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Possible health risks due to high concentrations of 3-MCPD and glycidyl fatty acid esters in certain foods

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3-Monochloropropanediol (3-MCPD), 2-monochloropropanediol (2-MCPD) and their fatty acid esters, and glycidyl fatty acid esters, are heat-induced contaminants in foods. The substances have been detected in various heated foods, such as certain (rich) baked goods, infant formula and cooking fats and oils. Free 3-monochloropropanediol (3-MCPD) and free 2-monochloropropanediol (2-MCPD) can be formed when foods containing both fat and salt are exposed to high temperatures during the manufacturing process. Based on current knowledge, the ester-bonded forms, i.e. 2-MCPD-, 3-MCPD- and glycidyl fatty acid esters are mainly formed during the refinement of plant-based fats and oils, i.e. during heat treatments for the purpose of cleaning and finishing.

The German Federal Institute for Risk Assessment (BfR) has estimated the total exposure (total intake via these foods) to these compounds for the relevant population groups and assessed the risks to health they present, based on current occurrence data and available consumption data for these foods.

For **3-MCPD** and its fatty acid esters, based on the exposure assessment carried out, the tolerable daily intake (TDI) derived by the European Food Safety Authority (EFSA) is not exceeded for normal and high adult consumers. An increased health risk is therefore not expected for this population group. By contrast, a clear exceedance of the TDI has been found for children and infants in some cases. An increased health risk from long-term consumption is therefore possible for this population groups.

For **2-MCPD** and its fatty acid esters, an assessment of possible health risks is not possible at the current time, due to a lack of toxicological data.

Glycidol and its ester-bound form are genotoxic and carcinogenic. Therefore, the internationally established MOE (margin of exposure) approach is used for risk characterisation. The MOE is calculated as a quotient from a suitable toxicological reference point and exposure to the substance in humans, and is used for prioritising the urgency of risk management measures.[1] The T25 (the chronic dose at which cancer occurs within a certain tissue in 25% of laboratory animals during their lifespan) of 10.2 mg/kg body weight and day derived from the data of a long-term study with laboratory animals is used as a reference point in this particular case. Here, a MOE of 25,000 or more is of little concern with regard to public health, and therefore represents a low priority for risk management measures.

[1] Neither the reference point derived from animal studies nor the MOE values are health-based guidance values. Rather, the MOE is for the purpose of prioritising risk management measures. The conclusion that a total intake quantity with a MOE of 25,000 or more is ‘less harmful’ with regards to possible cancer risks should not be equated with ‘harmless’ from a toxicology perspective, as health risks cannot even be ruled out with enough certainty for intake quantities in this area. Therefore, the recommendation of keeping exposure to substances with a genotoxic and carcinogenic effect as low as is reasonably achievable generally applies (ALARA principle).

For adults, most of the consumption scenarios of the conducted exposure assessment result in intake levels of bound glycidol, which lead to MOE values above 25,000.

However, a scenario for frequent consumers of frying fats with high contents of bound glycidol leads to an intake level, which results in a MOE value of 15,131. For children and infants, various consumption scenarios (normal and high consumers) also reveal MOE values of clearly less than 25,000 in some cases. For example, for infants fed exclusively with infant formula, who only consume infant formula with high concentrations of bound glycidol, is lower by a factor of around 10, at about 2,900. An increased health risk due to chronic intake therefore appears to be possible for certain population groups.

The BfR recommends further reducing the concentrations of 2-MCPD and 3-MCPD and their fatty acid esters, and the concentrations of ester-bound glycidol in food groups which are heavily consumed by children (doughnuts/Berliners, margarines/vegetable fats) and infants in particular (infant formula). This is especially applicable for infant formula, as there is no alternative food source for non-breastfed infants.

1 Subject of the assessment

The German Federal Institute for Risk Assessment (BfR) has assessed the health risks resulting from the occurrence of 2-monochloropropanediol (2-MCPD) and 3-monochloropropanediol (3-MCPD) and their fatty acid esters, and glycidyl fatty acid esters, in foods. For the health risk assessment, the concentrations of all key food groups for which there is current data on the occurrence of these substances were taken into account. This particularly focused on data on concentrations of these substances in different food groups, which were gathered as part of the decision support project (DS Project) ‘Investigation of the occurrence of 3-MCPD esters and related compounds in foods’ by the German Federal Ministry of Food and Agriculture (BMEL). Furthermore, data from the project ‘Minimisation of 2- and 3-MCPD, glycidol and their fatty acid esters in smoked and heat-treated fish products’ by the Max Ruber Institute (MRI) (FEI 2017) and data from the Federal Office of Consumer Protection and Food Safety (BVL) were also taken into consideration.

2 Results

The BfR achieved the following results, based on the available data:

- Assessment of possible health risks from exposure to 3-MCPD and its fatty acid esters:

  Based on the exposure assessment carried out, for adult normal and high consumers, no exceedance of the tolerable daily intake (TDI) of 2 µg/kg body weight (BW) as derived by the European Food Safety Authority (EFSA) for 3-MCPD and its fatty acid esters An increased health risk is therefore not expected. By contrast, a a clear exceedance of the TDI has been observed for children and infants. An increased health risk from long-term consumption is therefore possible for these population groups.

- Assessment of possible health risks from exposure to 2-MCPD and its fatty acid esters:

  The little toxicological data available with regards to 2-MCPD and its fatty acid esters does not allow for any conclusive assessment of possible health risks.

- Assessment of possible health risks from exposure to glycidyl fatty acid esters:

  For glycidol, with its genotoxic and carcinogenic mode of action, it is not possible to derive an intake level which can be considered safe, since no lower threshold levels can be determined for genotoxic substances. The margin of exposure (MOE) approach has therefore been used for the prioritisation of risk management measures. In principle, a MOE of 25,000 or more derived from a T25 (the chronic dose at which cancer occurs within a certain tissue in 25% of laboratory animals during their lifespan) as a reference point, is generally considered being of low concern from a public health point of view, and therefore represents a low priority for risk management measures.

  In most consumption scenarios for adults, the exposure assessment carried out reveals intake levelsof bound glycidol (glycidol from fatty acid esters, see Chapter 3.1.3 ‘Exposure’, Section 3.1.3.1.2 ‘Concentration data’) which result in MOE values above 25,000. A scenario for high consumers of frying fats with high concentrations of bound glycidol certainly results in intake levelswhich yield a MOE of 15,131.

  For children and infants, various consumption scenarios (normal high consumers) reveal MOE values of clearly less than 25,000. For example, the MOE value for infants fed exclusively with infant formula, who only consume infant formula with high concentrations of bound glycidol, is lower by a factor of around 10, at about 2,900. Thus, for certain population groups an increased health risk due to chronic intake seems to be possible.

The BfR recommends further reducing the concentrations of 2-MCPD and 3-MCPD and their fatty acid esters, and the concentrations of ester-bound glycidol in food groups which are frequently consumed by children (doughnuts/Berliners, margarines/vegetable fats) and infants in particular (infant formula). This is especially important for infant formula, as there is no alternative food source for non-breastfed infants.

3 Rationale

3.1 Risk assessment

3.1.1 Hazard identification

3-Monochloro-1,2-propanediol (3-MCPD; $C_3H_7ClO_2$; CAS number: 96-24-2) and 2-monochloro-1,3-propanediol (2-MCPD; $C_3H_7ClO_2$; CAS number: 497-04-1) are contaminants in food which are formed during heating. They belong to the chloropropanol group. Both substances have a glycerine skeleton (see Figure 1), with a hydroxyl group in position 3 or position 2 being replaced by a chlorine atom. Both substances can either occur unbound or as

fatty acid esters which consist of the chloropropanol and one or two fatty acids (monoesters and diesters) (Andres *et al.* 2017).

Glycidol ($C_3H_6O_2$; CAS number: 556-52-5) is also derived from glycerine (see Figure 1), though possessing an epoxide structure and can in principle occur in a free form or esterified with a fatty acid as glycidyl fatty acid ester (bound glycidol) (Andres *et al.* 2017). Due to the high reactivity of glycidol and based on theoretical considerations, it is however assumed that glycidyl fatty acid esters predominate in foods, .

3-MCPD-, 2-MCPD- and glycidyl fatty acid esters are predominantly formed during the refinement of plant-based fats and oils during 'deodorisation', where unwanted odours and flavours are removed at high temperatures (Bakhiya *et al.* 2011; Andres *et al.* 2013; EFSA 2016; Andres *et al.* 2017). Refined, i.e. non-natural cooking oils and fats, can therefore sometimes contain considerable quantities of these substances (Kuhlmann 2011; Abd Razak *et al.* 2012). Accordingly, these substances can be found in various foods which contain plant-based oils and fats, such as margarine and infant formula (Bakhiya *et al.* 2011; Jędrkiewicz *et al.* 2016; Andres *et al.* 2017; Cheng *et al.* 2017; Spungen *et al.* 2018). Furthermore, these substances can be found in other heated foods, such as strongly browned toast, bread crusts or soy sauces (Andres *et al.* 2013; Andres *et al.* 2017; Kowalska 2017). 3- and 2-MCPD and their fatty acid esters can be formed when foods containing fat and table salt are exposed to high temperatures during the manufacturing process (Andres *et al.* 2017).

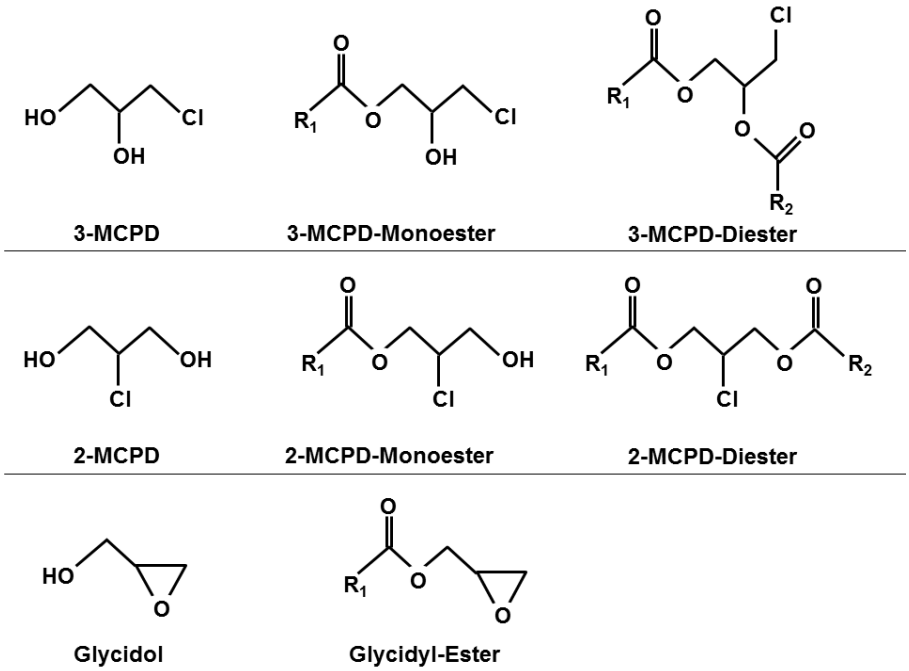

Figure 1. Schematic representation of the structural formulae of 3-MCPD, 2-MCPD and glycidol, and their fatty acid esters. R_1, R_2 = residual fatty acid.

3.1.2 Hazard characterisation

3.1.2.1 Toxicokinetics

3.1.2.1.1 3-MCPD and its fatty acid esters

Both free 3-MCPD and 3-MCPD from fatty acid esters are rapidly and efficiently reabsorbed in the gastrointestinal tract (GIT) after oral intake (Buhrke *et al.* 2011; Abraham *et al.* 2013; Onami *et al.* 2015; Gao *et al.* 2017). A study on bioavailability in rats has demonstrated that 3-MCPD fatty acid esters are almost completely cleaved and 3-MCPD is released during resorption in the intestine(BfR 2012). The free 3-MCPD is then absorbed. Such studies have not yet been done for 2-MCPD fatty acid esters. However, it can currently be assumed that this substance is also cleaved completely into 2-MCPD (Buhrke *et al.* 2015; EFSA 2016; Kaze *et al.* 2016). Therefore, characterization of the hazard potential of fatty acid esters can be made based on data regarding the hazard potential of the free substances. Elimination from serum of the 3-MCPD absorbed occurs rapidly. This arises from the results of studies on rats, in which the toxicokinetics of free 3-MCPD and its fatty acid ester 3-MCPD-dipalmitate were investigated (Abraham *et al.* 2013; Gao *et al.* 2017).

The metabolism of 3-MCPD has not yet been completely characterised. A known metabolic path which leads to the detoxification of the substance is the conjugation of 3-MCPD with glutathione in the liver. However, this metabolic conversion has only a limited capacity (Weber *et al.* 1995). The theoretically possible conversion of 3-MCPD into glycidol has not been detected *in vivo* as yet (Abraham *et al.* 2013; Gao *et al.* 2017). It has emerged from studies on rats that the oxidative conversion of 3-MCPD to β-chlorolactaldehyde, β-chlorolactic acid and oxalic acid represents an important metabolic route for this substance in mammals (Jones *et al.* 1978; Lynch *et al.* 1998). The precise process for the excretion of 3-MCPD and its metabolites is still not completely known. However, the available data suggests that excretion mainly occurs renally, and only to a lower extent via respiration and/or the intestine (Xiao *et al.* 2003; Abraham *et al.* 2013; Teng and Wang 2015; Gao *et al.* 2017).

3.1.2.1.2 2-MCPD and its fatty acid esters

Two *in vitro* studies have provided initial insights into the gastrointestinal biotransformation of 2-MCPD fatty acid esters and the resorption of free 2-MCPD (Buhrke *et al.* 2015; Kaze *et al.* 2016). Both studies have demonstrated that 2-MCPD fatty acid esters are hydrolysed in a similar way to 3-MCPD fatty acid esters by human Caco-2 cells, which were used as a model for the gastrointestinal barrier (Buhrke *et al.* 2015; Kaze *et al.* 2016). This led to a release of 2-MCPD. Furthermore, the results demonstrated that free 2-MCPD, but not the fatty acid esters, can pass through the gastrointestinal barrier (Buhrke *et al.* 2015; Kaze *et al.* 2016). Further studiesregarding the toxicokinetics of 2-MCPD or 2-MCPD fatty acid esters are not available.

3.1.2.1.3 Glycidol and its fatty acid esters

After oral intake of free or bound glycidol – after the hydrolysis of the esters, in the case of bound glycidol – a rapid and efficient resorption of free compounds in the GIT occurs. This has been revealed by a study on toxicokinetics with ^{14}C-marked glycidol on rats (Nomeir *et al.* 1995). Wakabayashi and colleagues also came to this conclusion (Wakabayashi *et al.*

2012). In this study, the oral administration of both glycidol and glycidyl linoleate led to a rapid rise in the plasma concentration of glycidol in rats and monkeys, respectively, which the authors concluded to be caused by both an extensive hydrolysis of bound glycidol and rapid resorption of free glycidol from the GIT of laboratory animals (Wakabayashi *et al.* 2012). Glycidol can be converted into different metabolites within the organism. Glutathione conjugates and mercapturic acid derivatives are among the most important metabolites (Appel *et al.* 2013). Further, the formation of 3-MCPD has been demonstrated *in vivo* to be part of the metabolisation of free and bound glycidol (Onami *et al.* 2015). Glycidol itself can react with a wide variety of nucleophilic biomolecules, such as proteins or deoxyribonucleic acid (DNA), due to the electrophilic epoxide ring structure (IARC 2000; Aasa *et al.* 2016; Abraham *et al.* 2017). The formation of DNA adducts is principally assumed as initial step in the process of chemical carcinogenesis (Melnick 2002; Aasa *et al.* 2016). Glycidol and its metabolites are mainly excreted via the kidneys and, to a lesser extent, via respiration and the gut (Nomeir *et al.* 1995; Bakhiya *et al.* 2011; Appel *et al.* 2013).

3.1.2.2 Assessment by national and international institutions

No extensive description of the hazard potential of 3-MCPD, 2-MCPD and glycidol, and their fatty acid esters, is presented in the present opinion. The description focuses on endpoints identified in previous assessments to be relevant for risk characterisation .

3.1.2.2.1 3-MCPD and its fatty acid esters

In 2001, 3-MCPD was assessed by the Scientific Committee on Food (SCF) of the European Commission and considered as a non-genotoxic carcinogen, on the basis of a long-term study in rats (SCF 2001). In the underlying study, rats (50 male and 50 female) were treated for 104 weeks with 3-MCPD (dose groups: 0; 1.1; 5.2 and 28 mg/kg BW and day (male) and 0; 1.4; 7.0 and 35 mg/kg BW and day (female) respectively) via their drinking water (Sunahara *et al.* 1993). Multiple cases of nephropathy and renal tubular hyperplasia were observed. Further, carcinogenic effects such as an increased incidence of renal tubular tumours in both sexes, and an increased incidence of Leydig cell tumours and fibroadenomas of the mammary gland tissue in male laboratory animals, were reported (Sunahara *et al.* 1993).

The carcinogenic effects described were considered as secondary effects, whereby the occurrence of renal tumours in both sexes could be traced back to 3-MCPD-associated chronic progressive nephropathy. The influence of 3-MCPD on the hormonal balance has been discussed as a cause for the formation of tumours in hormone-responsive tissues, such as the testes and mammary gland tissue in male rats (SCF 2001). The formation of renal hyperplasia has been considered as the most sensitive endpoint in SCF's assessment and a lowest observed adverse effect level (LOAEL; the lowest dose at which a toxic effect is observed in animal experiments) of 1.1 mg/kg BW and day has been identified. The SCF has derived a tolerable daily intake (TDI) of 2 µg/kg BW and day from this LOAEL, taking into account an extrapolation factor of 500 (SCF 2001).

The United Kingdom Committee on Carcinogenicity of Chemicals in Food, Consumer Products and the Environment (COC) has also assessed the genotoxic and carcinogenic potential of 3-MCPD (COC 2000). Considering the same data basis, (Sunahara *et al.* 1993) the COC also concluded that 3-MCPD did not have a genotoxic effect. The carcinogenic effects observed in the kidneys of both sexes, and in the hormone-responsive tissues of male rats (e.g. testes (Sunahara *et al.* 1993)) were also traced back to 3-MCPD-associated nephropathy

and hormonal disorders, and considered to be secondary (COC 2000). On this basis, the COC identified a dose of 1.1 mg/kg BW and day as no observed effect level (NOEL; the highest dose at which no statistically significant treatment-related effects occurred in animal experiments) for the carcinogenic effects observed. Taking into account the NOEL and an extrapolation factor ('*uncertainty factor*') of 1,000, which covers uncertainties regarding the data used (e.g. its quality and incompleteness), the COC concluded that it is unlikely that 3-MCPD represents a carcinogenic risk for humans, as long as the exposition is 1,000 times lower than the NOEL (COC 2000).

In 2002, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) performed a risk assessment on 3-MCPD in foods. In this assessment, the study by Sunahara *et al.* from 1993 was also identified as a key study. JECFA also concluded that 3-MCPD is not genotoxic and that the carcinogenic effects observed in the renal tubule and in hormone-responsive tissue (e.g. the testes) should be considered as a secondary effect (JECFA 2002). The increased occurrence of renal tubular hyperplasia was also identified by JECFA to be the most sensitive endpoint. JECFA identified the dose of 1.1 mg/kg BW and day as the lowest observed effect level (LOEL; the lowest dose at which a significant effect has been observed in animal experiments). Using an uncertainty factor of 500 (which takes into account the extrapolation from the LOEL to the NOEL, along with other uncertainties) a value of 2 µg/kg BW and day was derived for the provisional maximum tolerable daily intake (PMTDI) (JECFA 2002).

Utilising the results from Sunahara *et al*. (Sunahara *et al.* 1993) and current study data from Cho and colleagues (Cho *et al.* 2008), JECFA performed in 2017 a new risk assessment on 3-MCPD and its fatty acid esters (JECFA 2017). In this context, JECFA confirmed that the available experimental data indicates that 3-MCPD fatty acid esters are predominantly hydrolysed into free 3-MCPD in the GIT, and the free compound then mediates the toxic effects (JECFA 2017).

Similar to the work of Sunahara *et al*., rats were treated for a period of 2 years with 3-MCPD (male rats: 0; 2.0; 8.3 or 29.5 mg/kg BW and day; female animals: 0; 2.7; 10.3 or 37.0 mg/kg BW and day) in the study by Cho and colleagues (Cho *et al.* 2008). Exposure was also via drinking water. Carcinogenic effects occurred in the kidneys and hormone-responsive tissues, associated with the administration of 3-MCPD. An increased number of renal tubular adenomas and carcinomas occurred in animals of both sexes in the highest respective dose groups (29.5 and 37 mg/kg BW and day). Furtherore, administration of 3-MCPD to the male rats in the highest dose group (29.5 mg/kg BW and day) was associated with a significantly increased incidence of Leydig cell tumours (Cho *et al.* 2008). Beside these effects, various 3-MCPD-associated non-neoplastic changes, especially in the kidneys, were observed in the animal study. Doses of 2.0; 8.3 and 29.5 mg/kg BW and day (males) and 37.0 mg/kg BW and day (females) led to a significantly increased occurrence of renal tubular hyperplasia. Further, the formation of a chronic progressive nephropathy was noted in both sexes (male rats at: 2.0; 8.3 and 29.5 mg/kg BW and day; female rats at: 10.3 and 37.0 mg/kg BW and day) (Cho *et al.* 2008).

JECFA rated the carcinogenic effects in the kidneys and hormone-responsive (hormone-sensitive) tissues (e.g. the testes) as secondary and traced them back to chronic progressive nephropathy and the influence of 3-MCPD on the hormonal balance respectively (JECFA 2017). From the results of the aforementioned studies (Sunahara *et al.* 1993; Cho *et al.* 2008) , the formation of renal tubular hyperplasia in male rats was identified as the most sensitive endpoint for the effect of 3-MCPD and its fatty acid esters. For this endpoint, a $BMDL_{10}$

(benchmark dose lower confidence limit *10%*; estimated dose which causes an effect incidence of max. 10% in animal experiments with 95% certainty) of 0.87 mg/kg BW and day was calculated using the *benchmark dose* (BMD) approach. Under the assumption that 3-MCPD fatty acid esters are almost completely hydrolysed to free 3-MCPD in the GIT, and taking into account an extrapolation factor of 200 (for considering intraspecies and interspecies differences, and uncertainties with regard to the data situation for reproduction toxic effects), a new group PMTDI for free 3-MCPD and 3-MCPD from fatty acid esters of 4 µg/kg BW and day was derived from this $BMDL_{10}$ value (0.87 mg/kg BW) (JECFA 2017). This value replaced the PMTDI of 2 µg/kg BW and day published by JECFA in 2002 (JECFA 2002). As part of the following assessment and taking into account the performed exposure assessment, JECFA came to the conclusion that the group PMTDI of 4 µg/kg BW and day derived for free 3-MCPD and 3-MCPD from fatty acid esters has not been exhausted in the general population. However, in some countries a 2.5-fold exceedance of this group PMTDI may occur for infants fed with infant and follow-on formula.

The European Food Safety Authority (EFSA) has also assessed the occurrence of 3-MCPD and 3-MCPD fatty acid esters in foods (EFSA 2016; EFSA 2018). Newly acquired occurence and consumption data were considered in the risk assessment performed in 2016 for 3-MCPD and its fatty acid esters in foods (EFSA 2016). Both long-term studies on rats by Sunahara *et al*. and Cho and colleagues were used as key studies (Sunahara *et al.* 1993; Cho *et al.* 2008). The formation of hyperplasias of the renal tubules was identified as the most sensitive endpoint and the proliferation-enhancing effect was considered as a possible mechanism for the carcinogenic effect. EFSA also concluded that the available data did not provide any indications of a genotoxic mode of action (EFSA 2016). From the results of the study by Cho and colleagues (Cho *et al.* 2008) a $BMDL_{10}$ of 0.077 mg/kg BW and day was derived (EFSA 2016). Using an extrapolation factor of 100, which is intended to take into account intraspecies and interspecies differences, a group TDI for free 3-MCPD and 3-MCPD from fatty acid esters of 0.8 µg/kg BW and day was derived. The reason for this assumption was that 3-MCPD fatty acid esters were almost completely hydrolysed to free 3-MCPD in the GIT. The subsequent risk characterisation, which was based on available exposure data demonstrated that the group TDI can mainly be exceeded in younger population groups (‘*younger age groups*’), especially in high consumers (P95) of contaminated foods (EFSA 2016).

In 2018, EFSA updated the risk assessment on 3-MCPD and its fatty acid esters in foods (EFSA 2018) applying a new BMD approach (EFSA 2017b). For the evaluation of renal effects of 3-MCPD, long-term studies on rats by Sunahara *et al*. were used, along with those by Cho and colleagues (Sunahara *et al.* 1993; Cho *et al.* 2008). The formation of a renal tubular hyperplasia was confirmed as the most sensitive endpoint and used for further modelling in this context.

Beside renal effects, 3-MCPD-associated effects on fertility were also considered, focussing on two sub-acute studies by Ban *et al*. and by Kim *et al*. as well as a sub-chronic study on rats by Li and colleagues as key studies (Ban *et al.* 1999; Li *et al.* 2003; Kim *et al.* 2012).

In the study by Ban *et al*., the effect of 3-MCPD on the fertility of male rats was investigated via the percentage change of sperm motility (Ban *et al.* 1999). In this study, the animals (12 and 13 respectively per group) were treated orally (‘*gavage*’) for 9 days with 3-MCPD (dose groups: 0, 1; 3 and 10 mg/kg BW and day). Administration of 3-MCPD led to a significant and dose-related reduction in relevant sperm motility parameters, such as the curvilinear velocity of the sperm in the highest two dose groups of 3 and 10 mg/kg BW and day, respectively (Ban *et al.* 1999).

In the sub-acute study by Kim and colleagues, 3-MCPD was administered orally ('*gavage*') in different doses (0, 3, 10 and 30 mg/kg BW and day) to male rats (6 per dose group) (Kim *et al.* 2012) during a period of 7 days. Amongst other things, the vacuolisation of the epididymal epithelium and sperm motility were investigated as endpoints for the effects of 3-MCPD on fertility. The treatment of the animals led to a concentration-related increase in epididymal cell vacuolisation and a reduction in sperm motility in all dose groups, which was significant at 10 and 30 mg/kg BW and day (Kim *et al.* 2012).

In the third study by Li *et al*., male rats (21 animals per group) were treated for 90 days with 3-MCPD (0; 0.25; 0.5; 1; 2; 4; 8 and 16 mg/kg BW and day) by means of oral administration ('*gavage*') (Li *et al.* 2003). Amongst other things, the sperm count and sperm survival rate were studied with regards to the effects of 3-MCPD on fertility in this sub-chronic study. Administration of 4, 8 and 16 mg 3-MCPD/kg BW and day led to a significant reduction in the sperm count. The sperm survival rate was significantly reduced in rats of the two highest dose groups (8 and 16 mg/kg BW and day) (Li *et al.* 2003).

Together, the results of these studies demonstrated that 3-MCPD has a negative influence on fertility in rats. As part of the assessment of 3-MCPD by the EFSA in 2018, the curvilinear velocity of sperm (Ban *et al.* 1999), epididymal cell vacuolisation (Kim *et al.* 2012) and the sperm count reduction (Li *et al.* 2003) were identified as sensitive endpoints with regards to the effects of 3-MCPD on fertility and used for the BMD model averaging (EFSA 2018).

The endpoints identified with regard to the renal effects and effects on fertility in rats were modelled in accordance with the current EFSA guideline and the results of this analysis were comparatively shown as confidence intervals from the estimated lower limit (benchmark dose lower confidence limit; BMDL) and upper limit (benchmark dose upper confidence limit, BMDU) (EFSA 2018). This comparison led EFSA to the conclusion that the lowest $BMDL_{10}$ value resulted from data of the animal study by Cho and colleagues (0.2 mg 3-MCPD/kg BW and day) for renal tubular hyperplasia in rats (Cho *et al.* 2008). This reference point was lower than the values derived for the other endpoints considered, e g. with regard to fertility in rats, and was therefore considered as the most sensitive endpoint (EFSA 2018). From this $BMDL_{10}$ of 0.20 mg/kg BW and day for renal tubular hyperplasia as the most sensitive endpoint, and applying an extrapolation factor of 100 intended to take into account both intraspecies and interspecies differences, a new group TDI of 2 µg/kg BW and day was derived for free 3-MCPD and 3-MCPD from fatty ester acids (EFSA 2018). The results of the following risk characterisation by EFSA revealed that this group TDI has not been exhausted in the adult population group. Certainly, a slight exceedance of this value was observed for high consumers (P95) in the younger population group ('*younger age*') and especially in infants exclusively fed with infant formula ('*formula only*') (EFSA 2018).

In the context of an opinion on 3-MCPD in bread (BfR 2003b) and a further supplementary toxicological assessment of 3-MCPD, with special consideration given to the risk to children (BfR 2003a), reference was initially made to the underlying scientific data. In the context of the available study results on the genotoxic potential also considered by SCF and JECFA (SCF 2001; JECFA 2002),the BfR came to the conclusion that 3-MCPD is not genotoxic. For further evaluation of the toxic effects of 3-MCPD, results of a long-term study on rats done by Sunahara and colleagues were used as a basis (Sunahara *et al.* 1993) and renal hyperplasia was considered as the most sensitive endpoint. A TDI of 2 µg/kg BW and day was derived from these data (BfR 2003b, a).

In an opinion from 2007, the BfR assessed the health risk of 3-MCPD fatty acid esters in refined plant-based fats, and infant formula as well as follow-on formula (BfR 2007). Assuming a complete hydrolysis of 3-MCPD fatty acid esters in the GIT, the BfR concluded that the TDI

of 2 µg/kg BW and day for 3-MCPD is exceeded by adults with a high consumption of refined plant-based fats, and especially by infants fed with infant formula and follow-on formula (BfR 2007).

In 2012, the BfR performed an update assessment on 3-MCPD and its fatty acid esters in foods (BfR 2012). This was based on novel relevant research data on the toxic properties of 3-MCPD fatty acid esters, such as the abovementioned studies on bioavailability by Buhrke *et al*. and Abraham *et al*. as well as on the results of a longterm study with rats done by Cho and colleagues (Cho *et al.* 2008; Buhrke *et al.* 2011; Abraham *et al.* 2013). Due to the new findings on bioavailability from studies on rats and an *in vitro* model of the GIT with a human cell line, the BfR confirmed the validity of the previous assumption that 3-MCPD fatty acid esters are almost completely hydrolysed in the GIT and absorbed by the organism in the free form (BfR 2012). The results of animal studies done by Sunahara *et al*. and by Cho *et al*. were used as key studies for this risk assessment (Sunahara *et al.* 1993; Cho *et al.* 2008). From the results of both studies, the formation of hyperplasia of the renal tubules was identified as the most sensitive endpoint in rats. Moreover, based on the data obtained by Cho and colleagues (Cho *et al.* 2008), the TDI value of 2 µg 3-MCPD/kg BW and day was confirmed using the BMD approach and applying an extrapolation factor of 100, intended to take intraspecies and interspecies differences into consideration (BfR 2012).

The International Agency for Research on Cancer of the WHO (IARC) classified 3-MCPD in 2013 as '*possibly carcinogenic to humans (Group 2B)*' (IARC 2013). The possibility of a genotoxic effect was not completely ruled out by IARC (IARC 2013).

3.1.2.2.2 2-MCPD and its fatty acid esters

In several opinions on 3-MCPD, BfR and EFSA addressed the possible hazard potential of 2-MCPD and its fatty acid esters (BfR 2012, 2016; EFSA 2016). However, due to the very limited data, no conclusive risk assessment could be performed for these substances as yet.

In an opinion from 2012, BfR described the few available studies on toxic effects of 2-MCPD and its fatty acid esters, most of which are not published. In this opinion it was stated that 2-MCPD has a mutagenic effect *in vitro* in bacteria (Schilter et al. 2011). By contrast, no genotoxic effect could be demonstrated in mammal cells, (Schilter et al. 2011) or in a wing spot test with *Drosophila melanogaster* (Frei and Wurgler 1997). In 1994, a study was carried out on rats regarding the sub-chronic toxicity of 2-MPCD (Schilter *et al.* 2011; EFSA 2016). During this study, rats were orally administered different doses of 2-MCPD (2.16 and 30 mg/kg BW and day) over a period of 28 days. In the highest dose group (30 mg/kg BW and day), a few fatalities occurred, which were traced back to the emergence of heart failure. In this context, it was demonstrated that 16 and 30 mg 2-MCPD/kg BW and day led to severe dose-related damage to smooth muscles, especially in the cardiac muscle of the laboratory animals. This - in turn - resulted in the development of heart failure, which consequently led to deaths of some rats in the treatment group (EFSA 2016). Further, other toxic effects of 2-MCPD were observed in the kidneys of laboratory animals in the highest dose group (30 mg/kg BW and day), such as increased urinary excretion (diuresis), an elevated kidney weight, and histopathological changes to the proximal renal tubule. A NOAEL for 2-MCPD of 2 mg/kg BW and day was identified in this context, based on the observation that no toxic effects occurred in the lowest dose group (EFSA 2016). Further information regarding the toxicity of 2-MCPD and its fatty acid esters was not available. Due to the very limited data, BfR concluded in its opinion from 2012 that a conclusive risk assessment was not possible (BfR 2012).

Utilizing the same data basis, EFSA also came to this conclusion in its opinion on 2-MCPD and its fatty acid esters in foods from 2016, (EFSA 2016). With regard to the very limited data, EFSA stated that further studies would be necessary, especially on the toxicity, mode of action and toxicokinetics of 2-MCPD and its fatty acid esters, to perform a reliable risk assessment. It was also noted that reliable concentration data on these substances in different food groups should be collected (EFSA 2016). The demand for further data on 2-MCPD and its fatty acids was also emphasised by the BfR in an opinion (BfR 2016).

3.1.2.2.3 Glycidol and its fatty acid esters

In 2000, IARC assessed the genotoxic and carcinogenic potential of glycidol. The results of various experiments in bacterial test systems demonstrate that glycidol possesses genotoxic potential *in vitro* (IARC 2000). As an example, the Ames test (bacterial reverse mutation test) revealed positive findings for glycidol in different bacteria strains, e. g. *Salmonella typhimurium* TA100, TA1535 or TA98 (Wade *et al.* 1979; Thompson *et al.* 1981; NTP 1990). In mammal cells, such as human lymphocytes or V79 cells from Chinese hamsters, glycidol also mediated genotoxic effects *in vitro*, e. g. the triggering of genetic mutations, structural chromosomal aberrations and sister chromatid exchanges (Thompson *et al.* 1981; NTP 1990).

Various *in vivo* investigations also indicate a genotoxic potential for glycidol. In a test with *Drosophila melanogaster* (sex-linked recessive lethal mutation assay) a mutagenic effect of glycidol was observed (NTP 1990). Further, a micronucleus test on mice demonstrated that intraperitoneal (i. p.) administration of glycidol in different doses (0 - 150 mg/kg BW, two doses each at intervals of 24 hours) led to positive findings (NTP 1990). The results of a study by Thompson & Hiles on the formation of chromosomal changes in rats after administration of glycidol showed no indications of chromosome- damaging effects (Thompson and Hiles 1981). Taking into account individual findings as well as the reliability of the underlying studies, IARC concluded that glycidol exhibied a genotoxic activity (IARC 2000).

The aforementioned NTP long-term study on mice and rats from 1990 was used by IARC as a key study with regard to assessing the carcinogenic potential of glycidol (NTP 1990). Based on available data from animal experiments, IARC concluded that glycidol has a carcinogenic effect ('*There is sufficient evidence in experimental animals for the carcinogenicity of glycidol*') (IARC 2000). In summary, IARC classified glycidol as '*probably carcinogenic to humans (Group 2A)*', based on scientific data. It was clearly noted that the issue of glycidol-mediated mutagenic effects in different *in vitro* and *in vivo* test systems attract attention in this context(IARC 2000).

Based on initial available occurrence data, the BfR performed in 2009 a preliminary assessment of bound glycidol in refined plant-based cooking fats and foods containing them, such as margarine and infant milk formula (BfR 2009). Due to insufficient data regarding the toxicokinetics, complete hydrolysis of bound glycidol into free glycidol in the organism after intake was assumed as part of a *worst case* scenario.

Results of experiments on rats and mice (10 animals per dose group) during a 13-week study demonstrated that the oral administration ('*gavage*') of glycidol (mice: 0.75, 150 and 300 mg/kg BW and day; rats: 0, 100, 200 and 400 mg/kg BW and day, 5 days/week) led to various toxic effects in both sexes (NTP 1990). In mice, significant neurotoxic damage to the myelin sheats of nerve cells in the brain occurred (in males: 150 mg/kg and day; in females: 300 mg/kg BW and day) as did nephrotoxic effects (degeneration of the renal tubular cells in

males only: 300 mg/kg BW and day) and effects on the fertility of male mice (significantly reduced sperm count at 75 and 150 mg/kg BW and day). Also in rats neurotoxic (necroses in the cerebellum in males: 400 mg/kg BW and day; in females: 200 and 400 mg/kg BW and day) and nephrotoxic effects (degeneration of the renal tubular cells in males and females: 400 mg/kg BW and day) as well as effects on fertility (testicular atrophy in males: 200 and 400 mg/kg BW and day) were observed (NTP 1990).

Chronic toxicity of glycidol was assessed utilizing findings of long-term studies, in which glycidol was orally administered ('*gavage*') over 103 weeks to rats in different concentrations, (0; 37.5 or 75 mg/kg BW and day, every 5 days/weeks; 50 animals/dose group) or mice (0; 25 or 50 mg/kg BW and day, every 5 days/week; 50 animals/dose group) respectively (NTP 1990). Administration of glycidol to mice led to a significantly increased incidence of harderian gland tumours in male (50 mg/kg BW and day) and female animals (25 and 50 mg/kg BW and day). Moreover, further tumours also occurred in other tissues, such as the liver or forestomach, in both sexes in the highest dose group. Also in rats a significantly increased incidence of various tumours (males: e.g. mesotheliomas in the *Tunica vaginalis* and the peritoneum and gliomas of the brain; in females: e.g. tumours of the mammary gland and brain) was observed in both dose groups (37.5 or 75 mg/kg BW and day) and in the highest dose group (males: e.g. tumours of the forestomach and bowel; females: e.g. tumours of the clitoral gland and leukaemias) (NTP 1990). The results of this study (NTP 1990) show that the development of cancer must be considered to be the most important endpoint in case of long-term exposure to small amounts of glycidol (BfR 2009).

Findings of the above mentioned key study (NTP 1990) were used as a basis for the initial assessment on glycidol in refined plant-based fats (BfR 2009). Based on the data, which were also used by IARC for its assessment (IARC 2000), and especially based on the findings of long-term studies on rodents (NTP 1990), BfR concluded that glycidol exhibits genotoxic and carcinogenic effects (BfR 2009). From the long-term study on rats, the formation of a mesothelioma in the *Tunica vaginalis* and in the peritoneum in male rats were identified as the most sensitive endpoints regarding the genotoxic and carcinogenic effect of glycidol (NTP 1990). From the data of this NTP study, BfR derived a T25 value (the chronic dose at which cancer occurs within a certain tissue in 25% of laboratory animals within their lifespan) of 10.2 mg/kg BW and day as a reference point for this endpoint (BfR 2009).

In 2015, the Food Safety Commission of Japan (FSCJ) performed a risk assessment for free and bound glycidol in foods (FSCJ 2015). Based on the same data pool which the BfR had previously used in its risk assessment in 2009, the FSCJ also concluded in 2015 that glycidol is a genotoxic carcinogen (FSCJ 2015). The results of the previously mentioned long-term studies on rats and mice by the *National Toxicology Programme* (NTP) were used as key studies in this risk assessment (NTP 1990). In contrast to the opinion of the BfR in which the T25 values was used (BfR 2009), a $BMDL_{10}$ of 1.6 mg/kg BW and day was directly calculated from the results of this animal study by applying the BMD approach, (FSCJ 2015). The exposure assessment was performed under the assumption that bound glycidol is almost completely hydrolysed into free glycidol in the GIT. Using nationally acquired consumption data, the intake of bound glycidol from cooking oil and from foods manufactured therewith was calculated on average and at a maximum for different population groups (e. g. adults). Based on this data, the FSCJ also used the MOE approach to estimate the health risk from bound glycidol. MOE values have not been calculated for infants fed with infant formula based on these cooking fats. The FSCJ argued in this context that applying the MOE approach to such a risk assessment for infants would be unsuitable, as they consume infant formula only for a limited time period (during infancy) (FSCJ 2015).

Due to its genotoxic and carcinogenic potential, the **Senate Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area** ('MAK Commission') classified glycidol in 2015 in Category 2, 'Carcinogenic', and Category 3A, 'Germ Cell Mutagenic Effect' (DFG 2015).

EFSA also performed a risk assessment for bound glycidol in foods in 2016. In accordance with previous assessments by other national and international institutions (IARC 2000; BfR 2009; FSCJ 2015), EFSA also came to the conclusion that glycidol is genotoxic and carcinogenic (EFSA 2016). EFSA used occurence data provided by the EU member states for the exposure assessment. However, these data did not contain any information on the concentrations of free glycidol in foods. High concentrations of bound glycidol have been particularly found in oils and fats, and in foods which contain these, such as crisps or biscuits. As well as the aforementioned nephrotoxicity, neurotoxicity and reproductive toxicity (NTP 1990) , the carcinogenic potential of glycidol was particularly identified as a relevant endpoint (EFSA 2016). The dose-effect relationship for glycidol was therefore determined on the basis of animal studies on the emergence of tumours in various tissues (NTP 1990). The panel considered these dataan unsuited for BMD modelling. Therefore, the T25 was used as reference point for calculating the MOE with regard to the neoplastic effects in rats. This was in line with the assessment already published by BfR in the context of the risk assessment on bound glycidol carried out in 2009 (BfR 2009). As glycidol exhibits both a genotoxic and a carcinogenic potential, EFSA concluded that it would be not applicable to derive a TDI for further risk assessment of this substance. Therefore, the T25 of 10.2 mg/kg BW and day obtained from animal experiments was used to estimate the risk for different population groups in different consumption scenarios, using the MOE approach (EFSA 2016).

In 2017, JECFA performed a risk assessment for bound glycidol (JECFA 2017). In this assessment, it was critically noted that the available scientific data are very limited. However, taking into account previously mentioned available toxicokinetic data (Nomeir *et al.* 1995; Melnick 2002; Wakabayashi *et al.* 2012; Appel *et al.* 2013; Aasa *et al.* 2016), JEFCA concluded that bound glycidol is predominantly hydrolysed in the GIT into the genotoxic carcinogen glycidol, which can mediate health-damaging effects (e.g. carcinogenic effects). Therefore, the further assessment of the risk was done under the assumption that bound glycidol is almost completely hydrolysed into free glycidol in the GIT.

Based on the aforementioned studies on the genotoxic potential of glycidol (NTP 1990) described for example by IARC (IARC 2000) as well as newer data which substantiated this finding (Ikeda *et al.* 2012), JECFA confirmed that this substance is a genotoxic carcinogen (JECFA 2017). Beside other toxic effects, such as reproductive toxicity or immunotoxicity, the genotoxic and carcinogenic effect of glycidol was confirmed as its most significant toxic effect, especially due to the results of the aforementioned long-term study by the NTP on rodents (NTP 1990). Using these data – in accordance with the estimations of the BfR (BfR 2009) – the occurrence of mesotheliomas in the *Tunica vaginalis* and in the peritoneum of male rats was identified as the most sensitive endpoint for the further assessment (NTP 1990). A $BMDL_{10}$ of 2.4 mg/kg BW and day was derived for this endpoint utilzing the BMD approach. JECFA confirmed that it is not possible to derive a health-based guidance value for substances with genotoxic and carcinogenic effects, such as glycidol and its fatty acid esters. Therefore, the MOE approach was used for risk assessment.

3.1.3 Exposure

3.1.3.1 Data basis

3.1.3.1.1 Consumption data

Regarding the consumption by adolescents and adults, the National Nutrition Survey II (NVS II) by the Max Rubner Institute (MRI) was used as a data basis. The NVS II is the current representative study on the consumption habits of the German population. This study, which surveyed about 20,000 individuals between the ages of 14 and 80 using three different survey methods (diet history, 24-hour recall and weighing protocol), was done between 2005 and 2006 throughout Germany (Krems *et al.* 2006; MRI 2008). The consumption assessments considered in the present opinion are based on the data from the diet history interviews of NVS II, which was gathered with the help of the 'DISHES 05' programme. With the diet history method, 15,371 people were surveyed and their usual consumption habits were retrospectively recorded over the last four weeks. This provided good estimations of the long-term intake of substances when foods were summarised in groups or when foods which were regularly consumed were taken into account.

The consumption study for recording the food intake of infants and toddlers for assessing an acute toxicity risk from plant protection product residues (VELS) was used as a data basis for consumption by children under 5 years (Heseker *et al.* 2003; Banasiak *et al.* 2005). Between 2001 and 2002, the study was conducted on 816 infants and toddlers between the ages of 6 months and less than 5 years all over Germany. Parents recorded all consumed food in two 3-day nutritional protocols for each child. Due to the presence of single-day consumption data, two 3-day nutritional protocols are suitable for exposure assessments for both acute and chronic risks.

3.1.3.1.2 Occurrence data

At the time of writing, BfR held data on 3-MCPD, 2-MCPD and their fatty acid esters, and on bound glycidol in food from three sources. These were project data from the decision-support project 'Investigation of the occurrence of 3-MCPD fatty acid esters and related compounds in foods' (BLE 2017) and the project 'Minimisation of 2- and 3-MCPD, glycidol and their fatty acid esters in smoked and heat-treated fish products' carried out by the MRI (FEI 2017). On the other hand, data was available from the German Federal Office of Consumer Protection and Food Safety (BVL).

The project data was gathered on the order of the German Federal Office for Agriculture and Food (BLE) for the German Federal Ministry of Food and Agriculture (BMEL), whereby current analysis methods were used. Based on market data, SGS Germany GmbH investigated 1051 individual samples of 'croissants', 'donuts', 'sandwich spreads', 'French fries', 'infant formulas', 'frying fats', 'margarines', 'cooking oils' and 'Asian dishes with instant noodles' in 2016. The number of samples for each food (group) was between 20 and 221. In addition, 229 individual samples of 'fried herring products', 'tinned fish', 'breaded pre-fried frozen fish products' and 'smoked fish' were also investigated by the MRI in 2017. The number of samples for each food group was between 40 and 80. The MRI also investigated eleven bread samples, in which the concentration of the contaminants (see below) were below the limit of quantification in all cases. In the project, all foods were analysed for their concentrations of free 3-MCPD and 2-MCPD, and 3-MCPD and 2-MCPD fatty acid esters (buond 3- and 2-

MCPD) and glycidyl fatty acid esters (buond glycidol). The exceptions were ‘fried herring products’, ‘non-smoked tinned fish’ and ‘breaded pre-fried frozen fish products’. As previous investigations showed that the concentrations of free 3- and 2-MCPD in these food groups were below the limit of quantification of 10 µg/kg, these compounds were not quantified by the MRI. Under the assumption that the concentration of free compounds in all samples is below the limit of quantification, the corresponding data was treated accordingly in a further procedure. The concentration of free glycidol was not determined in the project, as it is currently generally assumed that it is unstable due to its high reactivity in a complex food matrix and does not occur as such.

The BVL submitted data on samples from the period 2000 to 2017. Due to extensive changes regarding the analytical method in recent years, the data gathered before 2012 were excluded from the evaluation performed here. Samples that were collected as complaint samples, NRKP[2] samples, import samples, suspect samples and follow-up samples, along with samples with the label ‘Other reasons for sampling and disclosure’, were also excluded. The remaining 5,929 data sets from 3,193 samples were then subjected to a more detailed inspection. The BVL data was relatively inhomogeneous, with regard to analytes, limits of detection and limits of quantification, even within the food groups.

After statistical evaluation of the contents, BVL data were compared with the project. The data available from both sources on the same food groups were compared. In addition, further food groups with relevant contents were identified in the BVL data, including "smoked sausage and meat products", "biscuits", "snack products made from potatoes" and "seasoning sauces" (soy sauce, liquid seasonings (protein hydrolysates)).

In principle, only concentration data which fulfilled the following selection criteria was used for the exposure assessment:

- at least 20 samples per food (group) in order to be able to estimate a 95th percentile of concentration data which differs from the maximum concentration
- availability of concentration data for 3- and 2-MCPD for both the free and buond forms in the same sample, as the free and buond concentrations must be summed for comparison with the group TDI. If both the BVL and project data for a food group fulfil these criteria, only the project data will be used for the exposure assessment, as:
- the analytical methods used for it were known and uniform
- the number of samples per food group was higher (exception: ‘other oils’)
- the limits of quantification were lower, meaning that there was a significantly lower number of undetected values

Using this approach, 19 food groups were selected for the exposure assessment. Almost all project data was included, with the exception of 33 individual samples of frozen ‘French fries’, 12 individual samples of ‘breaded frozen fish products’ and 50 individual samples of ‘Asian dishes with instant noodles’ (for the rationale, see also Section 3.1.3.2.6 ‘Further possible sources of exposure’*)*. The occurrence data for bread was included, despite the number

[2] National residue control plan

of samples (N = 11) being low. It must also be noted that all measured values for bread were below the limit of quantification. Since bread has a high relevance for consumption, this approach was selected for the upper bound estimation in a preventative sense, but has no relevance for the lower bound estimation. The data for sauces was used from the BVL data, although these were only investigated for 3-MCPD (as also practised in the EFSA assessment of 2016).

When preparing the selected data for the exposure assessment, the values below the limits of quantification and limits of detection were replaced by 0 (lower bound) or by the appropriate limit (upper bound) (see Table 1, footnote '[1]'). The average concentrations of the free and bound forms of 3-MCPD and 2-MCPD, respectively, were summed as a basis for the exposure assessment, with the exception of sauces.

Table 1: Limits of quantification for 3-MCPD, 2-MCPD, their buond forms and bound glycidol in 19 food groups in µg/kg.

Food group		Data source	LOQ (µg/kg)	
			Free compounds	Fatty acid esters
Baked goods	Bread and bread rolls	Project (MRI)	15	15
	Croissants and filled pastries for children	Project	5	10
	Donuts and Berliners	Project	5	10
Sandwich spreads	Savoury sandwich spreads	Project	5	10
	Sweet sandwich spreads	Project	5	10
French fries	French fries, outside the home	Project	5	10
Infant formula	Infant formula	Project	5	10
Fats and oils	Frying fats	Project	5	10
	Margarines and vegetable fats	Project	5	10
	Olive oil	Project	5	10
	Rapeseed oil	Project	5	10
	Sunflower oil	Project	5	10
	Other oils	Project	5	10
Fish products	Fried herring products	Project (MRI)	10	20
	Tinned fish	Project (MRI)	10	20
	Breaded pre-fried frozen fish products	Project (MRI)	10	20
	Smoked fish	Project (MRI)	10	20
Sauces	Liquid condiments	BVL	5 - 12.5 (3 - 50)[1]	N/A
	Soy sauce	BVL	4 - 20 (2 - 10)[1]	N/A

LOQ: *limit of quantification*, n. a.: not analysed

[1] in brackets the LOD: limit of detection

The limits of detection and limits of quantification for the considered foods (groups) are listed in Table 1. Table 2 shows an overview of the foods (groups) considered in the exposure assessment, the number of measured results of individual analytes and their sources, and an extensive presentation of occurrence data.

Table 2: Concentrations of 3-MCPD, 2-MCPD, their bound forms and bound glycidol in 19 food groups in µg/kg.

			Free 3-MCPD	Bound 3-MCPD	∑ 3-MCPD	Free 2-MCPD	Bound 2-MCPD	∑ 2-MCPD	Bound glycidol
Baked goods	Bread and bread rolls[2]	N	11						
		< LOQ (%)	100	100	-	100	100	-	100
		Mean lb	0	0	0	0	0	0	0
		Mean ub	15	15	30	15	15	30	15
		Median lb	0	0	-	0	0	-	0
		Median ub	15	15	-	15	15	-	15
		95th percentile lb	0	0	-	0	0	-	0
		95th percentile ub	15	15	-	15	15	-	15
	Croissants and filled pastries for children[1]	N	100						
		< LOQ (%)	10	47	-	89	66	-	59
		Mean lb	21	136	157	2	49	51	107
		Mean ub	21	141	162	7	56	62	113
		Median lb	10	14	-	0	0	-	0
		Median ub	10	14	-	5	10	-	10
		95th percentile lb	60	743	-	8	294	-	706
		95th percentile ub	60	743	-	8	294	-	706
	Donuts and Berliners[1]	N	50						
		< LOQ (%)	58	0	-	98	2	-	2
		Mean lb	6	507	513	0	268	268	435
		Mean ub	8	507	515	5	268	274	435
		Median lb	6	302	-	0	167	-	359
		Median ub	6	302	-	5	167	-	359
		95th percentile lb	21	716	-	0	420	-	1,095
		95th percentile ub	21	716	-	5	420	-	1,095
Sandwich spreads[1]	Savoury snacks[a]	N	50						
		< LOQ (%)	98	0	-	100	4	-	6
		Mean lb	0	229	229	0	103	103	71
		Mean ub	5	229	234	5	103	108	72
		Median lb	0	263	-	0	124	-	68
		Median ub	5	263	-	5	124	-	68
		95th percentile lb	0	463	-	0	195	-	221
		95th percentile ub	5	463	-	5	195	-	221
	Sweets[b]	N	50						
		< LOQ (%)	98	10	-	100	10	-	10
		Mean lb	0	196	196	0	92	92	99
		Mean ub	5	197	202	5	93	98	99
		Median lb	0	182	-	0	87	-	87
		Median ub	5	182	-	5	87	-	87
		95th percentile lb	0	484	-	0	234	-	289
		95th percentile ub	5	484	-	5	234	-	289

- *Table 2 continued* -

			Free 3-MCPD	Bound 3-MCPD	∑ 3-MCPD	Free 2-MCPD	Bound 2-MCPD	∑ 2-MCPD	Bound glycidol
French fries[1]	Outside the home	N	67						
		< LOQ (%)	48	1	-	76	1	-	4
		Mean lb	26	111	137	9	68	77	118
		Mean ub	29	111	140	13	68	81	119
		Median lb	6	90	-	0	51	-	82
		Median ub	6	90	-	5	51	-	82
		95th percentile lb	124	469	-	59	217	-	392
		95th percentile ub	124	469	-	59	217	-	392
Infant formula[1c]	Infant formula	N	221						
		< LOQ (%)	100	0	-	100	2	-	55
		Mean lb	0	137	137	0	53	53	27
		Mean ub	5	137	142	5	54	59	32
		Median lb	0	103	-	0	38	-	0
		Median ub	5	103	-	5	38	-	10
		95th percentile lb	0	283	-	0	108	-	159
		95th percentile ub	5	283	-	5	108	-	159
Fats and oils[1]	Frying fats	N	50						
		< LOQ (%)	100	0	-	100	0	-	0
		Mean lb	0	1,591	1.591	0	756	756	1,481
		Mean ub	5	1,591	1.596	5	756	761	1,481
		Median lb	0	1,70	-	0	706	-	1,006
		Median ub	5	1,570	-	5	706	-	1,006
		95th percentile lb	0	2,941	-	0	1,439	-	5,047
		95th percentile ub	5	2,941	-	5	1,439	-	5,047
	Margarines and vegetable fats	N	200						
		< LOQ (%)	97	0	-	100	0	-	0
		Mean lb	1	398	399	0	188	188	224
		Mean ub	5	398	404	5	188	193	224
		Median lb	0	384	-	0	180	-	204
		Median ub	5	384	-	5	180	-	204
		95th percentile lb	0	698	-	0	344	-	390
		95th percentile ub	5	698	-	5	344	-	390
	Olive oil	N	20						
		< LOQ (%)	100	5	-	100	5	-	5
		Mean lb	0	395	395	0	171	171	428
		Mean ub	5	396	401	5	172	177	428
		Median lb	0	385	-	0	167	-	307
		Median ub	5	385	-	5	167	-	307
		95th percentile lb	0	711	-	0	332	-	1,416
		95th percentile ub	5	711	-	5	332	-	1,416

- Table 2 continued -

			Free 3-MCPD	Bound 3-MCPD	∑ 3-MCPD	Free 2-MCPD	Bound 2-MCPD	∑ 2-MCPD	Bound glycidol
Fats and oils[1]	Rapeseed oil	N	50						
		< LOQ (%)	100	0	-	100	8	-	0
		Mean lb	0	172	172	0	69	69	176
		Mean ub	5	172	177	5	70	75	176
		Median lb	0	70	-	0	25	-	157
		Median ub	5	70	-	5	25	-	157
		95th percentile lb	0	596	-	0	261	-	343
		95th percentile ub	5	596	-	5	261	-	343
	Sunflower oil	N	58						
		< LOQ (%)	100	0	-	100	0	-	0
		Mean lb	0	376	376	0	176	176	412
		Mean ub	5	376	381	5	176	181	412
		Median lb	0	324	-	0	129	-	299
		Median ub	5	324	-	5	129	-	299
		95th percentile lb	0	953	-	0	462	-	1,233
		95th percentile ub	5	953	-	5	462	-	1,233
	Other oils[d]	N	52						
		< LOQ (%)	96	6	-	100	8	-	8
		Mean lb	0	1,371	1,372	0	619	619	578
		Mean ub	5	1,372	1,377	5	620	625	578
		Median lb	0	912	-	0	270	-	516
		Median ub	5	912	-	5	270	-	516
		95th percentile lb	3	3,663	-	0	1,727	-	1,433
		95th percentile ub	6	3,663	-	5	1,727	-	1,433
Fish products[2]	Fried herring	N	40						
		< LOQ (%)	100	13	-	100	43	-	50
		Mean lb	0	177	177	0	69	69	39
		Mean ub	10	180	190	10	77	87	49
		Median lb	0	99	-	0	30	-	10
		Median ub	10	99	-	10	30	-	20
		95th percentile lb	0	710	-	0	347	-	187
		95th percentile ub	10	710	-	10	347	-	187
	Tinned food	N	79						
		< LOQ (%)	84	56	-	97	80	-	47
		Mean lb	3	48	51	0	12	12	35
		Mean ub	11	59	70	7	28	35	44
		Median lb	0	0	-	0	0	-	24
		Median ub	10	20	-	10	20	-	24
		95th percentile lb	20	259	-	0	67	-	117
		95th percentile ub	20	259	-	10	67	-	117

- *Table 2 continued* -

			Free 3-MCPD	Bound 3-MCPD	∑ 3-MCPD	Free 2-MCPD	Bound 2-MCPD	∑ 2-MCPD	Bound glycidol
Fish products[2]	Breaded, pre-fried, frozen	N	40						
		< LOQ (%)	100	5	-	100	18	-	78
		Mean lb	0	231	231	0	94	94	8
		Mean ub	10	232	242	10	97	107	23
		Median lb	0	159	-	0	54	-	0
		Median ub	10	159	-	10	54	-	20
		95th percentile lb	0	678	-	0	363	-	49
		95th percentile ub	10	678	-	10	363	-	49
	Smoked	N	80						
		< LOQ (%)	29	90	-	100	99	-	70
		Mean lb	32	5	37	0	1	1	14
		Mean ub	34	23	57	4	20	24	28
		Median lb	18	0	-	0	0	-	0
		Median ub	18	20	-	3	20	-	20
		95th percentile lb	118	38	-	0	0	-	69
		95th percentile ub	118	38	-	10	20	-	69
Sauces[3]*	Liquid condiments	N	88	-	-	-	-	-	-
		< LOQ/LOD (%)	82	-	-	-	-	-	-
		Mean lb	26	-	-	-	-	-	-
		Mean ub	32	-	-	-	-	-	-
		Median lb	0	-	-	-	-	-	-
		Median ub	10	-	-	-	-	-	-
		95th percentile lb	12	-	-	-	-	-	-
		95th percentile ub	17	-	-	-	-	-	-
	Soy sauce	N	399	-	-	-	-	-	-
		< LOQ/LOD (%)	74	-	-	-	-	-	-
		Mean lb	14	-	-	-	-	-	-
		Mean ub	18	-	-	-	-	-	-
		Median lb	0	-	-	-	-	-	-
		Median ub	5	-	-	-	-	-	-
		95th percentile lb	11	-	-	-	-	-	-
		95th percentile ub	11	-	-	-	-	-	-

LOQ: limit of quantification, LOD: limit of detection
lb: lower bound, ub: upper bound
Data source: [1] Project (SGS Fresenius), [2] project (MRI), [3] BVL
[a] Clotted cream, animal fat, vegan spreads
[b] Nut nougat creams (N=44), peanut butter (N=6)
[c] Powder
[d] Corn, sesame, peanut, safflower, hazel nut, walnut, linseed, grape seed, pumpkin seed, red palm and wheat germ oil
* only free 3-MCPD analysed

The highest concentrations of 3-MCPD, 2-MCPD, their bound forms and bound glycidol were detected in the ‘frying fats’ and ‘other oils’ food groups.

A differentiated comparison of individual analytes between food groups (see also Appendix Ia-c) also shows by far the highest concentrations of 3-MCPD (the sum of free and bound forms) in ‘frying fats’ and ‘other oils’ (median lb - ub: 1,372 - 1,596 µg/kg). The other ‘fats and oils’ (exception: ‘rapeseed oil’) and ‘donuts/Berliners’ had concentrations in the median range (376 - 515 µg/kg). ‘Croissants/filled pastries for children’, ‘sandwich spreads’, ‘French fries outside the home’, ‘infant formula’, ‘rapeseed oil’, ‘fried herring products’ and ‘breaded pre-fried frozen fish products’ have somewhat lower concentrations (137 - 242 µg/kg). ‘Bread/bread rolls’, ‘tinned fish’, ‘smoked fish’ and ‘sauces’ had the lowest concentrations (0 - 70 µg/kg).

The comparison of 2-MCPD concentrations (sum of free and bound forms) showed a similar distribution between food groups with generally lower values. The highest concentrations by far were also detected in ‘frying fats’ and ‘other oils’ (619 - 761 µg/kg). The concentrations of other ‘fats and oils’ (exception: ‘rapeseed oil’) and ‘donuts/Berliners’ were also in the medium range (171 - 274 µg/kg), while the values of other food groups investigated were lower (0 - 108 µg/kg).

When comparing the contents of bound glycidol, the ‘frying fats’ group also stood out with very high concentrations (1481 µg/kg). The other ‘fats and oils’ and ‘donuts/Berliners’ followe at a clear distance (176 - 578 µg/kg), while the values for other food groups were lower (0 - 119 µg/kg).

3.1.3.2 Exposure assessment

The exposure assessment was performed using SPSS Version 21 individually for 3-MCPD, 2-MCPD (as the respective sums of free and buond forms) and for bound glycidol. Only the long-term consumption of the relevant foods was considered.

Consumption data for infant formula was partly for powder and partly for ready-to-eat preparations. As there was only occurrence data for the powdered form, consumption data for ready-to-eat preparations was converted into powder applying a factor of 7.7 (following the EFSA assessment from 2016).

The evaluation of consumption data revealed a very high proportion of unspecified oils, especially among adults, which originated from recipes stored in the German Federal Food Code. For the exposure assessment, ‘plant oils with linoleic acid < 30%’ were assigned to the ‘rapeseed oil’ group and ‘plant oils with linoleic acid 30 - 60%’ to the ‘sunflower oil’ group, based on their fatty acid patterns.

3.1.3.2.1 Intake of 3-MCPD, 2-MCPD (respective sum of free and bound forms) and bound glycidol from individual food groups

Median concentrations were linked with consumption quantities by children and adults for all test subjects on an individual level, and the median and the 95th percentile of intakes for each food group were considered. The use of median concentrations represents the average consumer, who sometimes consumes more contaminated and sometimes less contaminated sorts of the food under consideration. The median intake represents normal consumers and the 95th percentile represents high consumers.

The intake quantities of 3-MCPD, 2-MCPD (sum of free and bound form) and of bound glycidol for children and adults for individual food groups are listed in Tables 3a-c, based on

all respondents, and 4a-c, based on consumers only. The percentages of consumers among the total number of respondents vary greatly, both between individual food groups and between children and adults.

The food groups ‘infant formula’, ‘donuts/Berliners’, ‘breaded pre-fried frozen fish products’ and ‘French fries outside the home’ make up the largest contribution to the median intake of 3-MCPD, 2-MCPD, their bound forms and bound glycidol in children (only consumers). For 3-MCPD and 2-MCPD (respective sum of free and bound forms) ‘infant formula’ is in first place with 0.42 - 0.43 and 0.16 - 0.18 µg/kg BW and day (median lb - ub), followed by ‘donuts/Berliners’ and ‘breaded pre-fried frozen fish products’. For bound glycidol, ‘donuts/Berliners’ are in first place with 0.23 µg/kg BW and day, followed by ‘French fries outside the home’ and ‘infant formula’. The consumer percentage of the above groups is between 8 - 24%. Due to the high percentage of consumers (97%), despite having a somewhat lower intake percentage (0.04 - 0.08 µg/kg BW and day), ‘margarines/vegetable fats’ are also relevant to children being exposed to 3-MCPD, 2-MCPD (sum of free and bound forms) and bound glycidol.

The highest contribution to the median intake of 3-MCPD, 2-MCPD, their bound forms and bound glycidol in adults (consumers only) results from the food groups of ‘bread/bread rolls, ‘frying fats’ and ‘sweet sandwich spreads’ (88% of samples: nut nougat creams). However, the ub scenario of the exposure estimate for the ‘bread/bread rolls’ food group is subject to major uncertainties, as all measured values were below the limit of quantification. The number of samples was also very low (N = 11). For 94% of adults, 0.04 µg/kg BW and day of 3-MCPD, 0.02 µg/kg BW and day of 2-MCPD and 0.04 µg/kg BW and day of bound glycidol (median lb = ub) were consumed via ‘frying fats’. The highest median intake of 3-MCPD and bound glycidol was also revealed to be from the ‘croissants/filled pastries for children’ food group and in the case of 2-MCPD, from the ‘breaded pre-fried frozen fish products’ food group for consumers. ‘Donuts/Berliners’ are also relevant for the median intake of the three substances (0.02 - 0.03 µg/kg BW and day) for a very low percentage of adult consumers (3%).

The exposure of children (0.5 - < 5 years) and adults (14 - 80 years) to these substances is very different in terms of food groups, due to the different consumption behaviours of these population groups. This can be seen directly in the percentages of consumers depicted in Table 4a-c. For example, in the children’s group, a consumer percentage of 17.1% can be found for infant formula, while no consumers of this food group could be found among the adults. On the other hand, 1.3% of adults consumed ‘fried herring products’, while no consumers of this food group could be found among the children. For other food groups, such as ‘tinned fish’, it was demonstrated that the percentage of consumers was far higher among the adults at 34.2% than among the children at only 2.9%. For other food groups, such as ‘bread and bread rolls (94.1% vs. 98.4%) or ‘margarines and vegetable fats’ (97.1% vs. 96.8%), the percentage of consumers was at a comparable level for both children and adults (see Table 4a).

Table 3a: Median intake (monthly average) from the sum of free and bound 3-MCPD from 19 food groups in µg/kg BW and day (basis: all respondents).

		Children (N=732)[1]		Adults (N=15,371)[2]	
		lb	ub	lb	ub
Bread and bread rolls	Median	0*	0.064	0*	0.054
	95th percentile	0*	0.168	0*	0.133
Croissants and pastries for children	Median	0	0	0	0
	95th percentile	0.107	0.111	0.027	0.028
Donuts and Berliners	Median	0	0	0	0
	95th percentile	0.197	0.198	0.000	0.000
Savoury sandwich spreads	Median	0	0	0	0
	95th percentile	0.027	0.028	0	0
Sweet sandwich spreads	Median	0	0	0	0
	95th percentile	0.103	0.107	0.038	0.039
French fries, outside the home	Median	0	0	0	0
	95th percentile	0.220	0.224	0.036	0.037
Infant formula	Median	0	0	no consumers	
	95th percentile	0.575	0.596		
Frying fats	Median	0.037	0.037	0.041	0.041
	95th percentile	0.371	0.372	0.155	0.155
Margarines and vegetable fats	Median	0.077	0.078	0.009	0.009
	95th percentile	0.304	0.308	0.146	0.148
Olive oil	Median	0.011	0.012	0.005	0.005
	95th percentile	0.287	0.292	0.051	0.051
Rapeseed oil	Median	0.014	0.014	0	0
	95th percentile	0.079	0.081	0.006	0.006
Sunflower oil	Median	0	0	0.014	0.014
	95th percentile	0.056	0.056	0.054	0.055
Other oils	Median	0	0	0.007	0.007
	95th percentile	0.379	0.380	0.060	0.060
Fried herring products	Median	no consumers		0	0
	percentile 96			0	0
Tinned fish	Median	0	0	0	0
	95th percentile	0	0	0.011	0.016
Breaded pre-fried frozen fish products	Median	0	0	0	0
	95th percentile	0.244	0.256	0.030	0.032
Smoked fish	Median	0	0	0	0
	95th percentile	0.006	0.010	0.006	0.010
Liquid condiments	Median	0	0	0	0
	95th percentile	0.0002	0.0002	0.0001	0.0002
Soy sauce	Median	0	0	0	0
	95th percentile	0	0	0.0001	0.0001

lb: lower bound, ub: upper bound
[1] children who are no longer breastfed (0.5 to under 5 years), [2] adolescents and adults (14-80 years)
* all measured values < LOQ median = 0, if percentage of consumers < 50%; 95th percentile = 0, if percentage of consumers < 5%

Table 3b: Median intake (monthly average) of the sum of free and bound 2-MCPD from 17 food groups in µg/kg BW and day (basis: all respondents).

		Children (N=732)[1]		Adults (N=15,371)[2]	
		lb	ub	lb	ub
Bread and bread rolls	Median	0*	0.064	0*	0.054
	95th percentile	0*	0.168	0*	0.133
Croissants and pastries for children	Median	0	0	0	0
	95th percentile	0.035	0.042	0.009	0.011
Donuts and Berliners	Median	0	0	0	0
	95th percentile	0.103	0.105	0	0
Savoury sandwich spreads	Median	0	0	0	0
	95th percentile	0.012	0.013	0	0
Sweet sandwich spreads	Median	0	0	0	0
	95th percentile	0.049	0.052	0.018	0.019
French fries, outside the home	Median	0	0	0	0
	95th percentile	0.123	0.130	0.020	0.021
Infant formula	Median	0	0	no consumers	
	95th percentile	0.222	0.248		
Frying fats	Median	0.018	0.018	0.020	0.020
	95th percentile	0.176	0.177	0.073	0.074
Margarines and vegetable fats	Median	0.036	0.037	0.004	0.004
	95th percentile	0.143	0.147	0.069	0.071
Olive oil	Median	0.005	0.005	0.002	0.002
	95th percentile	0.124	0.129	0.022	0.023
Rapeseed oil	Median	0.006	0.006	0	0
	95th percentile	0.032	0.034	0.002	0.003
Sunflower oil	Median	0	0	0.006	0.007
	95th percentile	0.026	0.027	0.025	0.026
Other oils	Median	0	0	0.003	0.003
	95th percentile	0.171	0.172	0.027	0.027
Fried herring products	Median	no consumers		0	0
	percentile 96			0	0
Tinned fish	Median	0	0	0	0
	95th percentile	0	0	0.003	0.008
Breaded pre-fried frozen fish products	Median	0	0	0	0
	95th percentile	0.099	0.113	0.012	0.014
Smoked fish	Median	0	0	0	0
	95th percentile	0.0002	0.004	0.0002	0.0041

lb: lower bound, ub: upper bound
[1] children who are no longer breastfed (0.5 to under 5 years), [2] adolescents and adults (14-80 years)
* all measured values < LOQ
Median = 0, if percentage of consumers < 50%; 95th percentile = 0, if percentage of consumers < 5%

Table 3c: Median intake (monthly average) of bound glycidol from 17 food groups in µg/kg BW and day (basis: all respondents).

		Children (N=732)[1]		Adults (N=15,371)[2]	
		lb	ub	lb	ub
Bread and bread rolls	Median	0*	0.032	0*	0.027
	95th percentile	0*	0.084	0*	0.067
Croissants and pastries for children	Median	0	0	0	0
	95th percentile	0.073	0.077	0.019	0.020
Donuts and Berliners	Median	0	0	0	0
	95th percentile	0.167	0.167	0	0
Savoury sandwich spreads	Median	0	0	0	0
	95th percentile	0.008	0.009	0	0
Sweet sandwich spreads	Median	0	0	0	0
	95th percentile	0.052	0.052	0.019	0.019
French fries, outside the home	Median	0	0	0	0
	95th percentile	0.189	0.191	0.031	0.031
Infant formula	Median	0	0	no consumers	
	95th percentile	0.113	0.134		
Frying fats	Median	0.035	0.035	0.038	0.038
	95th percentile	0.345	0.345	0.144	0.144
Margarines and vegetable fats	Median	0.043	0.043	0.005	0.005
	95th percentile	0.171	0.171	0.082	0.082
Olive oil	Median	0.012	0.012	0.005	0.005
	95th percentile	0.311	0.311	0.055	0.055
Rapeseed oil	Median	0.014	0.014	0	0
	95th percentile	0.081	0.081	0.006	0.006
Sunflower oil	Median	0	0	0.015	0.015
	95th percentile	0.061	0.061	0.059	0.059
Other oils	Median	0	0	0.003	0.003
	95th percentile	0.159	0.159	0.025	0.025
Fried herring products	Median	no consumers		0	0
	percentile 96			0	0
Tinned fish	Median	0	0	0	0
	95th percentile	0	0	0.008	0.010
Breaded pre-fried frozen fish products	Median	0	0	0	0
	95th percentile	0.008	0.024	0.001	0.003
Smoked fish	Median	0	0	0	0
	95th percentile	0.002	0.005	0.002	0.005

lb: lower bound, ub: upper bound
[1] children who are no longer breastfed (0.5 to under 5 years), [2] adolescents and adults (14-80 years)
* all measured values < LOQ
Median = 0, if percentage of consumers < 50 %; 95th percentile = 0, if percentage of consumers < 5 %

Table 4a: Median intake (monthly average) of the sum of free and bound 3-MCPD from 19 food groups in µg/kg BW and day (basis: only consumers).

		Children (N=732)[1]		Adults (N=15,371)[2]	
		lb	ub	lb	ub
Bread and bread rolls	Percentage of consumers (%)	94.1		98.4	
	Median	0	0.068	0	0.054
	95th percentile	0	0.169	0	0.134
Croissants and filled pastries for children	Percentage of consumers (%)	14.1		12.5	
	Median	0.085	0.088	0.022	0.023
	95th percentile	0.216	0.223	0.120	0.124
Donuts and Berliners	Percentage of consumers (%)	7.7		2.9	
	Median	0.273	0.275	0.031	0.031
	95th percentile	1.012	1.016	0.202	0.203
Savoury sandwich spreads	Percentage of consumers (%)	7.8		4.3	
	Median	0.036	0.037	0.023	0.024
	95th percentile	0.149	0.152	0.139	0.142
Sweet sandwich spreads	Percentage of consumers (%)	42.2		14.8	
	Median	0.040	0.041	0.023	0.024
	95th percentile	0.163	0.168	0.148	0.152
French fries, outside the home	Percentage of consumers (%)	23.8		40.0	
	Median	0.112	0.115	0.012	0.012
	95th percentile	0.329	0.336	0.066	0.067
Infant formula	Percentage of consumers (%)	17.1		no consumers	
	Median	0.418	0.433		
	95th percentile	1.124	1.165		
Frying fats	Percentage of consumers (%)	67.2		94.4	
	Median	0.079	0.079	0.044	0.044
	95th percentile	0.430	0.432	0.159	0.160
Margarines and vegetable fats	Percentage of consumers (%)	97.1		96.8	
	Median	0.081	0.082	0.010	0.010
	95th percentile	0.310	0.313	0.149	0.151
Olive oil	Percentage of consumers (%)	68.4		80.9	
	Median	0.027	0.028	0.008	0.008
	95th percentile	0.352	0.358	0.056	0.057
Rapeseed oil	Percentage of consumers (%)	92.9		13.9	
	Median	0.016	0.016	0.004	0.004
	95th percentile	0.085	0.087	0.020	0.021
Sunflower oil	Percentage of consumers (%)	39.2		98.3	
	Median	0.021	0.021	0.014	0.014
	95th percentile	0.117	0.118	0.055	0.055
Other oils	Percentage of consumers (%)	34.3		81.0	
	Median	0.089	0.089	0.010	0.010
	95th percentile	0.723	0.725	0.067	0.068

- *Table 4a continued* -

		Children (N=732)[1]		Adults (N=15,371)[2]	
		lb	ub	lb	ub
Fried herring products	Percentage of consumers (%)	no consumers		1.3	
	Median			0.017	0.018
	95th percentile			0.059	0.063
Tinned fish	Percentage of consumers (%)	2.9		34.2	
	Median	0.009	0.013	0.004	0.006
	95th percentile	0.087	0.119	0.021	0.029
Breaded pre-fried frozen fish products	Percentage of consumers (%)	17.6		15.5	
	Median	0.182	0.191	0.021	0.022
	95th percentile	0.480	0.503	0.079	0.083
Smoked fish	Percentage of consumers (%)	9.8		18.7	
	Median	0.007	0.010	0.004	0.005
	95th percentile	0.020	0.031	0.017	0.027
Liquid condiments	Percentage of consumers (%)	14.3		18.2	
	Median	0.0001	0.0001	0.00009	0.00011
	95th percentile	0.0010	0.0013	0.00032	0.00040
Soy sauce	Percentage of consumers (%)	4.2		9.1	
	Median	0.0004	0.0005	0.0001	0.0002
	95th percentile	0.0028	0.0037	0.001	0.001

lb: lower bound, ub: upper bound
[1] children who are no longer breastfed (0.5 to under 5 years), [2] adolescents and adults (14-80 years)

Table 4b: Median intake (monthly average) of the sum of free and bound 2-MCPD from 17 food groups in µg/kg BW and day (basis: only consumers).

		Children (N=732)[1]		Adults (N=15,371)[2]	
		lb	ub	lb	ub
Bread and bread rolls	Percentage of consumers (%)	94.1		98.4	
	Median	0	0.068	0	0.054
	95th percentile	0	0.169	0	0.134
Croissants and filled pastries for children	Percentage of consumers (%)	14.1		12.5	
	Median	0.028	0.034	0.007	0.009
	95th percentile	0.070	0.085	0.039	0.047
Donuts and Berliners	Percentage of consumers (%)	7.7		2.9	
	Median	0.143	0.146	0.016	0.016
	95th percentile	0.529	0.540	0.105	0.108
Savoury sandwich spreads	Percentage of consumers (%)	7.8		4.3	
	Median	0.016	0.017	0.010	0.011
	95th percentile	0.067	0.070	0.063	0.066
Sweet sandwich spreads	Percentage of consumers (%)	42.2		14.8	
	Median	0.019	0.020	0.011	0.012
	95th percentile	0.077	0.082	0.069	0.074
French fries, outside the home	Percentage of consumers (%)	23.8		40.0	
	Median	0.063	0.066	0.007	0.007
	95th percentile	0.185	0.194	0.037	0.039

- Table 4b continued -

		Children (N=732)[1]		Adults (N=15,371)[2]	
		lb	ub	lb	ub
Infant formula	Percentage of consumers (%)	17.1		no consumers	
	Median	0.162	0.180		
	95th percentile	0.435	0.484		
Frying fats	Percentage of consumers (%)	67.2		94.4	
	Median	0.038	0.038	0.021	0.021
	95th percentile	0.204	0.206	0.076	0.076
Margarines and vegetable fats	Percentage of consumers (%)	97.1		96.8	
	Median	0.038	0.039	0.005	0.005
	95th percentile	0.146	0.150	0.070	0.072
Olive oil	Percentage of consumers (%)	68.4		80.9	
	Median	0.012	0.012	0.003	0.004
	95th percentile	0.153	0.158	0.024	0.025
Rapeseed oil	Percentage of consumers (%)	92.9		13.9	
	Median	0.006	0.007	0.002	0.002
	95th percentile	0.034	0.037	0.008	0.009
Sunflower oil	Percentage of consumers (%)	39.2		98.3	
	Median	0.010	0.010	0.007	0.007
	95th percentile	0.055	0.056	0.026	0.026
Other oils	Percentage of consumers (%)	34.3		81.0	
	Median	0.040	0.040	0.005	0.005
	95th percentile	0.326	0.329	0.030	0.031
Fried herring products	Percentage of consumers (%)	no consumers		1.3	
	Median			0.006	0.008
	95th percentile			0.023	0.029
Tinned fish	Percentage of consumers (%)	2.9		34.2	
	Median	0.002	0.006	0.001	0.003
	95th percentile	0.020	0.059	0.005	0.015
Breaded and pre-fried frozen fish products	Percentage of consumers (%)	17.6		15.5	
	Median	0.074	0.084	0.008	0.010
	95th percentile	0.195	0.222	0.032	0.037
Smoked fish	Percentage of consumers (%)	9.8		18.7	
	Median	0.0002	0.004	0.00010	0.002
	95th percentile	0.001	0.013	0.00047	0.011

lb: lower bound, ub: upper bound
[1] children who are no longer breastfed (0.5 to under 5 years), [2] adolescents and adults (14-80 years)

Table 4c: Median intake (monthly average) of bound glycidol for 17 food groups in µg/kg BW and day (basis: only consumers).

		Children (N=732)[1]		Adults (N=15,371)[2]	
		lb	ub	lb	ub
Bread and bread rolls	Percentage of consumers (%)	94.1		98.4	
	Median	0	0.034	0	0.027
	95th percentile	0	0.085	0	0.067
Croissants and filled pastries for children	Percentage of consumers (%)	14.1		12.5	
	Median	0.058	0.061	0.015	0.016
	95th percentile	0.147	0.155	0.082	0.086
Donuts and Berliners	Percentage of consumers (%)	7.7		2.9	
	Median	0.232	0.232	0.026	0.026
	95th percentile	0.858	0.858	0.171	0.171
Savoury sandwich spreads	Percentage of consumers (%)	7.8		4.3	
	Median	0.011	0.011	0.007	0.007
	95th percentile	0.046	0.047	0.043	0.044
Sweet sandwich spreads	Percentage of consumers (%)	42.2		14.8	
	Median	0.020	0.020	0.012	0.012
	95th percentile	0.083	0.083	0.075	0.075
French fries, outside the home	Percentage of consumers (%)	23.8		40.0	
	Median	0.097	0.098	0.010	0.010
	95th percentile	0.283	0.286	0.057	0.057
Infant formula	Percentage of consumers (%)	17.1		no consumers	
	Median	0.082	0.098		
	percentile 96	0.221	0.263		
Frying fats	Percentage of consumers (%)	67.2		94.4	
	Median	0.074	0.074	0.041	0.041
	95th percentile	0.400	0.400	0.148	0.148
Margarines and vegetable fats	Percentage of consumers (%)	97.1		96.8	
	Median	0.045	0.045	0.005	0.005
	95th percentile	0.174	0.174	0.084	0.084
Olive oil	Percentage of consumers (%)	68.4		80.9	
	Median	0.030	0.030	0.008	0.008
	95th percentile	0.382	0.382	0.061	0.061
Rapeseed oil	Percentage of consumers (%)	92.9		13.9	
	Median	0.016	0.016	0.004	0.004
	95th percentile	0.087	0.087	0.021	0.021
Sunflower oil	Percentage of consumers (%)	39.2		98.3	
	Median	0.023	0.023	0.015	0.015
	95th percentile	0.128	0.128	0.060	0.060
Other oils	Percentage of consumers (%)	34.3		81.0	
	Median	0.037	0.037	0.004	0.004
	95th percentile	0.304	0.304	0.028	0.028

- Table 4c continued -

		Children (N=732)[1]		Adults (N=15,371)[2]	
		lb	ub	lb	ub
Fried herring products	Percentage of consumers (%)	no consumers		1.3	
	Median			0.004	0.005
	95th percentile			0.013	0.016
Tinned fish	Percentage of consumers (%)	2.9		34.2	
	Median	0.006	0.008	0.003	0.003
	95th percentile	0.059	0.075	0.015	0.018
Breaded pre-fried frozen fish products	Percentage of consumers (%)	17.6		15.5	
	Median	0.006	0.018	0.001	0.002
	95th percentile	0.017	0.048	0.003	0.008
Smoked fish	Percentage of consumers (%)	9.8		18.7	
	Median	0.003	0.005	0.001	0.003
	95th percentile	0.007	0.015	0.007	0.013

lb: lower bound, ub: upper bound
[1] children who are no longer breastfed (0.5 to under 5 years), [2] adolescents and adults (14-80 years)

3.1.3.2.2 Total intake of 3-MCPD, 2-MCPD (respective sums of free and bound forms) and bound glycidol from the food groups considered, based on all respondents

For estimating the total intake, based on all respondents, the intake levels for the individual food groups were summed up, and the median and 95th percentile of the resulting intake was subsequently considered, on an individual level for all test subjects (Table 5).

Table 5: Median total intake (monthly average) of 3-MCPD, 2-MCPD (respective sums of free and bound forms) and bound glycidol from 19 (3-MCPD) and 17 (2-MCPD, glycidol) food groups in µg/kg BW and day (basis, all respondents).

		Children (N=732)[1]	Adults (N=15,371)[2]
		lb - ub	lb - ub
∑ Free 3-MCPD and bound 3-MCPD	Median	0.4 - 0.5	0.1 - 0.2
	95th percentile	1.6	0.4 - 0.5
∑ Free 2-MCPD and bound 2-MCPD	Median	0.2 - 0.3	0.1
	95th percentile	0.7 - 0.8	0.2 - 0.3
		lb - ub	lb - ub
Bound glycidol	Median	0.3	0.1
	95th percentile	0.9	0.3

lb: lower bound, ub: upper bound
[1] children who are no longer breastfed (0.5 to under 5 years), [2] adolescents and adults (14-80 years)

3.1.3.2.3 Total intake of 3-MCPD, 2-MCPD (respective sums of free and bound forms) and bound glycidol from the food groups considered, on the basis of consumers

If only consumers of different food groups are considered, it becomes clear that the percentages of respondents as a whole vary heavily. If the total intake was depicted in a way that portrayed consumers as people who have consumed at least one product from the selected food groups, several non-consumers would distort the total intake level.

Therefore, the consumers of the food groups with the highest intake levels were selected in three scenarios, as a population based on the ub approach, and the total intake was estimated for these. These three scenarios are:

- For children (Table 6a*)*:

  To estimate the total intake (monthly average) of 3-MCPD and 2-MCPD, the consumers of ‘infant formula’, ‘donuts/Berliners’ and ‘breaded pre-fried frozen fish products’ were considered.

  To estimate the total intake (monthly average) of glycidol, the consumers of ‘infant formula’, ‘donuts/Berliners’ and ‘French fries, outside the home’ were considered.

- For adults (Table 6b):

  To estimate the total intake (monthly average) of 3-MCPD and glycidol, the consumers of ‘frying fats’, ‘sweet sandwich spreads’ and ‘croissants and filled pastries for children’ were considered.

  To estimate the total intake (monthly average) of 2-MCPD, the consumers of ‘frying fats’, ‘sweet sandwich spreads’ and ‘breaded pre-fried frozen fish products’ were considered.

Other selection criteria were a minimum percentage for consumers of 5% and slight uncertainties with the exposure assessment (see Section 3.2.3 ‘Assessment of the quality of data to be used for the exposure assessment’).

Table 6a: Median total intake (monthly average) of 3-MCPD, 2-MCPD (respective sums of free and bound forms) and bound glycidol from 19 (3-MCPD) and 17 (2-MCPD, glycidol) food groups for children in µg/kg BW and day (basis: only consumers from one group).

Basis: Consumers of		total intake (ub) in µg/kg BW and day for children (N=732)[1]		
		∑ Free + bound 3-MCPD	∑ Free + bound 2-MCPD	Bound glycidol
Infant formula	Consumers (%)	17	17	17
	Median	1.2	0.6	0.6
	95th percentile	2.6	1.2	1.3
Donuts/Berliners	Consumers (%)	8	8	8
	Median	0.7	0.4	0.6
	95th percentile	1.7	1.0	1.4
Breaded pre-fried frozen fish products	Consumers (%)	18	18	–
	Median	0.7	0.4	
	95th percentile	1.5	0.7	
French fries, outside the home	Consumers (%)	–	–	24
	Median			0.4
	95th percentile			0.9

ub: *upper bound*
[1] children who are no longer breastfed (0.5 to under 5 years)

Table 6b: Monthly total intake (monthly average) of 3-MCPD, 2-MCPD (respective sums of free and bound forms) and bound glycidol from 19 (3-MCPD) and 17 (2-MCPD, glycidol) food groups for adults in µg/kg BW and day (basis: only consumers from one group).

Basis: Consumers of		total intake (ub) in µg/kg BW and day for adults (N=15,731)[1]		
		∑ Free + bound 3-MCPD	∑ Free + bound 2-MCPD	Bound glycidol
Frying fats	Consumers (%)	94	94	94
	Median	0.2	0.1	0.1
	95th percentile	0.5	0.3	0.3
Sweet sandwich spreads	Consumers (%)	15	15	15
	Median	0.3	0.2	0.2
	95th percentile	0.6	0.3	0, 4
Croissants and filled pastries for children	Consumers (%)	13	–	13
	Median	0.2		0.2
	95th percentile	0.5		0.4
Breaded pre-fried frozen fish products	Consumers (%)	–	16	–
	Median		0.2	
	95th percentile		0.3	

ub: *upper bound*
[1] adolescents and adults (14-80 years)

3.1.3.2.4. Total intake of 3-MCPD, 2-MCPD (respective sums of free and bound forms) and bound glycidol from the food groups considered, based on brand loyal consumers of frying fats with high concentrations (adults)

In another scenario, the total intake of 3-MCPD, 2-MCPD (respective sums of free and bound forms) and bound glycidol from 19 (3-MCPD) and 17 (2-MCPD, glycidol) food groups for adults was estimated in a way that the 95th percentile of concentrations was used for ‘frying fats’ as a basic food group, in order to represent consumers who consume frying fats with high concentrations over a long period (Table 7).

Table 7: Median total intake (monthly average) of 3-MCPD, 2-MCPD (respective sums of free and bound forms) and bound glycidol from 19 (3-MCPD) and 17 (2-MCPD, glycidol) food groups for adults in µg/kg BW and day (basis: only consumers of frying fats with high concentrations).

		Total intake (ub) for adults (N=15,371)[1]		
Basis: Consumers of frying fats with high concentrations		∑ Free + bound 3-MCPD	∑ Free + bound 2-MCPD	Bound glycidol
	Consumers (%)	94	94	94
	Median	0.3	0.2	0.3
	95th percentile	0.7	0.4	0.7

ub: *upper bound*
[1] adolescents and adults (14-80 years)

3.1.3.2.5 Total intake of 3-MCPD, 2-MCPD (respective sums of free and bound forms) and bound glycidol, based on non-breastfed infants who only consume infant formula

Another scenario describes the exposure of non-breastfed infants aged 1-4 months who are exclusively fed with 'infant formula' (Table 8). As there is no available consumption data for this population group in Germany, the median drinking quantity recommended for infants in the first four months of life was used for the chronic exposure assessment. This is equivalent to 170 ml and 22.1 g infant formula (powder)/day (EFSA 2017a). The median values for analytes in infant formula (powder) form the basis for occurrence data, as does the 95th percentile for brand loyal consumers of products with high concentrations.

Table 8: Median total intake of 3-MCPD, 2-MCPD (respective sums of free and bound forms) and bound glycidol from infant formula for non-breastfed infants who only consume infant formula, in µg/kg BW and day.

	Total intake in µg/kg BW and day non-breastfed infants (1 - 4 months) who only consume infant formula[1]		
	∑ Free + bound 3-MCPD	∑ Free + bound 2-MCPD	Bound glycidol
Mean ub	3.1	1.3	0.7
95th percentile ub	6.4	2.5	3.6

ub: *upper bound*
[1] basis: median drinking quantity for infants in their first four months of life (EFSA 2017)

3.1.3.2.6 Other possible exposure sources

The occurrence data for 'frozen French fries' and 'breaded non-pre-fried frozen fish products' was not included in the exposure assessment, as the significantly higher occurrence data which was also available for 'French fries outside the home' and 'breaded pre-fried frozen fish products' was of greater relevance to actual exposure, as they are more in keeping with ready-to-eat prepared products. Nevertheless, there may be an underestimation of exposure with regards to 'breaded pre-fried frozen fish products', as the occurrence data was for the

products in an unprepared condition, and concentrations may rise even further during thermal processing. Moreover, the current (relatively high) occurrence data from the BVL is an indication that other fried foods may represent another exposure source as well.

Occurrence data for ‘Asian dishes with instant noodles’ was not included in the exposure assessment as there is no available consumption data for these. An example of exposure to 3-MCPD and related compounds can be presented in a scenario of the consumption of ‘Asian dishes with instant noodles’. An adult weighing 70 kg would consume, on average, an additional 0.036 - 0.037 µg 3-MCPD and 0.027 - 0.028 µg 2-MCPD (respective sums of free and bound forms lb - ub) and 0.046 µg bound glycidol (lb = ub) per kg/BW daily, with a portion size of 60 g dry product and an assumed consumption of one portion per week.

The ‘biscuits’, ‘potato snack products’ and ‘smoked sausage and meat goods’ food groups were not included in the exposure assessment as free and bound 3-MCPD and 2-MCPD amounts could not be summed up due to an insufficient data situation. They also represent sources of exposure to these compounds (see Annex II). Compared to the foods considered in the exposure assessment, ‘biscuits’ and ‘potato snack products’ had average concentrations of the compounds analysed, while concentrations in ‘smoked sausage and meat goods’ were low.

Moreover, foods other than the foods considered which contain refined vegetable fats, and other heated foods such as roast meat or toasted bread, could represent sources of 3-MCPD and related compounds.

3.1.4 Risk characterisation

The following population groups were considered in the risk characterisation: children, infants and adults. For most consumption scenarios, the average concentrations of 3-MCPD, 2-MCPD and their fatty acid esters, and bound glycidol were linked with consumption quantities for the relevant population groups, and the median (normal consumers) and 95th percentile (high consumers) of the resulting intakes were considered for each food group. One exception is the assessment for the ‘frying fats’ food group for adults, where ‘frying fats with high concentrations of 3-MCPD, 2-MCPD, their fatty acid esters and bound glycidol’ were also considered. Both average and high concentrations of 3-MCPD, 2-MCPD, their fatty acid esters and bound glycidol were also considered for infants and the consumption of ‘infant formula’.

3.1.4.1 3-MCPD and its fatty acid esters

The BfR uses the TDI of 2 µg/kg BW and day derived by EFSA as a basis for the risk assessment of 3-MCPD and its fatty acid esters in foods (BfR 2007; EFSA 2018). The occurrence of renal tubular hyperplasia observed in male rats in long-term studies was considered as the most sensitive endpoint for deriving this TDI. The BfR also considers this endpoint to be the most sensitive effect, as the effects for the other endpoints identified (e.g. effects which are toxic for reproduction) occur at higher doses, as described in the current EFSA opinion from 2018 (EFSA 2018).

For children and infants (consumers aged 0.5 to under 5 years) the ‘infant formula’, ‘donuts/Berliners’, ‘breaded pre-fried frozen fish products’ and ‘margarines/vegetable fats’ food groups are especially associated with a high intake of 3-MCPD and its fatty acid esters (see Table 6a).

For children, the exhaustion of the group TDI for 3-MCPD (ub) was 34 - 61% on median (see Table 9), based on the consumption of 'infant formula', 'donuts/Berliners' and 'breaded pre-fried frozen fish products'. In the 95th percentile, the exhaustion rates were 75 and 85%, based on the respective consumption of 'breaded pre-fried frozen fish products' and 'donuts/Berliners'. A consideration of the intake of 3-MCPD and its fatty acids, only based on consumers of the 'infant formula' food group (for average concentrations across all 19 food groups), reveals an exhaustion of the TDI of 132% in this consumption scenario (see Table 9).

Table 9: Exhaustion of the TDI for 3-MCPD, based on the average total intake (monthly average) of free and bound 3-MCPD from 19 food groups for children[1] in µg/kg BW and day, as depicted in Table 6a (basis: only consumers from one group).

Basis: Consumers of		Exhaustion of the TDI (%)[2]
Infant formula	Consumers (17%)	
	Median	61
	95th percentile	**132**
Donuts/Berliners	Consumers (8%)	
	Median	37
	95th percentile	85
Breaded pre-fried frozen fish products	Consumers (18%)	
	Median	34
	95th percentile	75

ub: *upper bound*
[1] children who are no longer breastfed (0.5 to under 5 years; N=732)
[2] TDI = 2 µg/kg BW and day

For non-breastfed infants who exclusively consume 'infant formula', the exhaustion of the TDI for 3-MCPD was revealed to be 157% for consumers of 'infant formula' with average concentrations, and 318% for brand loyal consumers of products with high concentrations (see Table 10).

Table 10: Exhaustion of the TDI for 3-MCPD, based on the average total intake (monthly average) of free and bound 3-MCPD from 19 food groups for non-breastfed infants[1], who only consume infant formula, in µg/kg BW and day, as depicted in Table 8.

	Exhaustion of the TDI (%)[2]
Mean ub	**157**
95th percentile ub	**318**

ub: *upper bound*
[1] basis: median drinking quantity for infants in the first four months of life (EFSA 2017)
[2] TDI = 2 µg/kg BW and day

Among the high consumers of ‘infant formula’, a moderate exceedance of the TDI of 2 µg/kg BW and day by the factor 1.3 was also observed for children who only proportionally consume ‘infant formula’. TDI values do not apply to infants in the first month of life, as already determined in the BfR opinion from 2007 (BfR 2007). However, if the TDI concept is applied to infants, it is evident that the TDI (1.5-fold and 3-fold for the consumption of ‘infant formula’ with average and high concentrations respectively) is exceeded, especially for infants who only consume ‘infant formula’.

In adults, ‘frying fats’, ‘croissants/filled pastries for children’ and ‘donuts/Berliners’ clearly contribute to exposure to 3-MCPD and its fatty acid esters (see Table 6b). Based on the consumers of one food group (for average and high concentrations), the intake of 3-MCPD and its fatty acid esters across all 19 food groups considered (see Table 6b) does not lead to an exceedance of the TDI of 2 µg/kg BW and day in any of the consumption scenarios considered (median, 95th percentile) (see Table 11).

Table 11: Exhaustion of the TDI for 3-MCPD, based on the average total intake (monthly average) of free and bound 3-MCPD from 19 food groups for adults[1] in µg/kg BW and day, as depicted in Table 6a (basis: only consumers from one group).

Basis: Consumers of		Exhaustion of the TDI (%)[2]
Frying fats	Consumers (94%)	
	Median	10
	95th percentile	24
Sweet sandwich spreads	Consumers (15%)	
	Median	13
	95th percentile	30
Croissants and filled pastries for children	Consumers (13%)	
	Median	12
	95th percentile	26

ub: *upper bound*
[1] adolescents and adults (14-80 years; N=15,731)
[2] TDI = 2 µg/kg BW and day

The separate consideration of the consumption of ‘frying fats’ with high concentrations of free and bound 3-MCPD also did not result in an exceedance of the TDI of 2 µg/kg BW and day for adults in any consumption scenario (see Table 12). For free and bound 3-MCPD, a TDI exhasution of 14% (median) and 33% (95th percentile) was revealed for brand loyal adult consumers of ‘frying fats’ (see Table 12).

Table 12: Exhaustion of the TDI for 3-MCPD, based on the average total intake (monthly average) of free and bound 3-MCPD from 19 food groups for adults[1] in µg/kg BW and day, as depicted in Table 7 (basis: only consumers of frying fats with high concentrations).

Basis: consumers of frying fats with high concentrations		Exhasution of TDI (%)[2]
	Consumers (94%)	
	Median	14
	95th percentile	33

ub: *upper bound*
[1] adolescents and adults (14-80 years; N=15,371)
[2] TDI = 2 µg/kg BW and day

3.1.4.2 2-MCPD and its fatty acid esters

For children (including infants), 'infant formula', 'donuts/Berliners', 'breaded pre-fried frozen fish products' and 'margarines/vegetable fats' make a particularly significant contribution to total exposure to 2-MCPD and its fatty acid esters. For adults, 'frying fats', 'breaded pre-fried frozen fish products' and 'donuts/Berliners' contribute the most to the total intake of 2-MCPD and its fatty acid esters. The total intake (of average and high concentrations) of free and bound 2-MCPD with regard to body weight is higher for children (including infants) than for adults (Tables 4b, 6a, 6b, 7 and 8).

The available toxicological data regarding these substances is very limited, meaning that an assessment of possible health risks from exposure to 2-MCPD and its fatty acid esters is currently not possible.

3.1.4.3 Glycidol and its fatty acid esters

No confident lower threshold can be determined for the genotoxic and carcinogenic effect of glycidol, and therefore, no health-based guidance value has been derived. The internationally established MOE approach has therefore been used for the risk characterisation of bound glycidol. The MOE is the ratio of a suitable reference point and the human exposure to the substance. In this particular case, the T25 (the chronic dose at which cancer occurs within a certain tissue in 25% of laboratory animals during their lifespan) of 10,200 µg/kg BW and day derived from the data of an NTP study was used as a reference point to calculate the MOE for the intake of glycidol, together with the exposure assessment data. A MOE of 25,000 or more (if it is based on a T25 as a reference point) is generally considered being of low concern from a public health point of view, and therefore, represents a low priority for risk management measures (EFSA 2005).

Neither the reference point derived from animal studies nor the MOE values are health-based guidance values. The MOE is rather used for prioritising risk management measures. The conclusion that a total intake quantity with a MOE of 25,000 or more is 'of low concern' with regards to possible cancer risks should not be equated with 'no concern' from a toxicological perspective, as health risks cannot be ruled out with enough certainty even for intake quantities in this area. Therefore, the recommendation of keeping exposure to substances with a genotoxic and carcinogenic effect as low as is reasonably achievable generally applies (ALARA principle: as low as reasonably achievable).

For children (0.5 to under 5 years), ‘donuts/Berliners’, ‘French fries outside the home’, ‘infant formula’ and ‘margarines/vegetable fats’ particularly contributed to the total exposure to bound glycidol (see Table 6a).

For children, the assumption of an average total intake of bound glycidol, based on consumers of a food group with average concentrations for the ‘infant formula’, donuts/Berliners’ and ‘French fries outside the home’ food groups across all 17 food groups, has already resulted in MOE values clearly lower than 25,000 (see Table 13).

The calculated MOE values are 15,952 (median) and 7,693 (95th percentile) based on the consumers of ‘infant formula’, 18,489 (median) and 7,312 (95th percentile) for ‘donuts/Berliners’, and 24,130 for normal consumers and 10,833 for high consumers in the case of ‘French fries outside the home’ (see Table 13).

Table 13: MOE for glycidol, based on the average total intake (monthly average) of bound glycidol from 17 food groups for children[1] in µg/kg BW and day, as depicted in Table 6a (basis: only consumers from one group).

Basis: Consumers of		MOE[2]
Infant formula	Consumers (17%)	
	Median	**15,952**
	95th percentile	**7,693**
Donuts/Berliners	Consumers (8%)	
	Median	**18,489**
	95th percentile	**7,312**
French fries, outside the home	Consumers (24%)	
	Median	**24,130**
	95th percentile	**10,833**

ub: *upper bound*
[1] children who are no longer breastfed (0.5 to under 5 years; N=732)
[2] T25 = 10,200 µg glycidol/kg BW and day

As shown in Table 14, MOE values of 14,386 (‘infant formula’, with median concentrations) and 2,819 (‘infant formula’ with high concentrations) are given for non-breastfed infants exclusively consuming infant formula. Here, it should be emphasised that a very low MOE was observed for bound glycidol, especially in a consumption scenario of ‘infant formula’ with high concentrations of bound glycidol.

Table 14: MOE for glycidol based on the median total intake (monthly average) of bound glycidol, as depicted in Table 8, from 17 food groups for non-breastfed babies[1], who only consume infant formula in µg/kg BW and day.

	MOE[2]
Mean ub	**14,386**
95th percentile ub	**2,819**

ub: *upper bound*
[1] basis: median drinking quantity for infants in the first four months of life (EFSA 2017)
[2] T25 = 10,200 µg glycidol/kg BW and day

For adults (consumers), 'frying fats', 'croissants/filled pastries for children' and 'donuts/Berliners' are particularly associated with a high intake of bound glycidol (see Table 6b). The intake of bound glycidol, based solely on the consumers of a food group with median concentrations from all 17 food groups considered, does not result in MOE values lower than 25,000 in adults in any of the consumption scenarios considered (median, 95th percentile) (see Table 15).

Table 15: MOE for glycidol based on the median total intake (monthly average) of bound glycidol, as depicted in Table 6b from 17 food groups for adults[1] in µg/kg BW and day (basis: only consumers from one group).

Basis: Consumers of		MOE[2]
Frying fats	Consumers (94%)	
	Median	71,220
	95th percentile	30,252
Sweet sandwich spreads	Consumers (15%)	
	Median	57,485
	95th percentile	25,920
Croissants and filled pastries for children	Consumers (13%)	
	Median	60,389
	95th percentile	27,194

ub: *upper bound*
[1] adolescents and adults (14-80 years; N=15,371)
[2] T25 = 10,200 µg glycidol/kg BW and day

Based on consumers of 'frying fats with high concentrations', the review provided an intake quantity of bound glycidol, which resulted in a MOE of 40,208 for normal consumers, taking all 17 food groups considered into account. However, the consideration for high consumers (95th percentile) led to a MOE lower than 25,000 (see Table 16). In this scenario, a total intake of 0.7 µg bound glycidol/kg BW and day was calculated (see Table 7), which is equivalent to a MOE of 15,313 (95th percentile) (see Table 16).

Table 16: MOE for glycidol based on the median total intake (monthly average) of bound glycidol, as depicted in Table 7 from 17 food groups for adults[1] in µg/kg BW and day (basis: only consumers of frying fats with high concentrations).

Basis: consumers of frying fats with high concentrations		MOE[2]
	Consumers (94%)	
	Median	40,208
	95th percentile	**15,131**

ub: *upper bound*
[1] adolescents and adults (14-80 years; N=15,371)
[2] T25 = 10,200 µg glycidol/kg BW and day

3.2 Other aspects

3.2.1 Assessment of the quality of toxicological data

In principle, there are several uncertainties regarding toxicological data. In this context, the extent of the differences regarding resorption and/or hydrolysis rates of 2-MCPD and 3-MCPD fatty acid esters and bound glycidol, and 2-MCPD and 3-MCPD monoesters and diesters, is relatively unclear. Furthermore, the extent of metabolic transformation of both substances, glycidol and 3-MCPD, into each other cannot be estimated.

Relevant uncertainties for a conclusive risk assessment of 3-MCPD and its fatty acid esters have particularly been revealed by the lack of data on developmental toxicity and the effects of these substances on the development of the nervous system in children and adolescents.

Moreover, the long-term studies available so far only contain insufficient investigations on reproductive toxicity and fertility in male laboratory animals.

For 2-MCPD and its fatty acid esters, there are only very few data available regarding toxicity. Therefore, a risk characterisation of these substances is currently not possible.

There are also uncertainties with regard to data on the carcinogenic effect of glycidol observed in rats. The available data do not allow for any BMD modelling, meaning that a $BMDL_{10}$ cannot be derived. Instead, the T25 has to be used. Further, the laboratory animals in the study were reated with different glycidol doses only for 5 out of 7 days. To mathematically compensate for this, the derived T25 of 14,200 µg glycidol/kg BW and day was multiplied by a factor of 5/7. However, this has resulted in uncertainty as to whether this calculation correctly reflects the actual toxicokinetics after oral administration of glycidol over 7 days per week.

3.2.2 Assessment of the quality of data from an analytical chemistry perspective

It is hard to keep track of the number of analytical methods for determining MCPD, its derivatives and related compounds, and for glycidol and its esters. While the analytics of bound

MCPD and glycidol in plant-based oils and fats was considered as established and validated at the time the project was carried out, this is not the case for analytics in composite foods.

In order to bypass the known limitations of methods applied for determining free 2-MCPD and 3-MCPD parallel to MCPD esters and bound glycidol in composite foods as much as possible, a method was used and validated in-house as part of the EH project, and all relevant composite food groups could be analysed in the same manner using this method.

3.2.3 Assessment of the quality of data used for the exposure assessment

Despite the extensive occurrence data on which the present exposure assessment is based, a complete intake assessment of all foods containing 3-MCPD, 2-MCPD and glycidol is very difficult, due to their large prevalence as process contaminants. Concentrations in processed products varied particularly heavily, depending on the type of processing. These data gaps could be closed by the BfR MEAL study in future, which would allow for an improved assessment of the median exposure.

Uncertainties arise mainly from the following aspects:

- Due to insufficient data, different data sets were excluded, such as those for the 'biscuits', 'potato snack products' and 'smoked sausage and meat goods' food groups. This will presumably lead to an underestimation of total exposure.
- The 'bread/bread rolls' food group was included in the exposure assessment, due to its relevance to consumption, although occurrence data was only gathered for eleven samples and the measured levels for all analytes were below the limit of quantification. Applying the upper bound approach, concentrations for this food group were included with the value of the corresponding limit of quantification of 15 µg/kg, resulting in an overestimation of exposure for 'bread/bread rolls'.
- In particular, the concentrations of free and bound 3-MCPD and 2-MCPD, and of bound glycidol, could vary heavily in processed products, depending on the type of processing. This resulted in a considerable data gap, which also represents an uncertainty with regard to the exposure assessment performed, and has a tendency to lead to an underestimation of exposure.
- There was no occurrence data for free glycidol in most of the food groups considered. There was only occurrence data for free glycidol for a small portion of BVL samples of 'biscuits' and 'potato snack products', the capacity of which could not be estimated. However, these samples were excluded from the exposure assessment, due to the criteria catalogue. The exposure assessment for glycidol is therefore exclusively based on occurrence data for bound glycidol. It is assumed that free glycidol is not present in foods, due to its reactivity. However, this approach could lead to underestimation of total exposure.
- For various food groups, data were sometimes only available for a small proportion of consumers (see Table 4a-c), which is associated with a sampling uncertainty for estimating the 95th percentile.
- Further uncertainties to the exposure assessment performed arise, as the present work only included occurrence data for refined oils. By contrast, consumption data did not distinguish between refined and cold-pressed oils. A fairly large amount of cold-pressed forms of oil is consumed in Germany. As refined oils have higher concentrations of analytes compared to cold-pressed oils, the use of occurrence data for refined

oils leads to an overestimation of exposure to 3-MCPD, 2-MCPD (respective sums of free and bound forms) and bound glycidol from the consumption of vegetable oils. On the other hand, the exposure assessment for fats and oils is based on occurrence data for non-heated products. As a result of the use of 'frying fats', 'oils' and 'margarines/vegetable fats' in heating processes, the concentrations of ready-to-eat products may be higher, which leads to an underestimation of exposure.

- Finally, uncertainties have also arisen with regard to the exposure assessmentas both consumption studies (NVS II, VELS) were performed more than ten or more years ago. It is probable that consumption habits have changed over this period. Moreover, in the group of adults, the intake of foods that are consumed only sporadically and are not part of the daily diet may be underestimated when considering all respondents, due to the survey period of four weeks and the limited accuracy of the individual foods surveyed. 3.2.4 Comparison with the results of the EFSA exposure assessment for 2-MCPD, 3-MCPD (respective sums of free and bound forms) and bound glycidol in foods (EFSA 2016)

The EFSA exposure assessment is based on data on the occurrence of 2-MCPD and 3-MCPD and their fatty acid esters, as well as bound glycidol, in foods from a total of 23 European member states, which was gathered between 2009 and 2015. The comparison of the median total intake from the EFSA with the present BfR exposure assessment demonstrates that the EFSA has assessed exposure values which are almost twice as high in the median, while the 95th percentile is only marginally higher (Table 17).

Table 17: Comparison of the median total intake of 3-MCPD, 2-MCPD (respective sums of free and bound forms) and bound glycidol in the present study with the EFSA exposure assessment (EFSA 2016) in µg/kg BW and day (basis: all respondents).

		Children (lb - ub)		adults (lb - ub)	
		this work (0.5 - < 5 years)	EFSA (< 3 years)	this work (14-80 years)	EFSA (from 18 years)
∑ Free and bound 3-MCPD	Median	0.4 - 0.5	0.8 - 0.9	0.1 - 0.2	0.3 - 0.5
	95th percentile	1.6	1.6 - 1.9	0.4 - 0.5	0.6 - 0.9
∑ Free and bound 2-MCPD	Median	0.2 - 0.3	0.3 - 0.5	0.1	0.1 - 0.2
	95th percentile	0.7 - 0.8	0.6 - 1.0	0.2 - 0.3	0.2 - 0.5
Bound Glycidol	Median	0.3	0.6 - 0.7	0.1	0.2 - 0.3
	95th percentile	0.9	1.1 - 1.5	0.3	0.5 - 0.7

lb: lower bound, ub: upper bound

For children (only consumers) in both exposure assessments, 'infant formula' represents the food group with the highest contribution to the median total intake of the contaminants under consideration. While EFSA determined 'vegetable fats and oils', 'biscuits' (not considered in this work) and 'baked goods/cakes' as important sources, in the present exposure assessment, 'donuts/Berliners', 'breaded pre-fried frozen fish products' and 'French fries outside the home' (which were not considered in the EFSA opinion) were identified as food groups with a relevant contribution to the median total intake (Table 6a).

For adults (consumers), ‘margarine’ and similar products, ‘baked goods/cakes’ and ‘vegetable fats and oils’ were determined by EFSA as food groups with the highest contribution to the median total intake of the contaminants considered. In the present exposure assessment, ‘frying fats’, ‘croissants/filled pastries for children’, ‘sweet sandwich spreads’ and ‘breaded pre-fried frozen fish products’ were identified as food groups with a relevant contribution to the median total intake (Table 6a).

The EFSA exposure assessment revealed lower values than the present exposure assessment for total exposure to 2-MCPD and 3-MCPD for infants fed exclusively with ‘infant formula’. By contrast, glycidol exposure was higher in the EFSA opinion for infants fed exclusively with ‘infant formula’ (Table 8). One cause of this difference are the lower concentrations of 3-MCPD and 2-MCPD, and higher concentrations of glycidol in infant formula in the EFSA opinion.

Other possible reasons for the differences observed may be methodical deviations in the type and grouping of the foods included in the exposure assessment, as well as the national influence. Amongst other things, roasted meat was relevant to the total intake of the combinations investigated in both population groups in the EFSA exposure assessment (EFSA 2016). This food group could not be included in the present exposure assessment due to a lack of data, meaning that exposure could be underestimated. As a further example, ‘biscuits’ and ‘potato snack products’ were not included in the present exposure assessment, due to insufficient data, while they were included in the EFSA exposure assessment. One example of the different grouping of foods is ‘vegetable fats and oils’. These were considered as a group by tEFSA, while ‘frying fats’ and individual plant oils were considered separately in the present work. In the EFSA’s work, palm oil/fat (with very high concentrations) was also included in this food group (EFSA 2016).

The findings of a recently performed exposure study, in which eleven healthy volunteers consumed palm fat rich in glycidyl esters for four weeks, while levels of the haemoglobin adduct *N*-(2.3-dihydroxypropyl)valine were measured as a biomarker for the internal glycidol exposure, indicated that the test subjects had a background exposure of approx. 0.94 µg/kg BW (Abraham *et al.* 2019). This value is considerably higher than the current assessment by EFSA for the median total intake of bound glycidol from food (adults: 0.2 - 0.3 µg/kg BW, children: 0,6 - 0,7 µg/kg KG), or the assessment of the concentrations in selected foods performed in the present assessment for the median total intake of bound glycidol (adults: 0.1 µg/kg BW, children: 0.3 µg/kg BW). However, there are preliminary indications from initial experiments that consumers who exclusively consume foods which have not been heated at temperatures over 42 °C (‘raw food eaters’) have a baseline blood concentration of the above mentioned haemoglobin adduct. This could be caused by an exposure to glycidol from other sources, an endogenous exposure or a previously unknown C3 compound.

3.3 Summary

3.3.1 3-MCPD and its fatty acid esters

For adult normal and high consumers, no exceedance of the TDI of 2 µg/kg BW was observed, on the basis of the exposure assessment performed for 3-MCPD and its fatty acid esters. An increased health risk is therefore not expected.

For children (high consumers of infant formula), a moderate exceedance (factor 1.3) of the TDI of 2 µg/kg BW and day was found. The exposure assessment for infants, especially infants exclusively fed with infant formula, revealed a clear exceedance of the TDI (factor 1.5 to 3, depending on the consumption scenario). Therefore, an increased health risk is possible, especially for children and infants consuming infant formula over a long period.

3.3.2 2-MCPD and its fatty acid esters

Exposure to 2-MCPD and its fatty acid esters from foods was assessed in this opinion. However, due to the limited availability of toxicological data regarding 2-MCPD and its fatty acid esters no conclusive assessment of its potential health risks was possible.

3.3.3 Glycidol and its fatty acid esters

For glycidol, exhibiting genotoxic and carcinogenic effects, it is not possible to dderive an intake level which can be considered safe, since no lower threshold ca be determined for genotoxic substances. The margin of exposure (MOE) approach has therefore been used for the prioritisation of risk management measures.

Most consumption scenarios for adults reveal intake quantities of bound glycidol which result in MOE values over 25,000. A MOE value of 25,000 or more (if it is based on a T25 as the reference point) is generally considered as being of low concern from a public health point of view (EFSA 2005). A scenario for high consumers of frying fats with high concentrations of bound glycidol clearly results in intake quantities which yield a MOE value of 15,131.

For children and infants, various consumption scenarios (normal and high consumers) result in MOE values of clearly less than 25,000. A MOE of 2,900 is therefore calculated for infants consuming only infant formula with high concentrations of glycidol. An increased health risk due to a chronic intake therefore appears to be possible for certain population groups.

3.4 Risk management options, recommended measures

The assessment has shown that exposure to 2-MPCD and 3-MCPD and their fatty acid esters, and to bound glycidol is especially high for children, in particular non-breastfed infants exclusively fed with infant formula. Therefore, the BfR recommends the following measures, based on the available data:

- All efforts should be made to further reduce the concentrations of 2-MCPD and 3-MCPD and their fatty acid esters, and the concentration of bound glycidol in infant formula and food groups which are heavily consumed by children (‘donuts/Berliners’, ‘margarines/vegetable fats’, ‘breaded pre-fried frozen fish products, ‘French fries consumed outside the home’). This is especially relevant for infant formula, as there is no alternative food source for non-breastfed infants.
- For substances with genotoxic and carcinogenic effects, the recommendation of minimising exposure as much as is reasonably achievable generally applies within the European Union (ALARA principle: as low as reasonably achievable), as even low intake quantities, especially for regular consumption, may be associated with an increase in health risks. The BfR therefore generally recommends keeping total exposure to glycidol from all foods as low as possible.
- This is of particular importance for infant formula, as there is currently no alternative food source for non-breastfed infants. Possible high exposure to bound glycidol via consumption of ‘infant formula’ only extends over a relatively short lifespan for infants.

By contrast, the comparison of risks in accordance with the MOE concept refers to lifelong exposure. However, as EFSA also noted in its current opinion, this increased exposure occurs at a particularly critical stage of development, when humans are particularly sensitive to genotoxic carcinogens such as glycidol (EFSA 2016). The BfR is of the view that, with regard to possible carcinogenic effects on children, particularly strict standards must be set for avoidable exposures to substances with carcinogenic effetcs.

- The BfR endorses the demand for research addressed in the current EFSA opinions on 2-MPCD, 3-MCPD and glycidol, and their fatty acid esters (EFSA 2016; EFSA 2018).

Further information on the subject of 3-MCPD, 2-MCPD and glycidol fatty acid esters on the BfR website

https://www.bfr.bund.de/en/a-z_index/monochloropropanediol__3_mcpd_-130048.html

https://www.bfr.bund.de/en/a-z_index/monochloropropanediol__3_mcpd_-130048.html

BfR "Opinions app"

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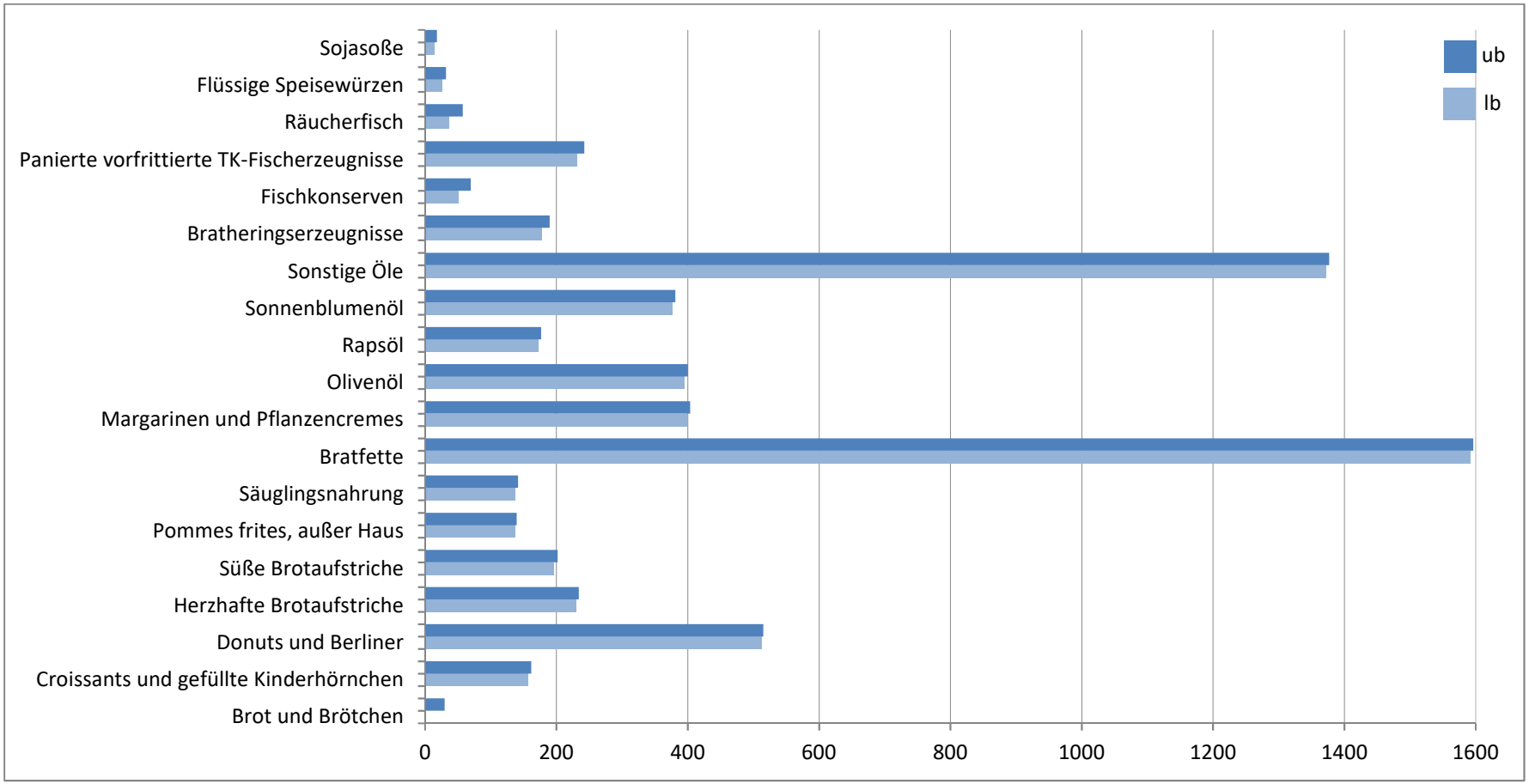
This text version is a translation of the original German text which is the only legally binding version.

Anhang Ia: Mittlere Gehalte an 3-MCPD (Summe freier und gebundener Form) in 19 Lebensmittelgruppen in µg/kg.



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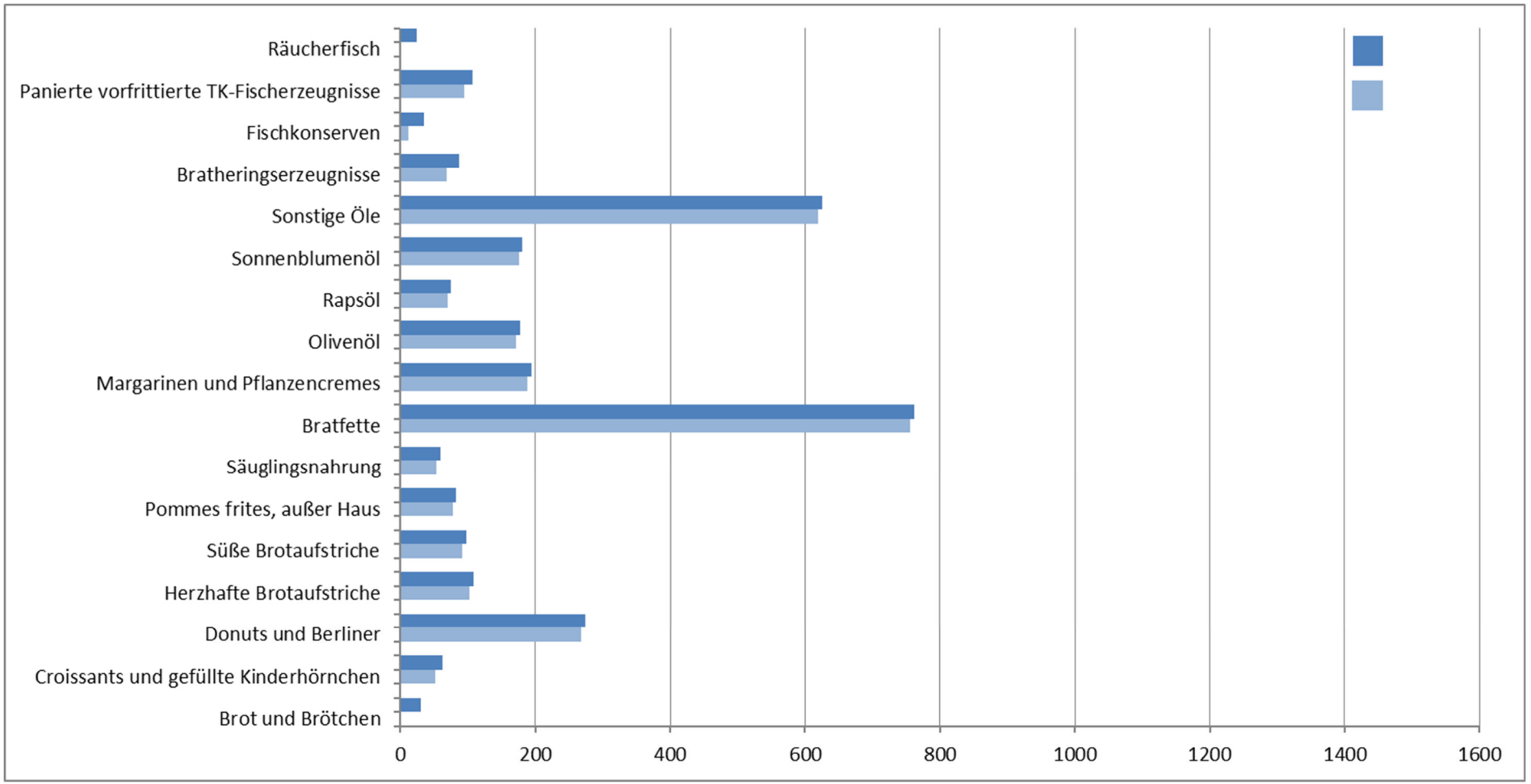
Anhang Ib: Mittlere Gehalte an 2-MCPD (Summe freier und gebundener Form) in 17 Lebensmittelgruppen in µg/kg.



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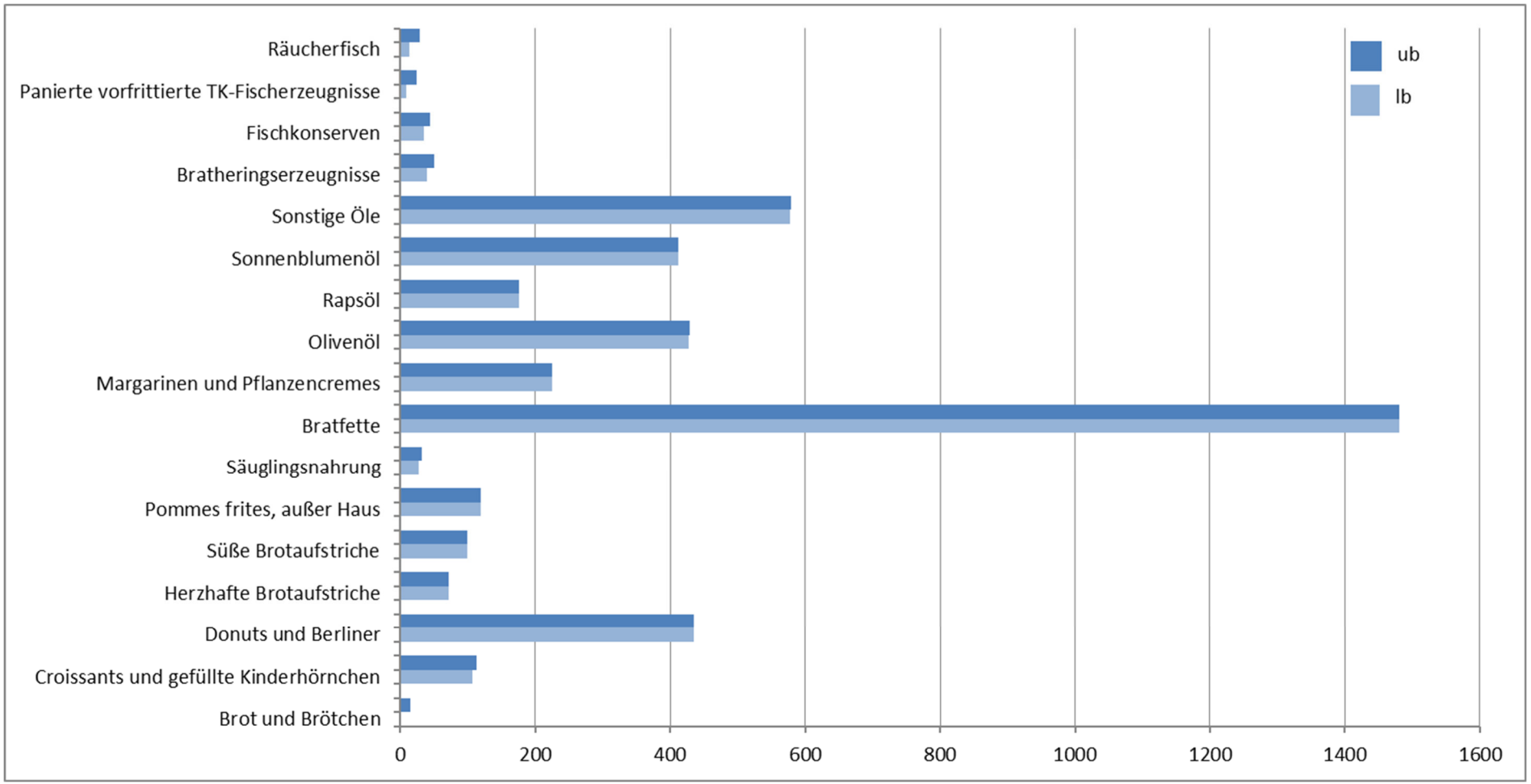
Anhang Ic: Mittlere Gehalte an gebundenem Glycidol in 17 Lebensmittelgruppen in µg/kg.



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Anhang II: Gehalte an 3-MCPD, 2-MCPD, Glycidol und ihren Fettsäureestern in von der Expositionsschätzung ausgeschlossenen Lebensmittelgruppen in µg/kg.

		Freies 3-MCPD	Gebundenes 3-MCPD	Freies 2-MCPD	Gebundenes 2-MCPD	Freies Glycidol	Gebundenes Glycidol
Pommes frites, tiefgekühlt[1]	N	33	33	33	33	-	33
	< LOQ (%)	100	12	100	52	-	33
	Mittelwert lb	0	16	0	5	-	10
	Mittelwert ub	5	17	5	11	-	13
	Median lb	0	16	0	0	-	12
	Median ub	5	16	5	10	-	12
	95. Perzentil lb	0	31	0	13	-	21
	95. Perzentil ub	5	31	5	13	-	21
Asia-Gerichte mit Instantnudeln[1]	N	50	50	50	50	-	50
	< LOQ (%)	44	18	98	20	-	20
	Mittelwert lb	8	289	0	218	-	373
	Mittelwert ub	10	291	5	220	-	375
	Median lb	6	247	0	214	-	273
	Median ub	6	247	5	214	-	273
	95. Perzentil lb	25	746	0	583	-	1.203
	95. Perzentil ub	25	746	5	583	-	1.203
Panierte nicht vorfrittierte tiefgekühlte Fischerzeugnisse[2]	N	5	5	5	5	-	5
	< LOQ (%)	100	100	100	100	-	100
	Mittelwert lb	0	0	0	0	-	0
	Mittelwert ub	20	10	20	10	-	10
	Median lb	0	0	0	0	-	0
	Median ub	20	10	20	10	-	10
	95. Perzentil lb	0	0	0	0	-	0
	95. Perzentil ub	20	10	20	10	-	10
Kekse[3]	N	31	57	-	57	20	47
	< LOQ (%)	35	26	-	79	0	72
	Mittelwert lb	217	288	-	44	318	91
	Mittelwert ub	218	316	-	158	318	154
	Median lb	188	213	-	0	181	0
	Median ub	188	213	-	125	181	125
	95. Perzentil lb	822	770	-	324	1.004	455
	95. Perzentil ub	822	770	-	324	1.004	455
Knabbererzeugnisse aus Kartoffeln[3,a]	N	6	84	-	40	30	40
	< LOQ/LOD (%)	67	35	-	55	3	93
	Mittelwert lb	3	236	-	80	538	8
	Mittelwert ub	4	267	-	116	542	79
	Median lb	0	170	-	0	420	0
	Median ub	4	170	-	98	420	98
	95. Perzentil lb	8	804	-	388	1.445	101
	95. Perzentil ub	8	804	-	388	1.445	116

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- Fortsetzung Anhang II –

		Freies 3-MCPD	Gebundenes 3-MCPD	Freies 2-MCPD	Gebundenes 2-MCPD	Freies Glycidol	Gebundenes Glycidol
Geräucherte Rohwurstwaren[3]	N	172	-	-	-	-	-
	< LOQ/LOD (%)	22	-	-	-	-	-
	Mittelwert lb	23	-	-	-	-	-
	Mittelwert ub	24	-	-	-	-	-
	Median lb	16	-	-	-	-	-
	Median ub	16	-	-	-	-	-
	95. Perzentil lb	71	-	-	-	-	-
	95. Perzentil ub	71	-	-	-	-	-
Geräucherte Schinken[3]	N	147	15	-	-	-	-
	< LOQ/LOD (%)	19	93	-	-	-	-
	Mittelwert lb	23	4	-	-	-	-
	Mittelwert ub	25	13	-	-	-	-
	Median lb	15	0	-	-	-	-
	Median ub	15	9	-	-	-	-
	95. Perzentil lb	71	19	-	-	-	-
	95. Perzentil ub	71	28	-	-	-	-
Geräucherter Speck[3]	N	34	-	-	-	-	-
	< LOQ/LOD (%)	32	-	-	-	-	-
	Mittelwert lb	15	-	-	-	-	-
	Mittelwert ub	15	-	-	-	-	-
	Median lb	11	-	-	-	-	-
	Median ub	11	-	-	-	-	-
	95. Perzentil lb	58	-	-	-	-	-
	95. Perzentil ub	58	-	-	-	-	-

LOQ: *limit of quantification*, LOD: *limit of detection*
lb: *lower bound*, ub: *upper bound*
Datenquelle: [1] Projekt (SGS Fresenius), [2] Projekt (MRI), [3] BVL
[a] Kartoffelchips, -sticks, Stapelchips

Über das BfR

Das Bundesinstitut für Risikobewertung (BfR) ist eine wissenschaftlich unabhängige Einrichtung im Geschäftsbereich des Bundesministeriums für Ernährung und Landwirtschaft (BMEL). Es berät die Bundesregierung und die Bundesländer zu Fragen der Lebensmittel-, Chemikalien- und Produktsicherheit. Das BfR betreibt eigene Forschung zu Themen, die in engem Zusammenhang mit seinen Bewertungsaufgaben stehen.

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