regulation (EU) 10/2011 on plastic materials and articles intended to come into contact with food, which in group restriction No. 36 determines the SML (T) (total specific migration limit) value of 0.6 mg/kg food to be the "sum of phthalic acid, dibutyl ester (DBP), diisobutyl phthalate (DIBP), phthalic acid, benzyl butyl ester (BBP) and phthalic acid, bis(2-ethylhexyl) ester (DEHP) expressed as DEHP equivalents using the following equation: DBP*5 + DIBP*4 + BBP*0,1 + DEHP*1" but does not consider DINP.

Both the approach by Kortenkamp & Koch and EFSA, do not strictly follow the logic of a recent harmonised classification proposal by France that aims to classify all anti-androgenic ortho-phthalates based on the well-established carbon backbone theory, which states that mono- and di-othro-phthalates with a carbon backbone with a C4-C6 length can cause phthalate syndrome. The carbon backbone is defined as "the

longest linear carbon chain from the ester function". The proposal also adds mono- and di-ortho-phthalates with either:

- *a benzyl moiety with methylene possibly substituted by methyl and/or ethyl groups;*
- *a cyclopentyl or cyclohexyl moiety with possible substitution by methyl and/or ethyl groups.*

Which also seem to promote phthalate syndrome. While this broad group definition would harmonise the reproductive toxicity 1b and endocrine disruptor 1 (HH&ENV) across a slew of different possible chemical structures (most of which are not intentionally produced or used in any industrial quantities), it excludes DiNP for the simple reason that DiNP has a carbon backbone of C8 and thus falls well outside of the definition[11].

For the purpose of this report, a mixture risk assessment based on the methodology developed by Kortenkamp and Koch (2020) will be followed. However, Reference Doses for Anti-Androgenic effect (RfD AA) will be recalculated using the same adjusted POD, but instead of using a total assessment factor applicable to the general population (100) an assessment factor for the working population will be used (50, comprised of 2.5 for allometric scaling and 4 for remaining interspecies differences and 5 for intraspecies differences). This approach is conservative as:

1. The PoDs used for DiBP, BBzP, and DiNP are based on BDML5 which tend to be an order of magnitude below the NOAEL of the substances.
2. The method includes DiNP which was not included in the scope of the CLH proposal for substances that contribute to phthalate syndrome nor in the final listing in Regulation (EU) 10/2011.

ENVIRONMENTAL EMISSIONS

Channelled emissions

All plants were equipped with air extraction systems that filter air before emissions. These systems will be summarised below. Furthermore, both plants did not use water in their recycling process, meaning there was no process water emissions or cleaning system that generated sludge.

Diffuse emissions

All plants were aware of the potential for diffuse emissions (e.g. spillages) and had put measures in place to address these. These will be discussed.

[11] There are possibly variants of DiNP that contain molecules with a C6 carbon backbone as an impurity resulting from the starting materials and possibly the production process. Manufacturers of the longer carbon backbone ortho-phthalates will have to determine whether their product would contain such impurities above 0.1% if the classification is adopted as proposed.

PLANT REPORTS

PLANT E

Plant E specialises in the recycling of pre-consumer flexible PVC waste streams mainly from cable producers (80 – 90% of annual capacity utilisation) but also processes post-consumer cable sheeting which is predominantly flexible PVC (10 – 20% of annual capacity utilisation. In total the plant recycles around 10 000 – 20 000 tons of waste per year. An outline of the facility can be found in Figure 3.

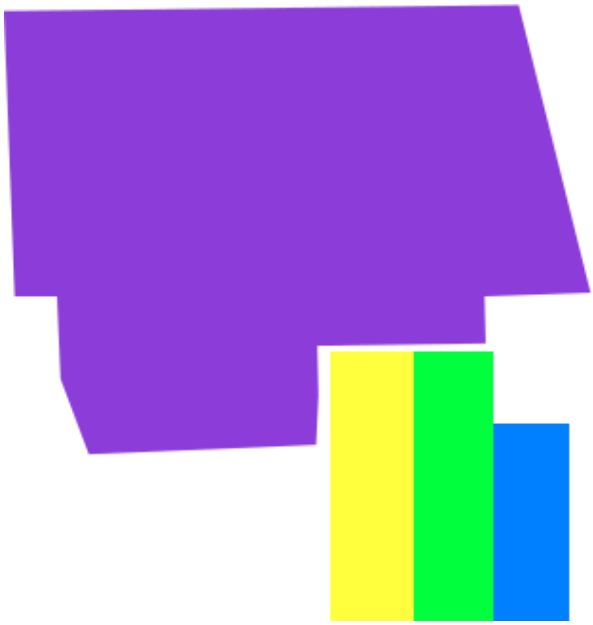


Figure 3 Layout of plant E. Blue is raw material receipt, green is mechanical treatment/sorting, yellow is extrusion with melt-filtration (and micronization) hall, purple is raw material and recyclate storage area.

The received material is stored either packaged in big bags outside where the plant has covered and uncovered storage areas or in bulk or packaged in an indoor area (Figure 4).

Figure 4 Raw material storage area. Left: outdoor covered and uncovered storage with sealed packaged waste and right: indoor storage area for waste in bulk.

The input waste material is first shredded and subsequently introduced into grinding equipment to achieve sufficient size reduction (Figure 5). The grinded material then either goes to compounding followed by extrusion with melt filtration or to a micronization process. The shredding and grinding equipment are connected to an air extraction system that filters the air of dust and reintroduces it into the mechanical treatment/sorting hall. In addition, the grinding equipment is completely boxed during operation.

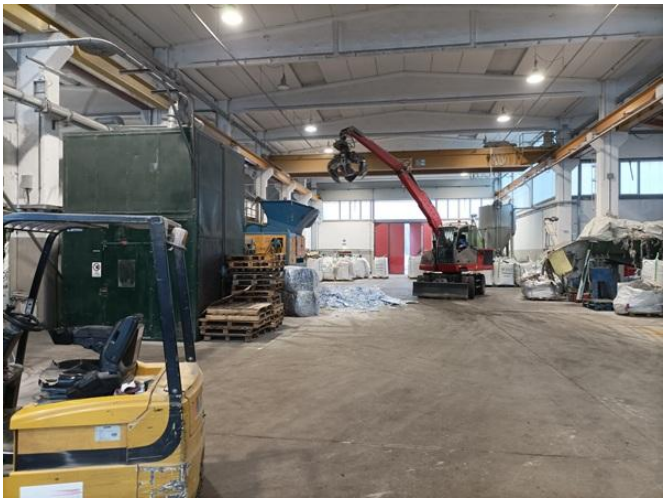



Figure 5 Mechanical Processing Hall. Left: crane used to feed the shredding equipment (blue machine). Right: Shredder being fed by pre-consumer foil and the subsequent grinding equipment (green boxed machine).

The majority of the regrind goes to the compounding followed by melt filtration process. To this end, big bags of regrind from the mechanical processing department are fed, with limited human intervention[12], into a hopper that feeds a silo storing the regrind (Figure 6).


Figure 6 Silo for regrind prior to compounding and extrusion with melt filtration. Left: hopper feeding station. Right: silo with sight glass showing two different types of regrind flexible PVC.

[12] The operation requires the positioning of forklift truck carrying the big bag over the mouth of the hopper and loosening the laces at the bottom of the beg. Afterwards the bag is left to drain without human intervention for minutes whereafter the worker collects the empty big bag.

The silo feeds a compounding (i.e. mixing) vessel into which various other additives such as carbon black and new plasticiser may be introduced (Figure 7).

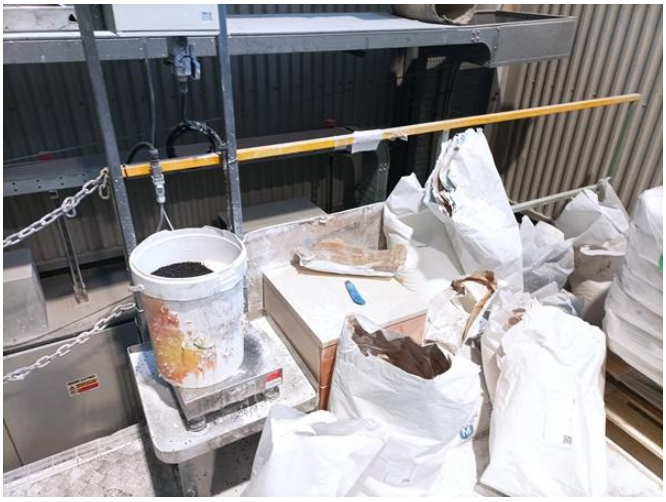



Figure 7 Compounding step. Left: compounding vessel with main raw material feed being regrinded material from silo that can be seen on the left side of the picture. Right: solid additive weighing station.

From this vessel, material is piped to an extruder that mainly through the application of mechanical energy with a screw, melts the material and causes intense homogenisation of the material. The molten plastic is pressed through metal mesh filters that cause the removal of non-melting parts[13] (Figure 8). This is the location where extrusion operators spend around 80% of their time and perform the critical operation of replacing the filters once the backpressure monitors indicate that so much non-melting parts have been collected that the filter needs to be changed.


Figure 8 Extrusion equipment. Left: shows compounding vessel from other side and the first part of the extruder. The big bags shown are where workers deposit filter cakes and spent metal mesh filters. Right: end of the extrusion line with a clear view of the melt filtration unit and the pipe transporting the pellets to the cooling station.

[13] Since flexible PVC tends to have a relatively low melting temperature, this step removed a variety of polymeric (e.g. polyurethane, crosslinked PE) and non-polymeric (e.g. residual metal) impurities from the material.

The operation is performed by the operator in close proximity to the warm material. First the layer of flexible PVC and non-melting parts is scraped from the metal mesh filter and the resulting warm filter cake is manually deposited in a nearby big bag (Figure 9 left). Subsequently, the metal filter is removed, placed in another big bag (Figure 9 right), and the filter is replaced. The filter cake can be recycled internally through micronization and the filter with some residues is residual waste of the recycling process and sent for disposal.


Figure 9 Residues from the melt filtration process. Left: filter cakes and Right: metal mesh filters.

It should be noted that at relevant parts of the extrusion with melt-filtration process there is specifically engineered extraction ventilation. The most crucial point for plasticiser exposure is the operation whereby the hot/warm filter cake is manually removed, and this is indeed equipped with extraction ventilation as can be seen in Figure 8. This is the crucial point because the temperature increases the vapour pressure, and therefore the possible volatilization, of the plasticisers.

Finally, the pellets are pneumatically transported through piping to a cooling station (Figure 10) prior to being fed automatically into silos and big bags for expedition to customers.


Figure 10 Pellet cooling station. Left: the first part of the cooling station where the material arrives pneumatically in the piping to a cyclone that then drops the pellets into the station. Right: the cooling station table where pellets are agitated and exchange their heat with an air stream.

In total plant G has 3 complete lines of hopper → silo → compounding → extrusion with melt filtration → cooling station → silo/big bag. At the time of the measurement 2 were in operation (TR2 and TR3). All these lines are connected to an air extraction system that is equipped not only for the removal of dust but has additional carbon filters to remove (plasticiser) vapours from the emitted airstream.

Next to the ability to process the regrind with compounding and extrusion with melt filtration, the Plant also has a micronization line where the material is very finely grinded to a powder and subsequently sieved. This process is also use to recycle the cooled filter cakes from the melt-filtration process. The resulting micronized material is put into a sealable big bag for expedition to customers (Figure 11). Where the extrusion with melt filtration process causes the removal of non-melting parts and the resulting pellet is of slightly higher quality, the micronization process reduces the size of the non-melting parts in such a way that they become less burdensome in subsequent conversion processes and simply become a form of a filler in the material.

Figure 11 Micronization equipment

This micronization equipment if connected to an air extraction system that is connected to the same dust cleaning system as the shredding and regrinding processes.

Risk Assessment for Human Health

Plant E normally has 3 - 7 workers operating in the plant:

- 1 – 2 Mechanical Treatment Operator
- 1 – 3 Extrusion Line Operators (1 per line)
- 1 Extrusion Supervisor and Micronization Operator

The mechanical treatment operator performs tasks like the opening of packaged flexible PVC waste, some limited manual sorting, and a major part of the time he is operating a crane to feed the shredder with waste material. It is relevant to know that in this hall all material is at room temperature or only slightly above[14] and as such, any plasticiser exposure will be mainly due to dust exposure or dermal contact with material.

Extrusion line operators spend 80% of their time around the machine and 20% of their time gathering regrind input material with a forklift truck and connecting these to a hopper to feed a silo or taking filled big bags away by forklift truck to storage areas for expedition. Roughly once every 5 – 6 minutes, the filter needs to be changed, and the operator takes around 5 minutes to do so. As such during a shift they are performing this task around x * 5 minutes. The processing of this hot/warm material does give rise to the possibility for plasticiser vapour exposure. The operation was not particularly dust generating, so any plasticiser exposure observed here will likely be due to volatile plasticiser and/or dermal contact. However, in the same hall there is some degree of dust generation from the micronization equipment.

The "Extrusion Supervisor and Micronization Operator" is assisting his peers in the extrusion department where necessary and ensures that the micronization equipment keeps running. Gathering and connecting big bags of regrind material to the hopper and disconnecting full big bags with micronized product and transporting this to storage areas.

In total, 4 workers were included in the air measurement campaign: 1 regrind operator, 2 extrusion operators, and 1 Extrusion Supervisor and Micronization Operator. For the biomonitoring campaign the same individuals were included as in the air measurement campaign, but an additional 4 workers with the same distribution of operators of the preceding shift were also included in the campaign.

Dust

Dust measurements performed in Plant E revealed that all workers were exposed to respirable and inhalable dust below the limit values adopted for this report of 1 and 5 mg/m³ for respirable and inhalable dust respectively (see Table 7 for results and Table 25 for raw measurement data). As such there is no adverse effect expected from exposure to dust.

Table 7 Results of the respirable and inhalable dust measurements in Plant E and Risk Characterisation Ratios (RCR) based on a limit of 1 mg/m³ for respirable and 5 mg/m³ for inhalable dust. RCR colour coding applied: <LOD = no shading, <0.1 = blue, 0.1 – 0.5 = green, 0.5 – 1.0 is yellow, and >1 = red.

ID	Sample Description	Sampling Time (min)	Respirable (mg/m³)	RCR	Inhalable (mg/m³)	RCR
E1	PAL - Mechanical Treatment Operator	292	<0.08	<0.08	0.46	0.09
E2	TER - Extrusion Operator (TR2)	280	0.23	0.23	0.66	0.13
E3	TET - Extrusion Operator (TR3)	320	0.76	0.76	3.19	0.64
E4	PER - Extrusion Supervisor and Micronization Operator	347	0.42	0.42	1.58	0.32

The higher reading for the extrusion operator manning the TR3 line is difficult to explain. It is possible that the micronization equipment has resulted in this increase, however this equipment was nearer to line TR2 than

[14] Shredding does tend to cause single digit temperature increases due to sheer forces.

TR3. Alternatively, since the dust was to a substantial part calcium (11 – 26%), it can potentially be partly explained by variation in powder handling technique in the compounding step (see Figure 7).

Lead and Cadmium

Cadmium and lead measurements in Plant E could not detect lead or cadmium with a limit of detection that was well below their occupational exposure limits in the CMRD (see Table 8 for results and Table 25 for raw measurement data).

Table 8 Results of the cadmium and lead in inhalable dust measurements in Plant E and Risk Characterisation Ratios (RCR) based on a limit of 1 µg/m³ for cadmium and 30 µg/m³ for lead. RCR colour coding applied: <LOD = no shading, <0.1 = blue, 0.1 – 0.5 = green, 0.5 – 1.0 is yellow, and >1 = red.

ID	Sample Description	Sampling Time (min)	Pb (µg/m³)	RCR	Cd (µg/m³)	RCR
E1	PAL - Mechanical Treatment Operator	292	<0.337	<0.011	<0.169	<0.17
E2	TER - Extrusion Operator (TR2)	280	<0.356	<0.012	<0.178	<0.18
E3	TET - Extrusion Operator (TR3)	320	<0.31	<0.01	<0.155	<0.16
E4	PER - Extrusion Supervisor and Micronization Operator	347	<0.287	<0.01	<0.144	<0.14

Organotin

No tin was detected in the inhalable dust fraction of plant E. As such all concentrations are calculated based on the limit of detection of the analytical method which was 100 ng/sample, which translates into various limits of detection for tin (range 0.14 – 0.18 µg/m³) dependent on the sampled air volume (see Table 9 for results and Table 25 for raw measurement data). The limit of detection was sufficiently low to conclude that the maximum RCRs are sufficiently below 1, indicating that workers in plant A are not at risk from organotin exposure.

Table 9 Results of the tin in inhalable dust measurements and calculation of maximum organotin concentrations in Plant E and Risk Characterisation Ratios (RCR) based on a limit of 25, 180, and 5750 µg/m³ for DOTE, DMTE, and MMTE. RCR colour coding applied: <LOD = no shading, <0.1 = blue, 0.1 – 0.5 = green, 0.5 – 1.0 is yellow, and >1 = red.

ID	Sample Description	Sn (µg/m³)	Max DOTE (µg/m³)	Max DMTE (µg/m³)	Max MMTE (µg/m³)	Max DOTE RCR	Max DMTE RCR	Max MMTE RCR
E1	PAL - Mechanical Treatment Operator	<0.17	<1.07	<0.79	<1.06	<0.04	<0.004	<0.0002
E2	TER - Extrusion Operator (TR2)	<0.18	<1.13	<0.83	<1.11	<0.05	<0.005	<0.0002
E3	TET - Extrusion Operator (TR3)	<0.15	<0.98	<0.72	<0.97	<0.04	<0.004	<0.0002
E4	PER - Extrusion Supervisor and Micronization Operator	<0.14	<0.91	<0.67	<0.9	<0.04	<0.004	<0.0002

Antimony and zinc

Traces of antimony were detected in the dust at levels far below the 250 µg Sb/m³ limit. Zinc was found at slightly higher concentrations but still well below the 500 µg Zn/m³ limit. No adverse impact on human health is to be expected from antimony or zinc exposure.

Table 10 Results of the antimony and zinc in inhalable dust measurements in Plant E and Risk Characterisation Ratios (RCR) based on a limit of 250 µg/m³ for antimony and 500 µg/m³ for zinc. RCR colour coding applied: <LOD = no shading, <0.1 = blue, 0.1 – 0.5 = green, 0.5 – 1.0 is yellow, and >1 = red.

ID	Sample Description	Sampling Time (min)	Sb (µg/m³)	RCR	Zn (µg/m³)	RCR
E1	PAL - Mechanical Treatment Operator	292	0.334	0.00134	<1.69	<0.003
E2	TER - Extrusion Operator (TR2)	280	0.327	0.00131	<1.78	<0.004
E3	TET - Extrusion Operator (TR3)	320	0.443	0.00177	14.78	0.030
E4	PER - Extrusion Supervisor and Micronization Operator	347	<0.14	<0.00056	3.53	0.007

Plasticisers

Since DMP and DEP are not plasticisers, there is no indication that these are used as additive in the flexible PVC value chain, there is no deliberate use in Plant E, and the calculated RCRs (see Table 30) are not concerning, these are not further evaluated here.

The absolute difference between pre and post shift estimated daily intake values is reported and visualised in Figure 12. To determine if there were statistically significant differences, Wilcoxon two sided signed-rank tests and paired two-sided t-tests were performed.

Post-shift estimated daily intakes of DiBP and DnDP were **lower** than pre-shift levels. Wilcoxon signed-rank tests indicated a significant decrease for DiBP ($W = 0$, $n = 8$, $p \leq 0.01$) and DnBP ($W = 4$, $n = 8$, $p \leq 0.1$). Paired t-tests on log-transformed concentrations yielded similar results for DiBP (geometric mean ratio = 0.71, 95% CI: 0.59-0.86; $p = 0.005$) and DnBP (geometric mean ratio = 0.72, 95% CI: 0.58-0.90; $p = 0.031$). Such results are congruent with the fact that these substances were used in rather moderate amounts in the past in industry and may not really be present in substantial amounts in recyclates. The main exposure route may be man-via-environment and the food may be an exposure source of larger relevance than work since the workers operate in shifts (6-14, 14-22, 22-6) and normally consume a large meal before coming to work and after work, but only take a short break/snack at work.

Post-shift estimated daily intakes of DEHP and DiNP were **higher** than pre-shift levels. The Wilcoxon signed-rank tests indicated a significant increase for DEHP ($W = 0$, $n = 8$, $p \leq 0.01$) and DiNP ($W = 0$, $n = 8$, $p \leq 0.01$). The paired t-tests on log-transformed concentrations yielded similar results for DEHP (geometric mean ratio = 2.04, 95% CI: 1.47-2.81; $p = 0.001$) and DiNP (geometric mean ratio = 1.82, 95% CI: 1.27-2.61; $p = 0.006$). There was a weak association indicating increased estimated daily intakes for DEHTP. The Wilcoxon signed-rank test indicated a significant increase ($W = 6$, $n = 8$, $p \leq 0.2$). The paired t-test on log-transformed concentrations yielded similar results (geometric mean ratio = 2.21, 95% CI: 0.65 – 7.45; $p = 0.177$). These results may be explained by the fact that DEHP was the dominant plasticiser for a period of decades prior to its regulation and can be present in the (post-consumer) input waste material. The primary alternatives for DEHP are DiNP and DEHTP, which are likely present in the (pre-consumer) input waste material. The lack of strong association for DEHTP was surprising as this substance is added by plant E in the compounding process.

Results for BBzP, DiDP, and DINCH were not statistically significant. BBzP was a niche plasticiser already during the time of DEHPs market dominance. DiDP and DINCH are alternatives to DEHP but are technically less suitable (e.g. lower plasticising effect) and more expensive than DiNP and DEHTP.

Statistical significance is not necessarily the same thing as relevance. The lower DiBP and DnBP values are at a low level at the start of the shift and consistently at a slightly lower level at the end of the shift. The average RCR for DiBP and DnBP decrease from 0.0024 to 0.0018 and from 0.0025 to 0.0019, respectively. A statistically significant finding with very low relevance. Similarly, the weak statistical significance of DEHTP does not make it irrelevant, for individual workers this plasticiser shows the greatest swing in estimated daily intake values interestingly in the mechanical treatment operator which is the furthest away from the extrusion processes occurring at elevated temperatures. As such the positions are discussed individually in a risk assessment framework. Since the anti-androgenic mixture risk assessment seems to provide the most sensitive endpoint, this is used primarily over individual substance risk assessments.

Table 11 Pre- and post-shift risk characterisation ratio for anti-androgenic effect (RCR_{AA})

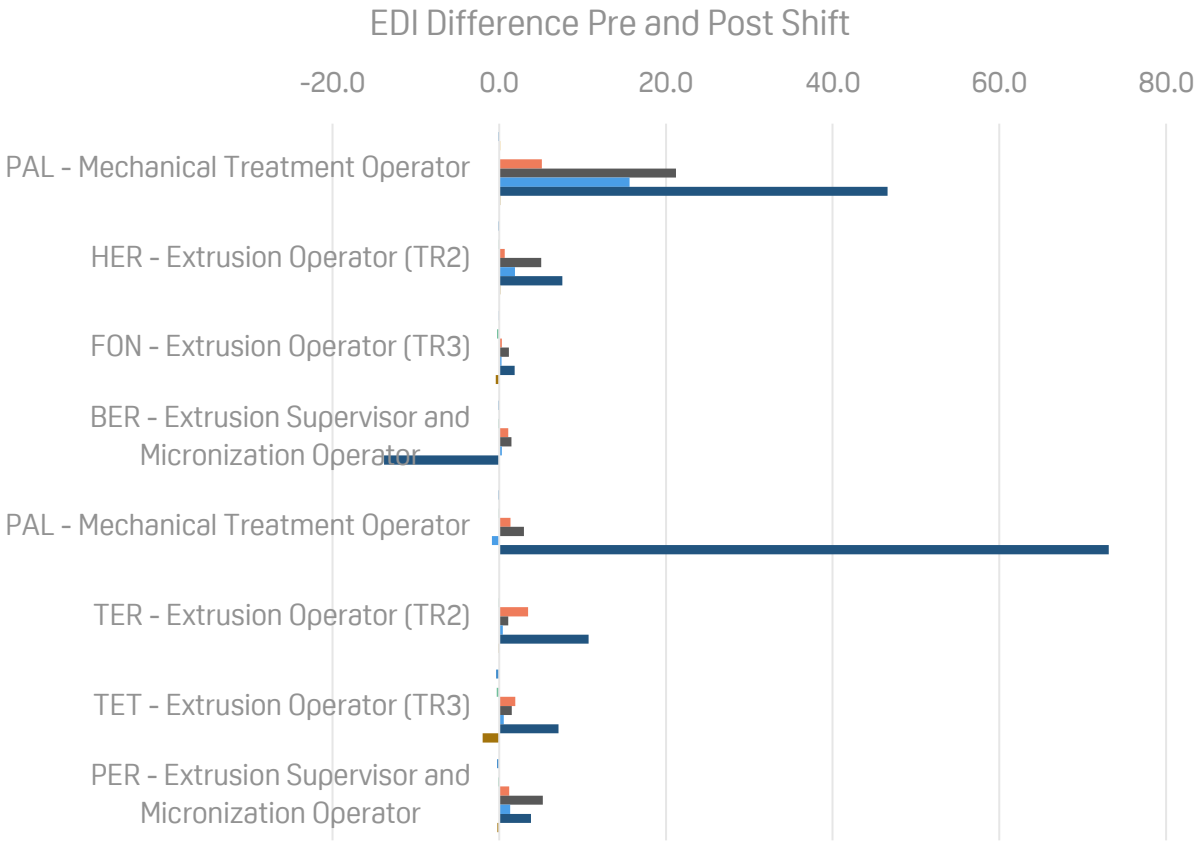
Worker Description	Shift	Pre-Sample-ID	Post-Sample-ID	Pre-Shift RCR_{AA}	Post-Shift RCR_{AA}	Change RCR_{AA}
PAL - Mechanical Treatment Operator	14-22	A01	A06	0.241	0.689	0.448
HER - Extrusion Operator (TR2)	14-22	A03	A08	0.192	0.260	0.068
FON - Extrusion Operator (TR3)	14-22	A04	A09	0.152	0.157	0.004
BER - Extrusion Supervisor and Micronization Operator	22-06	A05	A10	0.091	0.150	0.059
PAL - Mechanical Treatment Operator	06-14	A11	A16	0.440	0.528	0.088
TER - Extrusion Operator (TR2)	06-14	A13	A18	0.131	0.306	0.176
TET - Extrusion Operator (TR3)	06-14	A14	A19	0.192	0.275	0.083
PER - Extrusion Supervisor and Micronization Operator	06-14	A15	A20	0.096	0.188	0.092

Counterintuitively the mechanical treatment operator saw the largest swing in EDI values for plasticisers (mainly DEHTP) and had the highest post shift RCR_{AA} and in one of the shifts the greatest change in the RCR_{AA}. This is odd as the mechanical treatment processes tend to be at ambient temperature and one would expect greater emission from the material at the extrusion with melt filtration workstations. However, here it should be noted that in both cases the worker involved (PAL) operated in both shifts and exposure outside of the workplace may have contributed to this finding. Next to this it should be considered that the somewhat open shredding process and the entirely boxed grinding process are equipped with an air extraction system that emits filtered air back into the mechanical treatment hall. While the sheer forces in grinding do not cause melting of the material, it can at sheer points become warm. It is possible, that the filtering system employed focuses primarily on particles and would not (substantially) remove vapours. If so, this could cause elevated plasticiser vapour concentrations in the mechanical treatment hall.

Alternatively, the mechanical treatment operator was observed unpacking waste during his shift by hand without gloves. Biomonitoring does not allow one to differentiate whether exposure was caused by inhalation or dermal contact. As such dermal transfer of minute amounts of plasticiser from the handling of material to skin could have contributed to the increased measured metabolites in the urine of the worker.

The activities in the extrusion/micronization hall do seem to cause increases in exposure of workers to plasticisers, albeit at lower rates than in the mechanical treatment hall. This is hardly surprising since plasticisers are in integral part of the flexible PVC value chain and thus present in the waste input material and DEHTP is used directly in formulations by plant E. Vapour and dust emissions are extracted through specifically engineered extraction systems at the relevant points in the process. The only source that may contribute to exposure that is not under extraction ventilation are the big bags for filter cakes and spent filters. The relevance of this cannot be quantified based on the data generated.

Regardless all post shift RCR_{AA} were below 1, which indicates that these workers are not exposed to levels that would cause an unacceptable level of risk for the working age population.



	PER - Extrusion Supervisor and Micronization Operator	TET - Extrusion Operator (TR3)	TER - Extrusion Operator (TR2)	PAL - Mechanical Treatment Operator	BER - Extrusion Supervisor and Micronization Operator	FON - Extrusion Operator (TR3)	HER - Extrusion Operator (TR2)	PAL - Mechanical Treatment Operator
DiBP	-0.3	-0.4	0.0	-0.1	-0.1	-0.1	-0.2	-0.1
BBzP	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1
DnBP	-0.2	-0.3	-0.1	-0.1	-0.1	-0.3	-0.1	0.1
DEHP	1.2	1.9	3.4	1.3	1.1	0.3	0.6	5.1
DiNP	5.2	1.5	1.1	3.0	1.4	1.2	5.1	21.2
DiDP	1.3	0.6	0.4	-0.9	0.3	0.3	1.9	15.6
DEHTP	3.8	7.1	10.7	73.1	-13.8	1.8	7.6	46.6
DINCH	-0.2	-2.0	-0.1	0.0	0.0	-0.4	0.1	0.1

DiBP BBzP DnBP DEHP DiNP DiDP DEHTP DINCH

Figure 12 Differences in EDI of different plasticisers in workers in plant E.

Environmental Emissions

All processes occur indoor and are performed under engineered air extraction ventilation. There are three distinct air streams: 1) air extracted from the shredding and grinding process cleaned and emitted into the plant, 2) air extracted from the micronization equipment that passes through a cyclone and subsequently a particle filter system before being emitted, and 3) air extracted from the extrusion equipment that passes through an activated carbon filter system before being emitted (see Figure 13).

Figure 13 Channelled air emission systems Plant E. Extraction system to the left is equipped with a dust envelope filtering system. The extraction system to the right is equipped with carbon filters.

Plant E was able to provide an emission measurement report for each stack. During normal operation of the micronization equipment the mass flow remains below 0.0003 kg/h. The measurement report of the extrusion line emissions mentions that during full operation of all three extrusion lines the dust emissions are 0.014 kg/h and total VOC emissions were 0.0139 kg/h.

Table 12 Plant E emission measurement results

Stack	Micronization	Extrusion	
Parameter	Dust	Dust	VOCs
Airflow (m^3/h)	692		14635
Concentration (mg/m^3)	<0.5	0.937	0.91
Mass Flow (kg/h)	<0.0003	0.014	0.0139

The plant processes 1.5 – 2.5 tons of material per hour the fraction of solid material emitted to air by channelled emissions is between 0.0006 and 0.0009%. VOCs emitted account of a similar fraction of material. In total, the emission rate is between 0.0011 and 0.0019%.

In addition, it is worth noting that based on the emission measurement reports it could be deduced that plant E has emission limits in their permits of 0.030 kg solids/h for the micronization stack and 0.180 kg solids/h and 0.360 kg VOC/h for their extrusion stack. As such local legislation/permitting regimes put a hard limit of 0.023 – 0.038% on the emissions the plant is allowed to produce.

PLANT F

Plant F specialises in the recycling of flexible PVC flooring materials. The plant treats mainly pre-consumer material, but on the day of the measurement campaign operated solely on post-consumer input materials. In total it recycles between 500 and 5000 tons of flexible PVC per year.

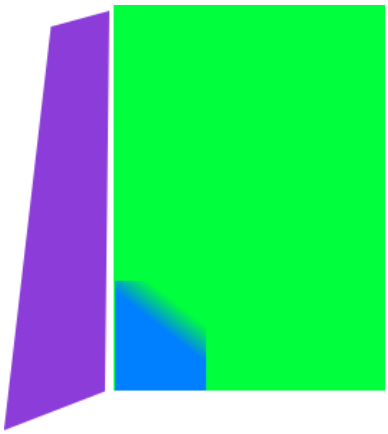


Figure 14 Layout of Plant F. Blue is raw material receipt, green is mechanical treatment/sorting, purple is an outdoor storage area for waste material and big bags of finished goods.

Waste material can arrive in packaged form or in bulk. When received packaged it can be stored outside for a period of time and when received in bulk it is deposited in the building hall (Figure 15).

Figure 15 Left outdoor material storage area. Right: bulk delivery of flooring materials.

In a first step, material is manually sorted. This sorting is particularly relevant for post-consumer material to ensure no asbestos containing flooring is introduced into the recycling process. Material is unloaded onto a hand sorting table from which it is sorted into a rejects bin (blue in Figure 16) or placed on a conveyer feeding a shredder (Figure 16), which chips the material to a size of around 3 cm.

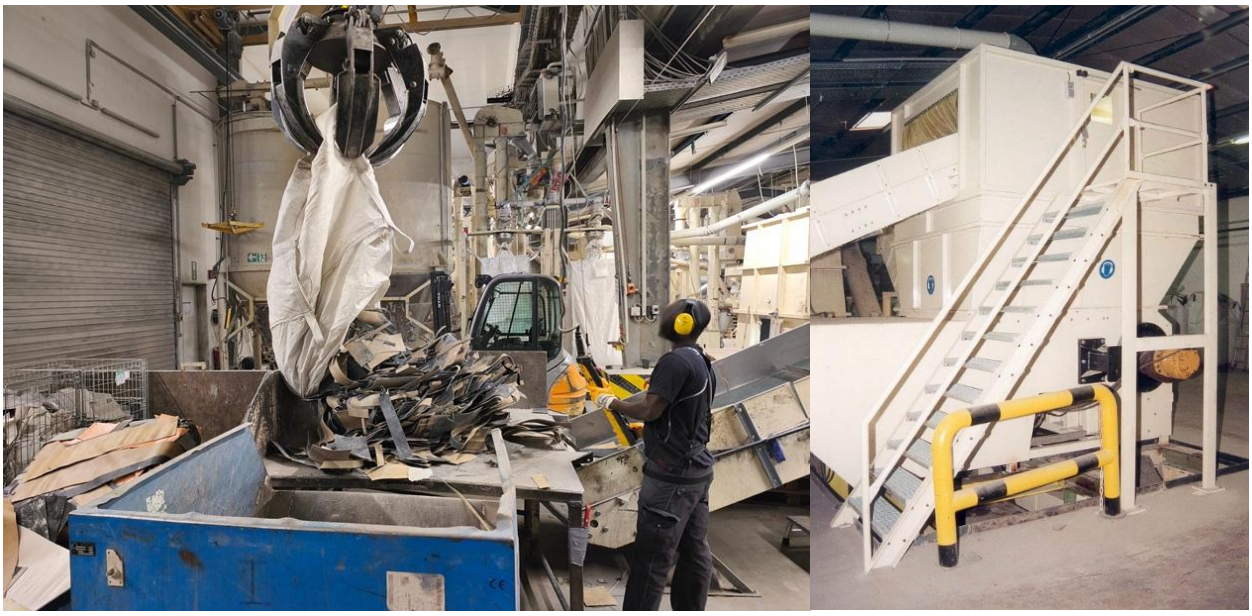


Figure 16 Left: Unloading of packaged waste onto manual sorting table. Right: Shredder

From the shredder, the material is treated in a hammer mill which causes glue and mineral (e.g. mortar) contamination that is annealed to the chips to fracture to a finer powder-like material. Following this, the material stream is subjected to sieving and wind sifting to remove the powder-like material as well as a metal separation step to remove metallic contamination (e.g. nails). The resulting material is principally composed of flexible flooring (Figure 17).

Figure 17 Left: Wind sifter. Middle: metal separation. Right: resulting flexible flooring material.

The flexible flooring material is subsequently subjected to cryogenic grinding (Figure 18 Left). Under cryogenic conditions, the flexible PVC becomes brittle, allowing it to be micronized to a fine powder. Other constituents of the chips of flexible flooring material obtain a different size distribution allowing subsequent separation by an industrial sieve (Figure 18 Right).

Figure 18 Left: cryogenic grinder. Right: industrial sieve.

The primary waste output of the sieving step is PET (or other polyester) fibre that is sometimes used in flooring to reinforce the structure of the material (see Figure 19 left). While the primary recycle output is micronized flexible PVC (Figure 19 right); which is automatically transported from the sieve into a silo from which it is transported to a big bag. The sealed big bags are stored until expedition to plastics converters.

Figure 19 Left: PET fibres making up the bulk of the waste output from the sieving step. Right: micronized flexible PVC recyclate.

Risk Assessment for Human Health

Plant F normally has 2 – 3 workers operating the plant:

- 1 Raw Materials (RM) Handling and Sorting Operator
- 1 Micronization Operator
- 1 Supervisor

The RM Handling and Sorting Operator collects waste material, unloads this onto the sorting table, and feeds the conveyer that leads to the shredder. He also takes care of the collection of bins into which various waste products flow from the line (e.g. minerals from sieve and wind sifter) and metal from metal separator.

The micronization operator takes care of all the operations that occur following the micronization steps which includes: the collection of containers into which waste product are collected (e.g. PET fibre), connection of big bags for filling of recyclate, and removal of big bags filled with recyclate.

The supervisor normally spends most of his time (80%) in an office environment attached to the plant. He takes over for the other operators when they are on a break and is responsible for any troubleshooting on the line with takes him into the production hall for a minority of the time (20%). On days when there is either no RM Handler and Sorting operator or micronization operator, the supervisor steps in to take this role.

On the day of the measurement 3 workers were operating the plant and all were included in the measurement campaign.

The plant was also able to provide a historical measurement report from 2002, where over the course of several days various static and personal respirable and inhalable dust samples were collected. Some of the inhalable dust samples were further analysed to quantify lead and cadmium. See Table 13.

Table 13 Historical measurement results of Plant F. <LoD = below limit of detection; NA = not available (i.e. not measured).

Sample Description	Type	Day	Sampling Time (min)	Respirable Dust (mg/m³)	Inhalable Dust (mg/m³)	Lead (µg/m³)	Cadmium (µg/m³)
Unloading Station	Static	1	205	<LoD	0.211	NA	NA
Unloading Station	Static	1	232	<LoD	0.305	0.11	0.02
Unloading Station	Static	2	353	<LoD	0.265	0.18	0.03
Unloading Station	Static	3	598	<LoD	0.166	NA	NA
Unloading Station	Static	4	255	<LoD	0.463	0.12	0.02
Sorting Line	Static	1	201	NA	0.125	NA	NA
Sorting Line	Static	1	232	NA	0.190	NA	NA
Sorting Line	Static	2	353	NA	0.172	NA	NA
RM Handling Operator	Personal	1	182	<LoD	<LoD	NA	NA
RM Handling Operator	Personal	1	205	0.249	<LoD	NA	NA
RM Handling Operator	Personal	2	166	<LoD	0.611	1.64	<LoD
RM Handling Operator	Personal	2	227	0.318	0.573	1.63	<LoD
RM Handling Operator	Personal	4	206	<LoD	<LoD	NA	NA
Grinding Station	Static	3	354	NA	0.108	NA	NA
Grinding Station	Static	4	241	NA	0.550	0.20	0.02

Dust

Dust measurements in Plant F demonstrated that all workers have exposures lower than the limits used in this report (see Table 14 and Table 25 for raw measurement data). The measured values for respirable and inhalable dust were largely in line with the results of the 2002 measurement campaign indicating that there has not been a deterioration of exposure controls.

Table 14 Results of the respirable and inhalable dust measurements in Plant F and Risk Characterisation Ratios (RCR) based on a limit of 1 mg/m³ for respirable and 5 mg/m³ for inhalable dust. RCR colour coding applied: <LOD = no shading, <0.1 = blue, 0.1 – 0.5 = green, 0.5 – 1.0 is yellow, and >1 = red.

ID	Sample Description	Sampling Time (min)	Respirable (mg/m³)	Respirable RCR	Inhalable (mg/m³)	Inhalable RCR
F1	CK - RM Handling / Sorting Operator	210	0.28	0.28	1.04	0.21
F2	BF - Micronization Operator	210	<0.11	<0.11	0.59	0.12
F3	DS - Supervisor	210	<0.11	<0.11	0.20	0.04

Lead and Cadmium

The lead and cadmium measurements in Plant F are below the EU OELs and thus the plant is compliant (Table 15). The lead exposure of workers in the production hall may have reduced over time since the 2002 measurements revealed concentrations of 1.6 µg/m³, whereas the current data is below 0.45 µg/m³. This could potentially be due to the stocks of lead containing post-consumer flooring becoming less over time.

There is no explanation for the unexpectedly high measurement of 3 µg/m³ of the supervisor that spends 80% of its time in the adjoining office. However, we note that this is still a factor 10 below the OEL.

Table 15 Results of the cadmium and lead in inhalable dust measurements in Plant F and Risk Characterisation Ratios (RCR) based on a limit of 1 µg/m³ for cadmium and 30 µg/m³ for lead. RCR colour coding applied: <LOD = no shading, <0.1 = blue, 0.1 – 0.5 = green, 0.5 – 1.0 is yellow, and >1 = red.

ID	Sample Description	Sampling Time (min)	Pb (µg/m³)	RCR	Cd (µg/m³)	RCR
F1	CK - RM Handling / Sorting Operator	210	<0.451	<0.015	<0.226	<0.23
F2	BF - Micronization Operator	210	<0.454	<0.015	<0.227	<0.23
F3	DS - Supervisor	210	2.995	0.100	<0.225	<0.23

Organotin

No airborne tin was detected in Plant F and all max RCRs are calculated based on the limit of detection of tin. The potential for these substances to cause harm to workers in Plant F can be excluded.

Table 16 Results of the tin in inhalable dust measurements and calculation of maximum organotin concentrations in Plant F and Risk Characterisation Ratios (RCR) based on a limit of 25, 180, and 5750 µg/m³ for DOTE, DMTE, and MMTE. RCR colour coding applied: <LOD = no shading, <0.1 = blue, 0.1 – 0.5 = green, 0.5 – 1.0 is yellow, and >1 = red.

ID	Sample Description	Sn (µg/m³)	Max DOTE (µg/m³)	Max DMTE (µg/m³)	Max MMTE (µg/m³)	Max DOTE RCR	Max DMTE RCR	Max MMTE RCR
F1	CK - RM Handling / Sorting Operator	<0.23	<1.43	<1.06	<1.41	<0.06	<0.006	<0.0002
F2	BF - Micronization Operator	<0.23	<1.44	<1.06	<1.42	<0.06	<0.006	<0.0002
F3	DS - Supervisor	<0.23	<1.43	<1.05	<1.41	<0.06	<0.006	<0.0002

Antimony and Zinc

Airborne antimony was not detected in Plant F, which is congruent with the understanding that there is no need for the flame retardant synergist antimony trioxide in flooring formulations. Zinc was detected albeit at a very low concentration and well below the limit maintained for this report.

Table 17 Results of the antimony and zinc in inhalable dust measurements in Plant F and Risk Characterisation Ratios (RCR) based on a limit of 250 µg/m³ for antimony and 500 µg/m³ for zinc. RCR colour coding applied: <LOD = no shading, <0.1 = blue, 0.1 – 0.5 = green, 0.5 – 1.0 is yellow, and >1 = red.

ID	Sample Description	Sampling Time (min)	Sb (µg/m³)	RCR	Zn (µg/m³)	RCR
F1	CK - RM Handling / Sorting Operator	210	<0.23	<0.00092	<2.26	<0.005
F2	BF - Micronization Operator	210	<0.23	<0.00092	2.68	0.005
F3	DS - Supervisor	210	<0.23	<0.00092	<2.25	<0.005

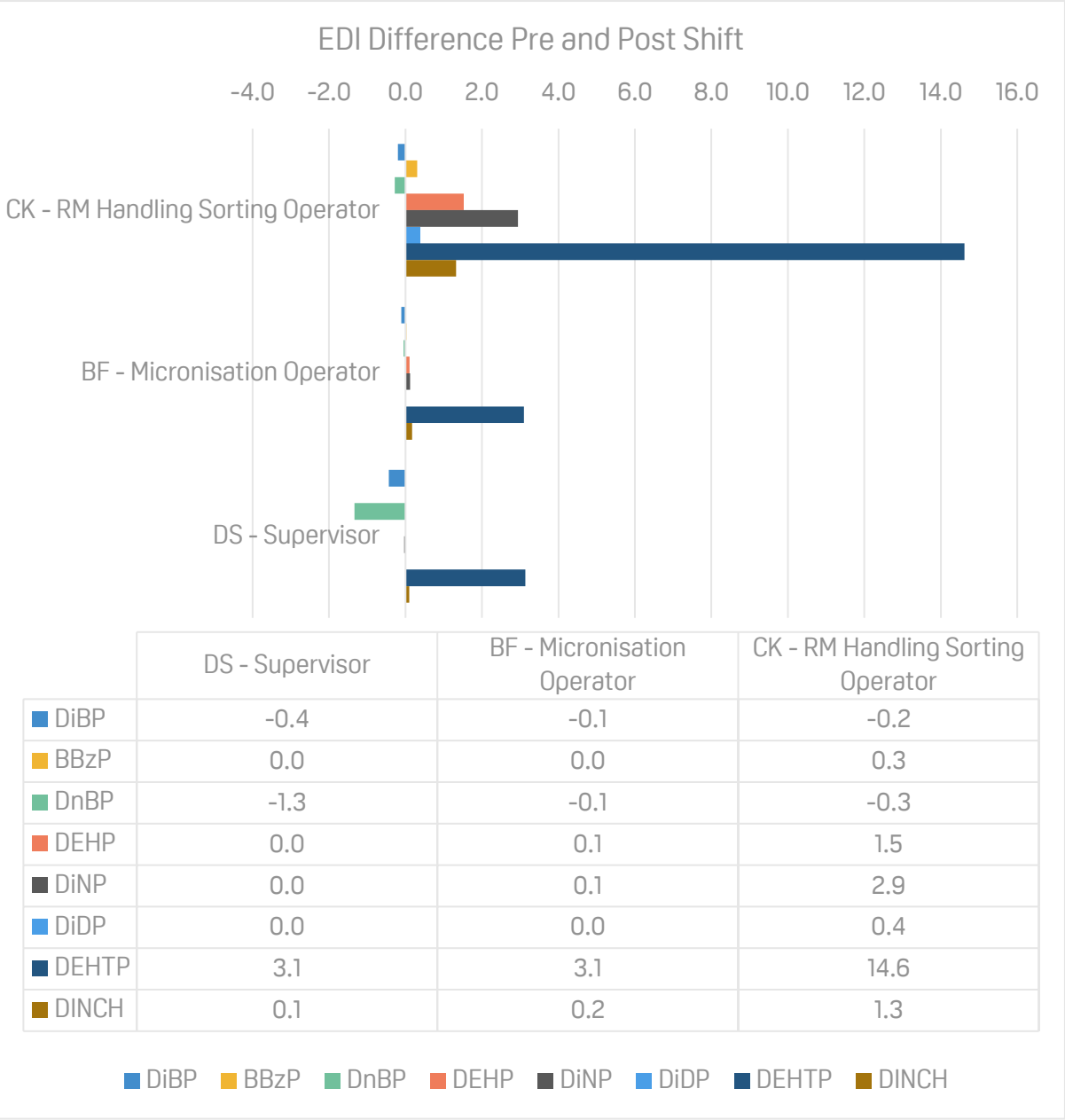
Plasticisers

Since DMP and DEP are not plasticisers, there is no indication that these are used as additive in the flexible PVC value chain, there is no deliberate use in plant F, and the calculated RCRs (see Table 30) are not concerning, these are not further evaluated here.

The absolute difference between pre and post shift estimated daily intake values is reported and visualised in Figure 20. Statistical analysis with 3 paired samples would be inappropriate.

However, it is noted that the greatest shift in EDIs is observed with the RM Handling and Sorting Operator, which is consistent, given the fact that this position involves the greatest proximity and dermal contact with the flooring material.

Another observation is that the main substance for which there seems to be an exposure is DEHTP. This is congruent with the fact that this substance has become the dominant plasticiser since the phase down/out of DEHP. Since the plant was treating mainly post-consumer material on the day of the measurement, it could be that this shift has occurred long enough ago that it is now starting to affect the composition of post-consumer flooring materials.



	DS - Supervisor	BF - Micronisation Operator	CK - RM Handling Sorting Operator
DiBP	-0.4	-0.1	-0.2
BBzP	0.0	0.0	0.3
DnBP	-1.3	-0.1	-0.3
DEHP	0.0	0.1	1.5
DiNP	0.0	0.1	2.9
DiDP	0.0	0.0	0.4
DEHTP	3.1	3.1	14.6
DINCH	0.1	0.2	1.3

Figure 20 Differences in EDI of different plasticisers in workers in plant F.

In terms of the mixture risk assessment for anti-androgenic effect, the calculated post shift RCR_{AA} are very low (Table 18) indicating workers are not exposed to levels that could give rise to concern for anti-androgenic effects. They are relatively lower compared to plant E (see Table 11). This is consistent with the fact that the processes in Plant F are all conducted at ambient or lower temperatures, meaning that little plasticiser will volatilise from the material and exposure would be through dust. Firstly, only the plasticiser contained in the respirable dust would be absorbed for 100% since the inhalable fraction would be cleared through the mucociliary pathway and ingested orally. The plasticiser contained in the dust is still encapsulated in a polymer matrix limiting the release and subsequent absorption. Furthermore, plant F, while producing a very fine powder as an output of its process, seems to have excellent dust control systems in their plant as is evidence by the inhalable and respirable dust measurements (Table 14).

Table 18 Pre- and post-shift risk characterisation ratio for anti-androgenic effect (RCR_{AA}) in plant F.

Worker Description	Shift	Pre-Sample-ID	Post-Sample-ID	Pre-Shift RCR_{AA}	Post-Shift RCR_{AA}	Change RCR_{AA}
CK - RM Handling Sorting	6-14	C01	C04	0.099	0.194	0.094
BF - Micronisation	6-14	C02	C05	0.034	0.037	0.003
DS - Supervisor	6-14	C03	C06	0.162	0.060	-0.103

Environmental Emissions

All treatment of waste material occurs indoors in plant F (i.e. there is no outdoor shredding) and all equipment used to process the flooring material is connected to a centralised air extraction and cleaning system. The dust removal cleaning systems are similar to the ones observed in rigid PVC recycling facilities and should be expected to be similarly performant meaning the process emission will be in the order of magnitude of grams per ton processed.

CONCLUSIONS

Workplace exposure

Dust

Measurements revealed that airborne concentrations of dust tend to be well below the limit used in this report of 5 mg/m³ for inhalable and 1 mg/m³ for respirable dust.

Calculated summary statistics for inhalable and respirable exposure in flexible PVC recycling facilities can be found in Table 19.

Table 19 Summary Statistics for Inhalable and Respirable Dust Exposure (in mg/m³) in Flexible PVC Recycling Facilities. For datapoints where exposure was below the limit of detection, the limit of detection was used to calculate the summary statistics.

	Inhalable	Respirable
n	7	7
25th Percentile	0.5	0.11
Median	0.7	0.23
75th Percentile	1.3	0.35
90th Percentile	2.2	0.55

In future, this data can be used to make reasonable worst-case estimates of exposure to substances used in flexible PVC. Not by using the figures itself as estimates (that would be unreasonable worst case), but by assuming a certain additive use rate. For example, if "Substance X" was used in cable formulations at a rate of at most 10% and there is an estimate that it was used in 20 – 30% of cables historically, then one can calculate a reasonable worst case estimate for workplace exposure in cable recycling plants based on the 90th percentile for inhalable dust: 2.2 [mg/m³] x 0.1 x 0.30 = 0.066 mg/m³ (or 66 µg/m³).

Lead and Cadmium

The airborne concentration lead and cadmium well below the occupational exposure limit of 30 and 1 µg/m³, respectively.

Table 20 Summary Statistics for Lead and Cadmium Exposure (in µg/m³) in Flexible PVC Recycling Facilities. For datapoints where exposure was below the limit of detection, the limit of detection was used to calculate the summary statistics.

	Lead	Cadmium
n	7	7
25th Percentile	0.3	0.16
Median	0.4	0.18
75th Percentile	0.5	0.23
90th Percentile	1.5	0.23

Next to the airborne limit of 30 µg/m³, a biological limit value for lead has been established in the Carcinogens, Mutagens, and Reproductive Toxicants Directive (CMRD) of 30 µg Pb/100 ml blood until 31 December 2028

followed by a limit of 15 µg Pb/100 ml blood starting 1 January 2029. With some transitional provisions that indicate that if limits are exceeded, but a downward trend exits workers may continue to work.

Furthermore, the CMRD indicates that if airborne exposure is greater than 0.015 mg/m³ [=15 µg/m³] or lead blood levels are greater than 9 µg Pb/100 ml blood there should be medical surveillance of the workers. For female workers of childbearing age there is an additional limit of 4.5 µg Pb/100 ml blood above which medical surveillance is required.

In a 2016 study performed by the Vinyl Recycling Consortium, an ad-hoc consortium of recycling and downstream converting companies managed by Polymer Comply Europe, together with Dr. Claudia Fruijtier-Pölloth ERT of CATS Consulting GmbH (Fruijtier-Pölloth 2016), whole blood measurements were performed in 127 workers in the PVC recycling and converting value chain. The results showed that for the 100 workers for which individual results were available that the lead blood levels were 4.4 µg Pb/100 ml blood on average with a range of 0.8 – 13.0 µg Pb/100 ml blood and a 90th percentile of 8.8. This data indicates that already in 2016, the lead blood limits of 2029 were not exceeded in the PVC recycling value chain.

Whether or not medical surveillance will need to be performed by PVC recycling facilities will depend entirely on the lead blood levels of their workers since the 15 µg/m³ airborne exposure trigger limit is not breached. It is likely that quite a few plants have a workforce where the blood lead level remains below 9 µg Pb/100 ml blood and below 4.5 µg Pb/100 ml blood in female employees of childbearing age.

Furthermore, any lead (and cadmium) present in PVC powder would have limited biological availability. A large fraction of the inhaled dust will have a particle size distribution that is deposited in the upper respiratory tract from which it is cleared by mucociliary clearance and subsequently ingested (Antunes and Cohen 2007). The release of substances from a polymer matrix has been extensively investigated (Schwope 1990; Piringer and Baner 2008) and will limit release of the lead ions as the dust passes the gastrointestinal tract. Only the respirable fraction of the dust, which deposits in the lower respiratory tract and is thus not subject to mucociliary clearance should be expected to be absorbed completely. As the exposure levels to respirable dust are limited in flexible PVC recycling, it is questionable whether the lead in PVC dust exposure will meaningfully contribute to lead blood levels.

Organotins

As explained in the methods section on Organotins, only tin was measured in the inhalable dust fraction. In none of the samples was tin detected with a limit of detection of 100 ng/sample, which dependent on the volume of air captured in the measurement translates to limits of detection for tin in air of 0.14 – 0.23 µg Sn/m³. These LoD values were used to calculate maximum concentrations of different organotin substances assuming that all tin is caused by the individual organotin. In reality, if tin is detected it will likely be due to the presence of various species of (organo)tins that have been used in PVC and potentially other sources of tin in the airborne dust. As such it is an inherently conservative worst-case approach.

The conservativeness of the approach is compounded by the fact that phase out of the substance with the lowest DNEL DOTE in favour of MOTE and other organotin species, has been underway for decades. Furthermore, in B&C applications historically there has been greater reliance on methyl tin stabilisers. As such any conclusions based on maximum DOTE concentrations should be considered extreme worst-case.

The approach is however much more practical. Quantifying organotin in the inhalable fraction directly is much more laborious, analytically difficult, associated with greater error margins, and greater cost than quantification of tin by ICP-MS. Furthermore, by using ICP-MS a range of other elements can be quantified at little to no additional laboratory effort and thus cost. This enables simultaneous investigation into lead and cadmium OEL compliance for example.

There is however a downside to the use of this method. If results would have shown that the calculated maximum concentration of an organotin is above its threshold, further investigation would need to be performed to determine the actual level of organotin as one cannot conclude that the plant is in non-compliance based on the maximum organotin concentration alone. Such further investigation could be done by either:

1. performing another inhalation exposure measurement campaign with a filter for inhalable dust and sending the filter for organotin quantification, or
2. taking a sample of recyclate and sending this for organotin quantification to establish the ratio of the different organotins in the recyclate and recalculating the organotin concentration in the indoor dust based on this ratio.

The second option would be considerably more economical since it would be 1 analysis instead of several. Furthermore, from an analytical chemistry perspective, it tends to be easier and much more precise to quantify a concentration in a larger quantity of sample (e.g. 10 grams of recyclate) than in a smaller quantity (e.g. 0.3 mg of inhalable dust on a glass fibre filter).

There is of course a third alternative path for such hypothetical cases where the maximum organotin concentrations exceed the limit values. One could implement more risk management measures to reduce the exposure to tin to reduce the calculated maximum organotin exposure.

Regardless, the summary statistics for measured tin exposure and calculated maximum organotin exposure in flexible PVC recycling facilities can be found in Table 21.

Table 21 Summary Statistics for Tin Exposure (in µg/m³) and calculated maximum organotin concentrations in Flexible PVC Recycling Facilities (highlighted for clarity; see text for explanation). For datapoints where exposure was below the limit of detection, the limit of detection was used to calculate the summary statistics.

	Sn	Max DOTE	Max DMTE	Max MMTE
n	7			
25th Percentile	0.2	1.0	0.7	1.0
Median	0.2	1.1	0.8	1.1
75th Percentile	0.2	1.5	1.1	1.4
90th Percentile	0.2	1.5	1.1	1.4

When the 90th percentile concentrations of DOTE, DMTE, and MMTE of 1.5, 1.1, 1.4 µg/m³ are compared to the DNELs used in the ECHA investigation report of 25, 180, and 5750 µg/m³ in can be concluded that workers in flexible PVC recycling are operating safely.

It should however be noted that there is uncertainty with regards to the toxicological properties of the organotin substances. For example, it used to be understood that dialkyl bis-alkylthio esters would degrade to a dialkyl dichloride metabolites. Toxicological studies of dialkyl dichlorides have indicated reproductive toxicant properties and thus it was presumed that the dialkyl bis-alkylthio esters would be reproductive toxicants as well. It is the basis for their harmonised classification (and thus in some case SVHC identification).

New evidence however suggests that dialkyl bis-alkylthio esters do not metabolise to dialkyl dichloride and new reproductive toxicity studies of DOTE, DBTE, DMTE show no developmental toxicity (Costlow et al. 2021; Kirf et al. 2023; Costlow, Nasshan, and Frenkel 2017). These studies do however show a common maternal

toxicity effect in the thymus of the experimental animals for which the rabbit seems to be less susceptible than rats and mice. It is possible that such findings can impact further investigation into an occupational exposure limit currently being conducted by the European Chemicals Agency may result in new insights that could result in new occupational exposure limits that may be higher or lower than the currently used DNELs.

Antimony, Zinc, Titanium, and Calcium

As explained in the individual plant chapters, exposure to antimony and zinc is controlled to below their respective limit values. No risk assessment is performed for titanium and calcium given the relatively limited toxicological hazard of these potential associated molecules (i.e. titanium dioxide, calcium carbonate, and calcium fatty acid salts). Summary statistics for exposure to these elements can be found in Table 22.

Table 22 Summary Statistics for Antimony, Zinc, Titanium and Calcium Exposure (in µg/m³) in Flexible PVC Recycling Facilities. An assigned protection factor of 10 was used for those measurements where RPE is worn. For datapoints where exposure was below the limit of detection, the limit of detection was used to calculate the summary statistics.

	Antimony	Zinc	Titanium	Calcium
n	7	7	7	7
25th Percentile	0.23	2.3	0.68	173
Median	0.33	3.1	0.75	311
75th Percentile	0.38	8.0	0.97	372
90th Percentile	0.23	2.0	0.68	89

Of some note here is that antimony was found in 3 of the inhalable dust samples. As alluded to in the Antimony, Calcium, Zinc, and Titanium section of the methods chapter, antimony was included for its potential presence due to the use of antimony trioxide (ATO) as a synergist to the halogenated flame retardant action of the chlorine of the PVC polymer. It was expected that ATO may be present in certain flexible PVC (cable) wastes. The concentration of the antimony was in the inhalable dust was just 0.046% and thus only slightly higher than in rigid PVC 0.023%, where no ATO was expected since rigid PVC is self-extinguishing[15]. It is possible the input waste was either not cable waste or made of cables not requiring very high levels of flame retardancy (e.g. low voltage cables or data cables).

Biomonitoring results

As demonstrated in the plant chapters, the risk characterisation ratio for each individual substance is below the toxicological limit value and even the much more sensitive sum-RCR for anti-androgenic effect remained below 1 indicating that workers are operating safely. These RCRs were calculated based on post-shift EDI concentrations, which combine exposure obtained outside of the workplace (e.g. through food) and exposure at the workplace.

To determine whether there is workplace exposure to these substances, the paired pre- and post- shift samples for which the EDI estimation was based fully on measured metabolite concentration above the respective limits of detection, were subtracted. As such a change in EDI during the period at work can be calculated. This approach did not allow for the calculation of EDI changes for DnPrP, DMoxyEP, DiPeP, DnPeP, DnHexP, DiHepP, and DnHepP, as for those substances there was not a single pair where either or both pairs

[15] ATO use is limited to some specific flexible PVC formulations as the addition of normal plasticiser dilutes the chlorine content and therefore decreases the inherent flame retardancy. Furthermore, in most flexible PVC applications flame retardancy is not needed and ATO is not used. Only in applications requiring flame retardancy to reach a high level (e.g. certain types of electrical cables), would ATO be used.

were not below the limit of detection. To determine whether there are significant differences between pre- and post-shift measurements, Wilcoxon signed-rank tests were performed (Table 23).

For the solvent ortho-phthalates DMP and DEP, no change is observed between pre- and post-shift EDIs. This is congruent with the fact that DMP and DEP are not used in flexible PVC as plasticiser nor are they used on the premises of the recyclers directly.

Of the ortho-phthalate plasticisers, a significant decrease in EDI is observed for DnBP and DiBP, indicating that exposure decreased during the period at work. This may indicate that these plasticisers are no longer or no longer substantially present in post-consumer PVC waste and that the main exposure pathway is through other sources such as food. Food as an exposure source would make sense since (flexible PVC) recycling facilities tend to work based on shift times which tend to be 6-14, 14-22, and 22-6. Workers working on such timings tend to eat larger meals before and after the shift and only have a smaller intake during the mid-shift break.

An increase in the EDI values for the ortho-phthalate plasticisers DEHP, DiNP as well as the alternative plasticiser DEHTP is observed, which is congruent with the fact that DEHP was the dominant plasticiser in the past and DiNP and DEHTP are the dominant substitutes for DEHP.

No significant change is observed in the change in EDI value for BBzP, DiDP, and DINCH (based on an alfa of 0.05). For BBzP this likely has a similar background to DnBP and DiBP: while it was used in the past it was by far not a dominant plasticiser meaning that its presence in post-consumer waste is likely to be low. For DiDP and DINCH these are alternatives to DEHP that did not become the dominant alternatives given lower plasticising efficacy, PVC compatibility, and price levels. They have roles to play in more niche markets where some of their properties may be beneficial, for example their lower vapour pressure may make them more suitable for higher temperature environments and/or requirements[16].

Table 23 Change in EDI in pre- and post-shift paired samples for ortho-phthalates solvents (gray shaded) ortho-pthalate plasticizers (yellow shade) and alternative plasticisers (orange shade). Data only calculated for paired samples where both pre- and post- measurements were >LoD. p calculated using Wilcoxon signed-rank tests.

Paired Samples	DMP	DEP	DiBP	BBzP	DnBP	DEHP	DiNP	DiDP		DEHTP	DINCH
A06 - A01	0.039	-0.590	-0.144	0.123	0.099	5.099	21.209	15.630		46.571	0.129
A08 - A03	-0.002	-0.781	-0.160	0.001	-0.084	0.647	5.054	1.891		7.578	0.115
A09 - A04	-0.016	-3.246	-0.068	-0.025	-0.262	0.313	1.161	0.261		1.841	-0.400
A10 - A05	-0.014	-0.112	-0.140	0.034	-0.120	1.093	1.447	0.315		-13.816	-0.011
A16 - A11	0.046	0.160	-0.131	0.019	-0.059	1.336	2.971	-0.860		73.110	0.043
A18 - A13	0.008	-0.098	-0.025	-0.004	-0.074	3.449	1.081	0.445		10.730	-0.086
A19 - A14	0.016	8.573	-0.387	-0.044	-0.308	1.937	1.513	0.560		7.097	-1.996
A20 - A15	-0.029	-8.071	-0.262	0.009	-0.157	1.210	5.239	1.317		3.814	-0.212
C04 - C01	-0.045	5.074	-0.197	0.306	-0.282	1.521	2.942	0.396		14.618	1.323
C05 - C02		-0.132	-0.111	0.027	-0.054	0.104	0.117			3.097	0.169
C06 - C03		-0.300	-0.436	0.005	-1.338		-0.038			3.133	0.096
n	9	11	11	11	11	10	11	9		11	11
Average	0.0	0.0	-0.2	0.0	-0.2	1.7	3.9	2.2		14.3	-0.1
p	> 0.20	> 0.20	0.001	0.2	0.01	0.002	0.002	0.1		0.05	> 0.20

[16] In a car dashboard this may be beneficial for example, as the lower volatility would result in less emissions as measured by certain automotive test standards such as VDA 278.

Environmental Emissions

The ECHA investigation report on PVC and its additives stated that: "*end-of-life (recycling and landfills) can be considered the main contributor to the overall releases of prioritised PVC additives.*". Figure 5 of the report indicates that 28.5% of all emissions are caused by recycling (while the accompanying text indicates the recycling accounts for ~31% of releases).

To understand how this relatively large fraction is attributed to recycling, table 52 of the appendix A+B of the report provides clarity. For conventional life cycle stages, refined release factors from the OECD Emission Scenario Document for Plastics Additives (OECD 2014) are available and used. However, for the professional and waste stages generic, thus conservative, factors from ECHA guidance R16 (ECHA 2016b) and R18 (ECHA 2012b) are used, respectively (Table 24). For recycling, an emission factor of 10% to air is applied, although this assumption has been questioned by the thermoplastics recyclers based on their practical experience.

*Table 24 Emission factors used in the ECHA Investigation Report, their methodological basis, and a heatmap of total emission factor used. Based on: ECHA Investigation Report Appendix A+B Table 52. * R16 was used to establish a release factor for soil, which is not done in the OECD ESD.*

Exposure Scenario	Estimation Method	Water	Air	Soil	Total
ES1: Formulation (PVC compounding)	OECD	0.08%	0.03%	0%	0.1%
ES2: Article production (PVC conversion)	OECD	0.25%	0.25%	0%	0.5%
ES3: Article service life. Use in pipes and pipe fittings	OECD | R16*	3.20%	0.05%	1.60%	4.9%
ES4: Article service life. Use in window frames	OECD | R16*	1.60%	0.05%	1.60%	3.3%
ES5: Article service life. Use in cables	OECD	0.05%	0.05%	0%	0.1%
ES6: Article service life. Use in flooring	OECD | R16*	1.60%	0.05%	1.60%	3.3%
ES7: Article service life. Use in packaging)	OECD | R16*	1.60%	0.05%	1.60%	3.3%
ES8: Article service life. Use in toys	OECD | R16*	1.60%	0.05%	1.60%	3.3%
ES9: Article service life. Use in artificial leather	OECD | R16*	1.60%	0.05%	1.60%	3.3%
ES10: Article service life. Automotive interiors	OECD | R16*	1.60%	0.05%	1.60%	3.3%
ES11: Article service life. Medical application	OECD	0.05%	0.05%	0.00%	0.1%
ES12: Professional use. Handling plastic articles	R16	2.50%	2.50%	2.50%	7.5%
ES13: Waste stage. Recycling	R18	0.00%	10%	0.00%	10.0%
ES14: Waste stage. Landfill	R18	1.60%	0.05%	3.20%	4.9%
ES15: Waste stage. Incineration	R18	0.01%	0.01%	0.00%	0.0%

The ECHA Guidance R18 contains a table R18-6 which forms the basis for this 10% to air estimate (reproduced for clarity in Figure 21), which clarifies that this factor is based on "expert judgment". The footnote 68 on this expert judgement on this states that: "*In this guidance expert judgement has been used as source to derive default values when necessary due to lack of basic data in literature. Experience and sector knowledge have been used to derive default values when available information does not allow to correlate emissions of single substances to the amount of that substance entered into a waste treatment process.*". In other words, an emission factor was necessary, no data was available, and the emission factor was established using expert judgement and the best available technical assumptions. However, some logic went into the estimates is presented (e.g. what can be expected to be a dusty process or not) and there is great transparency on the strength of evidence behind the factors.

Table R.18- 6: Defaults for shredding

Parameter	Default	Reasoning
# of installations	210[67]	Amount of installations in EU-27
Emission days	330	Normal operating days
WWTP	not relevant	It is assumed that no onsite wastewater treatment plant exists.
Release factor to air (F_{air})	0.1	Paper and plastics, minerals: material has low weight and/or dust is likely to occur – expert judgement[68]
	0.05	Rubber: material has medium weight, some release likely - expert judgement
	0.01	Metals,: emitted dust is heavy and the majority of the release settles shortly after emission – expert judgement
Release factor to water (F_{water})	0	No water contact
Release factor to soil (F_{soil})	0	Processing does not give rise to releases to soil

Figure 21 Table R.18-6 as included in ECHA Guidance R.18

The factor for shredding is more extensively investigated, discussed, and refined in the rigid PVC report and used to quantify the emissions from shredding which occurs outdoor in large rigid PVC recycling facilities. However, as can be seen in the plant reports the shredding of materials in flexible PVC recycling facilities occurs indoor and is performed under specifically engineered air extraction/dedusting systems that are connected to air cleaning systems that have been shown to be extremely effective in the rigid PVC recycling facilities and in Plant E included in this report. While plant F did not provide an emission measurement result for this report, their air cleaning systems were of an equivalent quality. As such the emissions to air that are to be expected from these facilities are likely well below 0.002% of material processed.

All in all, the emissions of dust, and as a consequence microplastics from flexible PVC recycling facilities, are far less than the 10% that is assumed in the PVC Investigation Report.

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ANNEX I EXPOSURE MEASUREMENT RESULTS

Table 25 Inhalable and respirable dust measurements. RCR colour coding applied: <LOD = no shading, <0.1 = blue, 0.1 – 0.5 = green, 0.5 – 1.0 is yellow, and >1 = red.

ID	Sample Description	Time (min)	Inhalable Volume (l)	Inhalable Dust (mg)	Inhalable (mg/m^3)	Inhalable RCR	Respirable Volume (l)	Respirable Dust (mg)	Respirable (mg/m^3)	Respirable RCR
E1	PAL - Mechanical Treatment Operator	292	593	0.27	0.46	0.09	648	<0.05	<0.08	<0.08
E2	TER - Extrusion Operator (TR2)	280	562	0.37	0.66	0.13	620	0.14	0.23	0.23
E3	TET - Extrusion Operator (TR3)	320	646	2.06	3.19	0.64	709	0.54	0.76	0.76
E4	PER - Extrusion Supervisor and Micronization Operator	347	696	1.1	1.58	0.32	771	0.32	0.42	0.42
F1	CK - RM Handling / Sorting	210	443	0.46	1.04	0.21	463	0.13	0.28	0.28
F2	BF - Micronization	210	441	0.26	0.59	0.12	467	<0.05	<0.11	<0.11
F3	DS - Supervisor	210	444	0.09	0.20	0.04	462	<0.05	<0.11	<0.11

Table 26 Results of the tin in inhalable dust measurements and calculation of maximum organotin concentrations and Risk Characterisation Ratios (RCR) based on a limit of 25, 180, and 5750 $\mu g/m^3$ for DOTE, DMTE, and MMTE. RCR colour coding applied: <LOD = no shading, <0.1 = blue, 0.1 – 0.5 = green, 0.5 – 1.0 is yellow, and >1 = red.

ID	Sample Description	Sn ($\mu g/m^3$)	Max DOTE ($\mu g/m^3$)	Max DMTE ($\mu g/m^3$)	Max MMTE ($\mu g/m^3$)	Max DOTE RCR	Max DMTE RCR	Max MMTE RCR
E1	PAL - Mechanical Treatment Operator	<0.17	<1.07	<0.79	<1.06	<0.04	<0.004	<0.0002
E2	TER - Extrusion Operator (TR2)	<0.18	<1.13	<0.83	<1.11	<0.05	<0.005	<0.0002
E3	TET - Extrusion Operator (TR3)	<0.15	<0.98	<0.72	<0.97	<0.04	<0.004	<0.0002
E4	PER - Extrusion Supervisor and Micronization Operator	<0.14	<0.91	<0.67	<0.9	<0.04	<0.004	<0.0002
F1	CK - RM Handling / Sorting	<0.23	<1.43	<1.06	<1.41	<0.06	<0.006	<0.0002
F2	BF - Micronization	<0.23	<1.44	<1.06	<1.42	<0.06	<0.006	<0.0002
F3	DS - Supervisor	<0.23	<1.43	<1.05	<1.41	<0.06	<0.006	<0.0002

*Table 27 Airborne concentrations of lead (Pb), Cadmium (Cd), Antimony (Sb), Zinc (Zn), Titanium (Ti), and Calcium (Ca) as well as risk characterisation ratios for Pb, Cd, Sb, Zn, Ti, and Ca. RCR colour coding applied: <LOD = no shading, <0.1 = blue, 0.1 – 0.5 = green, 0.5 – 1.0 is yellow, and >1 = red. * Operator wore appropriate tight fitting half mask respirator which will have awarded a protection factor of at least 10 indicating that the operator was working safely.*

ID	Sample Description	Pb (µg/m³)	RCR	Cd (µg/m³)	RCR	Sb (µg/m³)	RCR	Zn (µg/m³)	RCR	Ti (µg/m³)	Ca (µg/m³)
E1	PAL - Mechanical Treatment Operator	<0.337	<0.011	<0.169	<0.17	0.334	0.00134	<1.69	<0.003	0.74	57.20
E2	TER - Extrusion Operator (TR2)	<0.356	<0.012	<0.178	<0.18	0.327	0.00131	<1.78	<0.004	0.77	172.83
E3	TET - Extrusion Operator (TR3)	<0.31	<0.01	<0.155	<0.16	0.443	0.00177	14.78	0.030	1.28	351.86
E4	PER - Extrusion Supervisor and Micronization Operator	<0.287	<0.01	<0.144	<0.14	<0.14	<0.00056	3.53	0.007	0.49	403.30
F1	CK - RM Handling / Sorting	<0.451	<0.015	<0.226	<0.23	<0.23	<0.00092	<2.26	<0.005	<0.68	269.30
F2	BF - Micronization	<0.454	<0.015	<0.227	<0.23	<0.23	<0.00092	2.68	0.005	0.68	110.14
F3	DS - Supervisor	2.995	0.100	<0.225	<0.23	<0.23	<0.00092	<2.25	<0.005	<0.68	68.09

Table 28 Urinary concentrations of 41 plasticiser metabolites belonging to 21 plasticisers and creatinine concentration in pre- and post- shift samples of workers in flexible PVC recycling. All values are in µg/L unless otherwise specified. Shaded metabolites were used to calculate EDI values.

Parent	DMP	DEP	DnPrP	DMoxyEP	BBzP	DiBP		DnBP			DCHP	DnPeP			DiPeP		DnHexP			DiHepP	DnHepP			DEHP				DiNP			DiDP			DnOP		DEHTP			DINCH			Creatinine (mg/L)
Metabolite	MMP	MEP	MnPrP	MMoxyEP	MBzP	MiBP	2OH-MiBP	MnBP	3OH-MnBP	MCPP	MCHP	MnPeP	4OH-MnPeP	4cx-MnBP	MiPeP	4OH-MiPeP	MnHexP	5OH-MnHexP	5cx-MnPeP	MiHepP	MnHepP	6OH-MnHepP	6cx-MnHexP	MEHP	5OH-MEHP	5oxo-MEHP	5cx-MEPP	OH-MiNP	oxo-MiNP	cx-MiNP	OH-MiDP	oxo-MiDP	cx-MiDP	MnOP	7cx-MnHepP	5cx-MEPTP	5OH-MEHTP	5oxo-MEHTP	OH-MINCH	oxo-MINCH	cx-MINCH	
LOQ	1	0.5	0.2	0.5	0.2	1	0.2	1	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.3	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.05	0.05	0.05	
A01	1.3	41.5	<0.2	<0.5	1.3	11.4	3.3	9.5	1.2	1.3	<0.2	<0.2	<0.2	2.5	<0.2	<0.2	<0.2	<0.2	0.4	<0.2	<0.2	<0.2	<0.2	2.1	9.4	7.7	10.0	12.9	9.3	30.0	12.7	7.3	11.4	<0.2	<0.2	66.1	7.7	6.2	1.7	0.8	1.6	726
A03	3.6	75.2	<0.2	<0.5	9.1	45.5	19.1	16.4	2.9	1.9	<0.2	<0.2	<0.2	3.8	<0.2	<0.2	<0.2	<0.2	0.4	<0.2	<0.2	<0.2	<0.2	12.4	18.5	13.0	16.8	25.6	18.8	31.8	8.5	4.8	3.4	<0.2	<0.2	34.2	6.7	5.1	1.7	1.9	1.2	1794
A04	4.9	471.0	<0.2	<0.5	10.4	39.4	20.5	50.6	11.5	2.5	<0.2	<0.2	<0.2	2.8	<0.2	<0.2	<0.2	<0.2	0.9	<0.2	<0.2	<0.2	<0.2	4.6	18.4	14.2	13.7	16.0	11.3	12.6	4.0	2.5	1.0	<0.2	<0.2	21.0	25.1	19.1	7.4	3.5	2.8	2540
A05	3.7	34.8	<0.2	<0.5	3.5	15.0	3.0	11.7	1.0	1.1	<0.2	<0.2	<0.2	2.5	<0.2	<0.2	<0.2	<0.2	0.3	<0.2	<0.2	<0.2	<0.2	1.5	7.3	5.5	9.8	13.3	9.6	30.0	2.8	1.6	2.9	<0.2	<0.2	124.0	9.8	8.2	2.9	1.5	3.2	1878
A06	2.0	28.9	<0.2	<0.5	3.5	8.4	2.8	10.7	1.3	3.6	<0.2	<0.2	<0.2	8.1	<0.2	<0.2	<0.2	<0.2	1.5	<0.2	<0.2	<0.2	<0.2	5.1	28.1	22.9	25.8	70.5	57.8	63.0	59.0	42.1	14.0	<0.2	<0.2	206.0	24.3	18.6	2.1	0.9	1.4	693
A08	2.6	29.1	<0.2	<0.5	7.0	29.1	10.4	9.7	1.8	2.8	<0.2	<0.2	<0.2	5.5	<0.2	<0.2	<0.2	<0.2	0.5	<0.2	<0.2	<0.2	<0.2	8.0	19.8	14.1	17.5	48.8	33.9	41.0	18.7	10.8	3.9	<0.2	<0.2	71.4	18.1	13.4	1.7	1.3	1.2	1357
A09	3.9	264.0	<0.2	<0.5	9.0	36.4	21.7	35.9	9.6	2.8	<0.2	<0.2	<0.2	4.4	<0.2	<0.2	<0.2	<0.2	1.3	<0.2	<0.2	<0.2	<0.2	5.0	24.7	19.3	16.3	29.8	23.1	17.7	7.5	4.4	1.4	<0.2	<0.2	43.2	32.1	23.8	4.0	2.3	2.1	2631
A10	3.3	32.0	<0.2	<0.5	5.6	9.3	2.4	6.9	0.6	1.6	<0.2	<0.2	<0.2	4.8	<0.2	<0.2	<0.2	<0.2	0.6	<0.2	<0.2	<0.2	<0.2	2.1	20.8	14.9	22.8	33.0	23.6	29.3	5.9	3.8	1.5	<0.2	<0.2	10.0	11.9	9.7	3.7	1.6	2.8	2051
A11	2.3	45.7	<0.2	<0.5	3.1	19.4	6.0	19.9	2.8	5.1	<0.2	<0.2	<0.2	12.6	<0.2	<0.2	<0.2	<0.2	2.4	<0.2	<0.2	<0.2	<0.2	3.0	24.4	20.7	35.6	83.1	63.6	115.0	80.4	49.1	18.2	<0.2	<0.2	62.3	20.7	15.9	3.2	1.2	2.0	1305
A13	1.6	25.1	<0.2	<0.5	6.3	8.4	3.6	9.3	1.8	1.2	<0.2	<0.2	<0.2	1.6	<0.2	<0.2	<0.2	<0.2	0.3	<0.2	<0.2	<0.2	<0.2	5.2	10.6	7.4	12.0	12.5	9.0	18.4	3.9	2.0	1.1	<0.2	<0.2	24.3	5.9	4.9	4.5	1.7	2.4	1476
A14	2.4	96.8	<0.2	<0.5	8.2	42.2	12.9	29.4	4.0	3.9	<0.2	<0.2	<0.2	7.6	<0.2	<0.2	0.3	<0.2	2.8	<0.2	<0.2	<0.2	<0.2	1.9	11.4	9.7	12.9	40.6	31.6	74.3	11.4	7.7	3.2	<0.2	<0.2	86.3	32.2	27.0	24.1	8.7	15.4	1873
A15	6.7	639.0	<0.2	<0.5	3.2	30.5	8.7	23.3	2.9	1.7	<0.2	<0.2	<0.2	3.6	0.3	<0.2	<0.2	<0.2	0.5	<0.2	<0.2	<0.2	<0.2	0.9	7.8	6.4	13.7	18.8	13.0	31.4	8.5	4.4	3.5	<0.2	<0.2	5.5	5.5	4.3	3.1	1.3	2.6	2438
A16	2.8	36.0	<0.2	<0.5	2.6	10.7	3.0	12.7	1.6	3.9	<0.2	<0.2	<0.2	9.0	<0.2	<0.2	<0.2	<0.2	1.5	<0.2	<0.2	<0.2	<0.2	4.8	24.1	19.2	29.4	59.2	44.1	108.0	50.7	34.4	22.3	<0.2	<0.2	341.0	36.6	21.1	1.8	0.8	2.1	918
A18	1.6	18.6	<0.2	<0.5	5.3	6.6	2.5	5.9	1.0	1.7	<0.2	<0.2	<0.2	1.9	<0.2	<0.2	<0.2	<0.2	0.3	<0.2	<0.2	<0.2	<0.2	14.4	29.4	23.9	30.6	15.4	10.8	22.1	5.6	4.0	1.5	<0.2	<0.2	82.4	7.4	5.9	3.4	1.3	2.2	1288
A19	1.9	301.0	<0.2	<0.5	3.5	14.2	4.9	9.1	1.3	2.0	<0.2	<0.2	<0.2	4.0	<0.2	<0.2	<0.2	<0.2	1.4	<0.2	<0.2	<0.2	<0.2	5.2	18.1	15.3	14.4	28.7	22.0	50.5	9.0	6.7	3.0	<0.2	<0.2	84.4	8.4	6.9	7.5	2.6	6.5	1089
A20	1.5	38.5	<0.2	<0.5	1.2	4.6	1.5	4.4	0.7	1.0	<0.2	<0.2	<0.2	2.3	<0.2	<0.2	<0.2	<0.2	0.3	<0.2	<0.2	<0.2	<0.2	1.9	7.0	5.8	8.3	23.2	15.5	19.5	7.1	4.5	1.6	<0.2	<0.2	14.6	5.4	5.1	0.7	0.4	0.4	762
C01	5.4	330.0	<0.2	<0.5	2.9	34.8	8.1	43.2	5.1	0.9	<0.2	<0.2	<0.2	0.7	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	0.7	3.6	2.5	4.5	4.8	4.0	5.0	1.5	0.8	0.3	<0.2	<0.2	3.5	1.9	1.2	6.8	2.8	2.9	1972
C02	<1	14.7	<0.2	<0.5	1.9	17.3	6.7	10.5	1.6	0.4	<0.2	<0.2	<0.2	0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	0.8	1.7	1.3	1.4	1.1	0.8	1.6	0.3	<0.2	<0.2	<0.2	<0.2	3.5	2.6	1.6	0.4	0.2	0.4	1763
C03	5.6	16.7	<0.2	<0.5	2.5	23.7	6.8	63.8	8.8	1.7	<0.2	<0.2	<0.2	0.4	<0.2	<0.2	0.8	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	0.8	3.0	1.9	2.9	2.7	1.7	3.0	1.0	0.5	0.3	<0.2	<0.2	10.9	1.7	0.9	4.4	1.6	2.7	1495
C04	1.8	349.0	<0.2	<0.5	11.2	14.9	4.2	17.6	2.4	2.6	<0.2	<0.2	<0.2	2.0	<0.2	<0.2	na	<0.2	0.4	<0.2	<0.2	<0.2	<0.2	2.7	12.3	6.5	12.1	14.3	12.5	14.4	3.6	1.2	0.9	<0.2	<0.2	77.3	6.1	4.2	8.7	4.0	3.4	1162
C05	<1	4.3	<0.2	<0.5	1.6	6.2	1.8	4.1	0.4	0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	0.8	1.4	1.0	0.8	0.9	0.6	1.2	<0.2	<0.2	<0.2	<0.2	<0.2	13.7	1.3	<0.2	0.6	0.3	0.4	873
C06	<1	1.3	<0.2	<0.5	0.7	2.0	0.6	4.0	0.6	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.3	0.6	0.4	0.7	0.6	0.4	0.7	<0.2	<0.2	<0.2	<0.2	<0.2	8.0	0.7	<0.2	0.8	0.3	1.1	378

Table 29 Estimated Daily Intake Values (in µg/kg bw/d) for 15 ortho-phthalates and 2 alternative plasticisers. Values precede by a less than sign were solely based on the limit of detection of the analytical method. Values precede by a bracketed less than sign were based on a mixture of measured values and limits of quantification for the analysed metabolites. Values not preceded by a less than or bracketed less than sign are based on measured metabolite concentrations.

	DMP	DEP	DnPrP	DiBP	DMoxyEP	BBzP	DnBP	DiPeP	DnPeP	DnHexP	DiHepP	DEHP	DnHepP	DiNP	DiDP	DEHTP	DINCH
A01	0.06	2.18	<0.01	0.64	<0.03	0.07	0.55	<0.02	<0.02	<0.02	<0.14	2.64	<0.14	7.36	3.63	20.56	1.09
A03	0.07	1.60	<0.00	1.03	<0.01	0.19	0.38	<0.01	<0.01	<0.01	<0.06	2.23	<0.06	4.38	0.98	4.31	0.40
A04	0.07	7.07	<0.00	0.63	<0.01	0.15	0.83	<0.01	<0.01	<0.01	<0.04	1.31	<0.04	1.63	0.34	1.87	0.96
A05	0.07	0.71	<0.00	0.33	<0.01	0.07	0.26	<0.01	<0.01	<0.01	<0.06	0.84	<0.06	2.88	0.31	14.91	0.77
A06	0.10	1.59	<0.01	0.50	<0.03	0.19	0.64	<0.02	<0.02	<0.02	<0.15	7.73	<0.15	28.57	19.26	67.13	1.22
A08	0.07	0.82	<0.01	0.87	<0.02	0.19	0.30	<0.01	<0.01	<0.01	<0.08	2.88	<0.08	9.43	2.87	11.88	0.51
A09	0.05	3.83	<0.00	0.56	<0.01	0.13	0.57	<0.01	<0.01	<0.01	<0.04	1.62	<0.04	2.79	0.60	3.71	0.56
A10	0.06	0.60	<0.00	0.19	<0.01	0.10	0.14	<0.01	<0.01	<0.01	<0.05	1.93	<0.05	4.33	0.62	1.10	0.76
A11	0.06	1.34	<0.01	0.61	<0.02	0.09	0.64	<0.01	<0.01	<0.01	<0.08	4.20	<0.08	20.64	13.10	10.78	0.95
A13	0.04	0.65	<0.01	0.23	<0.01	0.16	0.26	<0.01	<0.01	<0.01	<0.07	1.57	<0.07	2.78	0.53	3.72	1.13
A14	0.05	1.97	<0.00	0.92	<0.01	0.16	0.66	<0.01	<0.01	0.01	<0.06	1.25	<0.06	8.03	1.34	10.41	5.06
A15	0.10	10.00	<0.00	0.51	<0.01	0.05	0.40	0.01	<0.01	<0.01	<0.04	0.77	<0.04	2.66	0.70	0.51	0.56
A16	0.11	1.50	<0.01	0.47	<0.02	0.11	0.58	<0.02	<0.02	<0.01	<0.11	5.53	<0.11	23.61	12.24	83.89	1.00
A18	0.05	0.55	<0.01	0.21	<0.02	0.15	0.19	<0.01	<0.01	<0.01	<0.08	5.02	<0.08	3.86	0.97	14.45	1.04
A19	0.06	10.54	<0.01	0.53	<0.02	0.12	0.35	<0.01	<0.01	<0.01	<0.10	3.19	<0.10	9.54	1.90	17.50	3.07
A20	0.07	1.93	<0.01	0.25	<0.03	0.06	0.24	<0.02	<0.02	<0.02	<0.14	1.98	<0.14	7.90	2.02	4.33	0.34
C01	0.10	6.38	<0.00	0.72	<0.01	0.05	0.91	<0.01	<0.01	<0.01	<0.05	0.37	<0.05	0.72	0.15	0.41	1.19
C02	<0.02	0.32	<0.00	0.40	<0.01	0.04	0.25	<0.01	<0.01	<0.01	<0.06	0.19	<0.06	0.21	(<)0.04	0.45	0.11
C03	0.14	0.43	<0.01	0.65	<0.01	0.06	1.78	<0.01	<0.01	0.04	<0.07	0.37	<0.07	0.51	0.14	1.65	1.13
C04	0.06	11.46	<0.01	0.52	<0.02	0.36	0.63	<0.01	<0.01	na	<0.09	1.90	<0.09	3.67	0.55	15.02	2.51
C05	<0.04	0.19	<0.01	0.29	<0.02	0.07	0.19	<0.02	<0.02	<0.02	<0.12	0.29	<0.12	0.32	<0.06	3.54	0.27
C06	<0.1	0.13	<0.02	0.21	<0.05	0.07	0.44	<0.04	<0.04	<0.04	<0.28	(<)0.36	<0.28	0.47	<0.14	4.78	1.23

Table 30 Risk characterisation ratios for individual ortho-phthalates and 2 alternative plasticisers. Colour coding applied: RCR<0.01 = blue, RCR0.01-0.1 = green, RCR 0.1-0.5 = yellow, RCR0.5-1 = orange, RCR>1 = red, and RCRs solely based on limit of detection = no shading. Values precede by a less than sign were solely based on the limit of detection of the analytical method. Values precede by a bracketed less than sign were based on a mixture of measured values and limits of quantification for the analysed metabolites. Values not preceded by a less than or bracketed less than sign are based on measured metabolite concentrations.

	DMP	DEP	DiBP	BBzP	DnBP	DEHP	DiNP	DiDP	DEHTP	DINCH
A01	0.000003	0.0015	0.0026	0.0001	0.0027	0.0227	0.0245	0.0110	0.0130	0.0011
A03	0.000004	0.0011	0.0041	0.0002	0.0019	0.0192	0.0146	0.0030	0.0027	0.0004
A04	0.000004	0.0047	0.0025	0.0002	0.0042	0.0113	0.0054	0.0010	0.0012	0.0010
A05	0.000004	0.0005	0.0013	0.0001	0.0013	0.0072	0.0096	0.0009	0.0094	0.0008
A06	0.000005	0.0011	0.0020	0.0002	0.0032	0.0667	0.0952	0.0584	0.0425	0.0012
A08	0.000004	0.0005	0.0035	0.0002	0.0015	0.0248	0.0314	0.0087	0.0075	0.0005
A09	0.000003	0.0026	0.0023	0.0001	0.0028	0.0140	0.0093	0.0018	0.0023	0.0006
A10	0.000003	0.0004	0.0007	0.0001	0.0007	0.0167	0.0144	0.0019	0.0007	0.0008
A11	0.000003	0.0009	0.0024	0.0001	0.0032	0.0362	0.0688	0.0397	0.0068	0.0010
A13	0.000002	0.0004	0.0009	0.0002	0.0013	0.0135	0.0093	0.0016	0.0024	0.0011
A14	0.000002	0.0013	0.0037	0.0002	0.0033	0.0108	0.0268	0.0041	0.0066	0.0051
A15	0.000005	0.0067	0.0020	0.0000	0.0020	0.0067	0.0089	0.0021	0.0003	0.0006
A16	0.000006	0.0010	0.0019	0.0001	0.0029	0.0477	0.0787	0.0371	0.0531	0.0010
A18	0.000002	0.0004	0.0008	0.0002	0.0009	0.0433	0.0129	0.0030	0.0091	0.0010
A19	0.000003	0.0070	0.0021	0.0001	0.0017	0.0275	0.0318	0.0058	0.0111	0.0031
A20	0.000004	0.0013	0.0010	0.0001	0.0012	0.0171	0.0263	0.0061	0.0027	0.0003
C01	0.000005	0.0043	0.0029	0.0001	0.0046	0.0032	0.0024	0.0005	0.0003	0.0012
C02	<0.000001	0.0002	0.0016	0.0000	0.0012	0.0016	0.0007	(<)0.0001	0.0003	0.0001
C03	0.000007	0.0003	0.0026	0.0001	0.0089	0.0032	0.0017	0.0004	0.0010	0.0011
C04	0.000003	0.0076	0.0021	0.0004	0.0032	0.0163	0.0122	0.0017	0.0095	0.0025
C05	<0.000002	0.0001	0.0012	0.0001	0.0010	0.0025	0.0011	<0.0002	0.0022	0.0003
C06	<0.000005	0.0001	0.0008	0.0001	0.0022	(<)0.0031	0.0016	<0.0004	0.0030	0.0012

Table 31 Risk Characterisation Ratios for Individual anti-androgenic effect and the Sum RCR for anti-androgenic effect (Sum-RCR_{AA}). Colour coding applied: RCR<0.01 = blue, RCR0.01-0.1 = green, RCR 0.1-0.5 = yellow, RCR0.5-1 = orange, and RCR>1 = red. Measured values below limit of detection were used at limit of detection.

	DiBP	BBzP	DnBP	DEHP	DiNP	Sum-RCR_{AA}
A01	0.003	0.003	0.041	0.132	0.062	0.241
A03	0.005	0.010	0.028	0.111	0.037	0.192
A04	0.003	0.008	0.062	0.066	0.014	0.152
A05	0.002	0.003	0.019	0.042	0.024	0.091
A06	0.002	0.009	0.048	0.387	0.242	0.689
A08	0.004	0.010	0.022	0.144	0.080	0.260
A09	0.003	0.006	0.043	0.081	0.024	0.157
A10	0.001	0.005	0.010	0.097	0.037	0.150
A11	0.003	0.004	0.048	0.210	0.175	0.440
A13	0.001	0.008	0.020	0.078	0.024	0.131
A14	0.005	0.008	0.049	0.063	0.068	0.192
A15	0.003	0.002	0.030	0.039	0.023	0.096
A16	0.002	0.005	0.043	0.277	0.200	0.528
A18	0.001	0.008	0.014	0.251	0.033	0.306
A19	0.003	0.006	0.026	0.160	0.081	0.275
A20	0.001	0.003	0.018	0.099	0.067	0.188
C01	0.004	0.003	0.068	0.019	0.006	0.099
C02	0.002	0.002	0.019	0.010	0.002	0.034
C03	0.003	0.003	0.133	0.019	0.004	0.162
C04	0.003	0.018	0.047	0.095	0.031	0.194
C05	0.001	0.003	0.014	0.015	0.003	0.037
C06	0.001	0.003	0.033	0.018	0.004	0.060

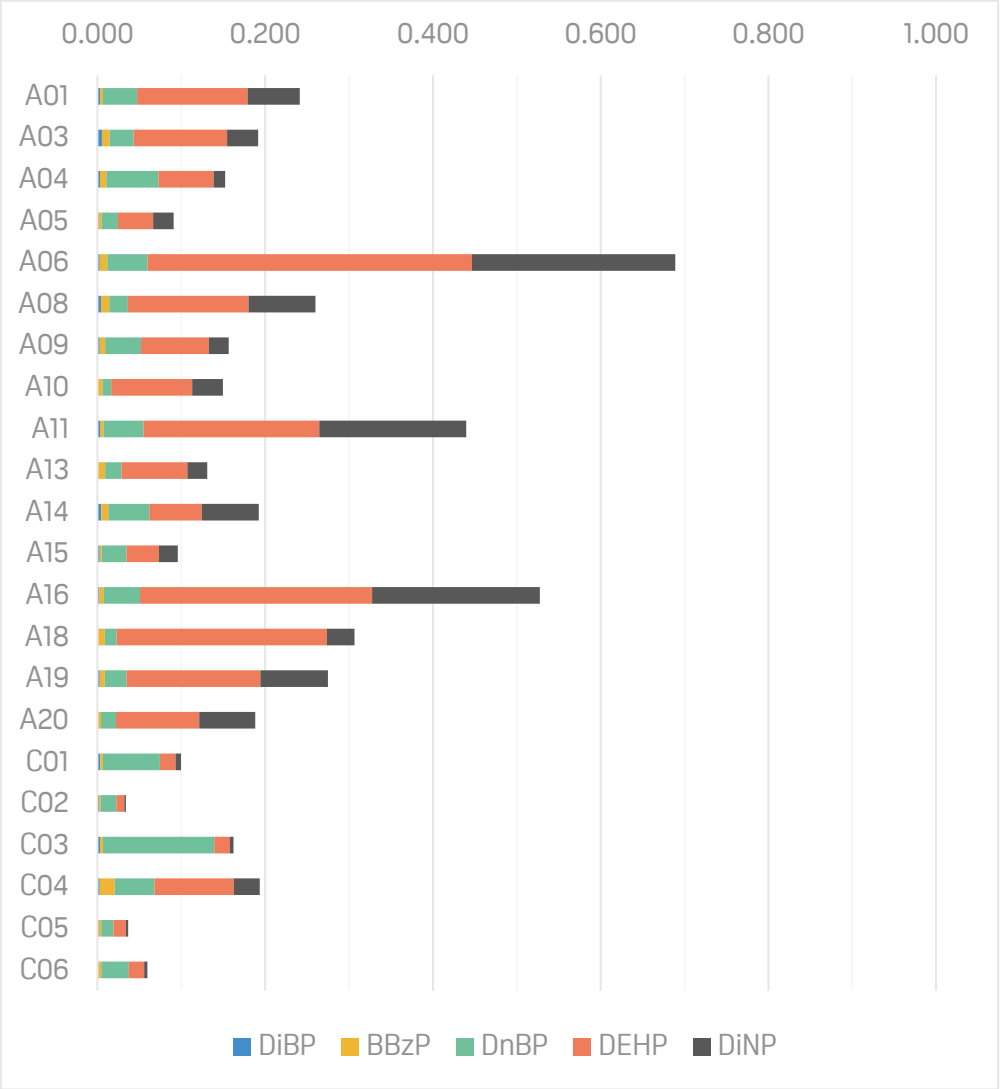

Figure 22 Visualisation of the contribution of different ortho-phthalate plasticisers to the anti-androgenic RCR. Measured values below limit of detection were used at limit of detection.