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VKM Report 2022: 16

Risk assessment of dioxins, furans, and dioxin-like PCBs in food in Norway

Scientific Opinion of the Panel on Contaminants of the Norwegian Scientific Committee for Food and Environment

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Preparation of the opinion

The Norwegian Scientific Committee for Food and Environment (Vitenskapskomiteen for mat og miljø, VKM) appointed a project group to draft the opinion. Two referees commented on and reviewed the draft opinion. The Committee, by the Panel on Contaminants, assessed and approved the final opinion.

Authors of the opinion

The authors have contributed to the opinion in a way that fulfils the authorship principles of VKM. The principles reflect the collaborative nature of the work, and the authors have contributed as members of the project group and/or the VKM Panel on Contaminants.

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VKM would like to thank the referees Senior Advisor Monica Andreassen (Norwegian Institute of Public Health) and Professor Jan Alexander (Retired, former Norwegian Institute of Public Health) for their valuable comments through critical review of the draft opinion. VKM emphasises that the referees are not responsible for the content of the final opinion. In accordance with VKM's routines for approval of a risk assessment, VKM received their comments before evaluation and approval by VKM Panel on Contaminants, and before the opinion was finalised for publication.

Competence of VKM experts

Persons working for VKM, either as appointed members of the Committee or as external experts, do this by virtue of their scientific expertise, not as representatives for their employers or third-party interests. The Civil Services Act instructions on legal competence apply for all work prepared by VKM.

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Erratum

The following have been corrected (13.06.2022):

- Chapter 5.1.1: The text is revised and two new references have been included.
- Appendix: Some table numbers have been corrected.
- Appendix: The unit has been added for some tables.

Summary

The Norwegian Food Safety Authority asked VKM to perform exposure assessments of dioxins and dioxin-like PCBs (DL-PCBs) for the total Norwegian diet and assess if the Norwegian population or sub-groups of the population, have different eating patterns leading to different dietary dioxin and DL-PCB exposures compared to what EFSA reported for the European population (EFSA, 2018a). Furthermore, VKM was asked to assess the risk from dioxins and DL-PCBs exposures from marine oils taken as food supplements and from reindeer consumption. VKM should also assess health consequences of exceeding the tolerable weekly intake (TWI), both related to duration and degree of TWI exceedances. Finally, VKM was asked to identify risk-reducing factors, which could reduce dioxin and DL-PCB exposure in the population.

Dioxins and DL-PCBs are a group of persistent, lipid soluble and highly toxic organic pollutants that accumulate in the food chain. In this risk assessment of dioxins and DL-PCBs, 29 individual substances (congeners) belonging to polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs) and dioxin-like polychlorinated biphenyls (DL-PCBs) are included.

These 29 individual substances act in the same manner and are therefore assessed together as a group. Because each of the substances have different toxic potential, weighting factors (toxic equivalence factor, TEF) are used to describe the toxicity of each compound relative to the most toxic substance. The total amount of PCDD/Fs and DL-PCBs is given as picogram toxic equivalents (pg TEQ), which is an expression of the total toxic potential of the sum of PCDD/Fs and DL-PCBs.

In Norway as well as in Europe a strong decline in the levels of PCDD/Fs and DL-PCBs in blood and breast milk since the 1980ties has been documented. This indicate that measures taken to decrease the environmental release and exposure from food have been effective. From 1986 to 2005 the concentration of PCDD/Fs and DL-PCBs in breast milk from first-time mothers in Norway decreased by approximately 70%. However, concentration in breast milk from other European countries indicate that the declining trend is levelling off the last decade.

Tolerable weekly intake (TWI)

A TWI of 2 pg TEQ/kg bw per week was established by EFSA (2018a), based on decreased sperm concentration in men after prenatal and childhood exposure to PCCD/Fs and DL-PCBs. The TWI was used to characterize the risk related to estimated exposure to PCCD/Fs and DL-PCBs from food for the Norwegian population.

Consumption data

Food consumption data used for the exposure estimation was obtained from four national dietary surveys: Norkost 3 (18-70-year-olds), Ungkost 3 (4-, 9- and 13-year-olds), Småbarnskost 3 (2-year-olds), and Spedkost 3 (1-year-olds). These are national dietary surveys designed to estimate habitual intake in a representative sample of the Norwegian population. Different dietary assessment methods are used in these surveys.

Occurrence data

Occurrence data for eggs, fish, shellfish, grain and grain products, marine oil supplements, meat, milk and other dairy products, and other food groups (including composite foods and food for infants and young children), were used to estimate the exposure. Two different occurrence datasets were used to estimate the exposure; the EFSA dataset containing only occurrence data from European countries as reported by EFSA (2018a), and the VKM dataset containing Norwegian occurrence data for fish, shellfish, meat, eggs, and milk combined with data from EFSA for foods where Norwegian data were lacking.

The available occurrence data indicate that concentrations of PCDD/Fs and DL-PCBs are lower in food produced in Norway than in similar food reported to EFSA by other European countries. This is the case for fish species commonly consumed in Norway (e.g. farmed salmon, mackerel, herring) and in eggs, milk and meat sampled in Norway compared to the concentrations in similar products submitted by European countries. For some foods (e.g. meat) this conclusion is based on only a few samples and has high degree of uncertainty, whereas for fish, milk and eggs a larger number of samples form basis for the conclusion.

Concentrations in food are shown at lower bound (LB) and upper bound (UB). At LB, non-quantifiable PCDD/F and DL-PCB concentrations in food are replaced by zero. At UB, non-quantifiable PCDD/F and DL-PCB concentrations in food are replaced by the limit of quantification. The true concentration values are in the range between the LB and the UB. The LB estimate represents an underestimate of the true concentration whereas the UB exposure estimate represents an overestimate of the true concentration.

Quantified concentrations of PCDD/Fs and DL-PCBs in fruits, vegetables and potatoes were reported by EFSA, whereas no Norwegian occurrence data for fruits, vegetables and potatoes were available. As fruits, vegetables and potatoes generally have low fat content (0.1-0.4%) and are low in the food chain, and PCDD/Fs and DL-PCBs are lipid soluble and accumulate in the food chain, the detection of PCDD/Fs and DL-PCBs in fruits, vegetables and potatoes was unexpected. It is not known whether this is due to local contamination of the food items analysed or if there are other explanations. As high quantities of fruits, vegetables and potatoes are consumed, even low levels of PCDD/Fs and DL-PCBs will influence the total exposure. To account for the uncertainties related to the occurrence data for fruits, vegetables and potatoes, the exposure was estimated both with and without fruits, vegetables and potatoes. However, the results not including fruits, vegetables and potatoes was considered most appropriate, as the presence of PCDD/Fs and DL-PCBs in these foods are hard to explain.

Analytical results from recent (2022) samples of apples, banana, carrots, cauliflower, broccoli, cabbage, and potatoes on the Norwegian market became available after the exposure was calculated by VKM. These results confirm that the concentrations are low and that fruit, vegetables and potatoes are not major contributors to exposure in Norway. New analytical results on meat (cattle, pig, chicken and liver pâté, not included in the exposure assessment) also confirmed low concentrations (NFSA 2022, results made available to VKM).

Approaches used for the exposure estimation

The mean concentrations of PCDD/Fs and DL-PCBs in food were combined with the data on consumption to estimate the exposure.

Chronic exposure was estimated using three different approaches. Using the observed individual mean (OIM) approach, individual daily exposure was estimated from the mean reported food intakes over the dietary survey days. In the weighted OIM (W-OIM) and the mixed model (MM) approaches, survey respondent characteristics including age, the Norwegian county of registration, education level and gender, were weighted to increase the representativity for the general population. Using the MM approach, day-to-day variation within individuals was also corrected. The exposure estimates obtained using the MM approach were considered to result in the most appropriate long-term exposure for Norkost 3 and Ungkost 3. The W-OIM was considered to give the most appropriate results for Småbarnskost 3 and Spedkost 3.

Exposure estimations and evaluation of the risk related to the exposure

In addition to the mean exposure, the high dietary exposure level was also estimated. The high dietary exposure level was considered to be the numeric value at which 95 percent of participants in a survey have exposure below this numeric value, whereas five percent have exposure exceeding this value (the 95th percentile; P95).

An overview of the lowest and highest estimated exposure, from the total diet (not including fruits, vegetables and potatoes), is shown in Table 1. Using the VKM dataset, the estimated exposure ranged from 2.3 (mean LB) to 24.2 (P95 UB) pg TEQ/kg bw per week. Using the EFSA dataset, the estimated exposure ranged from 5.8 (mean LB) to 41 (P95 UB) pg TEQ/kg bw per week.

Table 1. The lowest and highest estimated exposure to PCDD/Fs and DL-PCBs (in pg TEQ/kg bw per week) from the total diet (without fruit, vegetables and potatoes).

Estimated exposure	**VKM dataset**		**EFSA dataset**	
	Lowest exposure	**Highest exposure**	**Lowest exposure**	**Highest exposure**
Mean lower bound	2.3 13-year-olds	7.3 1-year-olds	5.8 Women 18-45 years	18 2-year-olds

Estimated exposure	VKM dataset		EFSA dataset	
	Lowest exposure	Highest exposure	Lowest exposure	Highest exposure
Mean upper bound	4.4 Women 18-45 years	12 2-year-olds	7.2 Women 18-45 years	22 2-year-olds
High exposure (P95) lower bound	4.3 13-year-olds	16 1-year-olds	10 Women 18-45 years	34 2-year-olds
High exposure (P95) upper bound	7.4 Women 18-45 years	24 1-year-olds	12 Women 18-45 years	41 1-year-olds

VKM decided to use the exposure estimates based on the VKM dataset (Norwegian occurrence data for fish, meat, eggs, and dairy products combined with data from EFSA for other foods) without fruit and vegetables as basis for the risk characterisation. This was considered most likely to represent the true exposure for the Norwegian population, after considering the uncertainties connected to contribution of fruits, vegetables and potatoes to the total exposure, comparison of concentrations in food produced domestically to the concentrations in food submitted to ESFA, and the degree of self-sufficiency of different types of food. A comparison of the exposures in adults reported by EFSA and the exposures estimated by VKM based on consumption data in Norkost 3 indicate that the exposure estimates reported by EFSA and by VKM are quite similar, indicating that the exposure in Norway is in similar range as in the rest of Europe.

An overview of the estimated exposure for the different age groups, using the VKM dataset and not including fruits, vegetables and potatoes, is shown in Table 2.

Table 2. Estimated exposure to PCDD/Fs and DL-PCBs (pg WHO_{2005}-TEQ/kg bw/week).

Age group	Sum of PCDD/Fs and DL-PCBs[c]			
	Mean		95-percentile	
	Lower bound	Upper bound	Lower bound	Upper bound
Adults (18-70 years)[a]	2.8	4.6	5.2	7.9
18-45 years (women)[a]	2.5	4.4	4.8	7.4
13-year-olds[a]	2.3	4.7	4.3	8.5
9-year-olds[a]	3.2	6.6	5.6	11
4-year-olds[a]	5.9	11	9.9	16
2-year-olds[b]	7.1	12	15	22
1-year-olds[b]	7.3	12	16	24

[a]mixed model approach; [b]weighted observed individual mean approach [c]without fruits, vegetables and potatoes, obtained using the VKM dataset.

Food groups contributing most to the exposure

At the LB exposure estimates, fish (fatty species), meat and dairy products were the main contributors in all age groups. At the UB exposure, also foods grouped into "other" were

important contributors, in particular for children and adolescents in the age groups 2-13 years of age. This category includes food oils (except for marine oils and butter), drinks, sweets, spices, and food for infants and young children.

Risk characterisation

In the risk characterisation, the estimated exposure is compared to the TWI (2 pg TEQ/kg bw per week). For all age groups included in the exposure assessments, both the mean and the P95 exposures were above the TWI. The percent of the population with mean LB exposure above the TWI ranged from 55% for 13-year-olds to 100% for 4-year-olds, whereas the proportion with mean UB exposures above the TWI ranged from 96% for 13-year-olds to 100% for 2-, 4-, and 9-year-olds.

Estimated exposure based on scenarios for consumption of selected food items, and evaluation of the risk related to the exposure

The Norwegian national dietary surveys do not supply sufficient information about consumption of rarely consumed food. Therefore, consumption scenarios were used to estimate exposure to PCDD/Fs and DL-PCBs from liver from crabs, fish liver, liver from livestock animals and reindeer meat, liver and fat. In addition, consumption scenarios for marine oil supplements and liver pâté were included.

The tolerable intake of PCDD/Fs and DL-PCBs was set by EFSA on a weekly (and not daily or monthly) basis by EFSA. This was done to account for the fact that higher exposure on a single day may not have high impact on the concentration of these substances in the blood provided that the exposure over a week is not exceeding the TWI. If the TWI would have been set on a monthly basis, the blood concentration of PCDD/Fs and DL-PCBs could have been increased substantially by occasional consumption of food with high concentrations. This could lead to elevated exposure of the foetus or sensitive tissues in a critical developmental stage. VKM has therefore not averaged exposure from seldomly consumed foods over a longer time period than one week.

One weekly portion of whole crab meat leads to an exposure 2.8-times the TWI in 13-year-olds and 2.5-times the TWI in adults. Eating filled crab shells that are commercially available, which contains less brown crab meat than the whole crab, leads to exposure that is 2.1-times the TWI in 13-year-olds and 1.4-times the TWI in adults. Crabs are not a major part of the diet and the exposure from crab meat consumption therefore comes in addition to that from the regular diet, which already exceed the TWI. Exposure for children, pregnant and lactating women and women of childbearing age was not calculated because the Norwegian Food Safety Authority has warnings against consumption of brown crab meat for these population groups.

Regarding cod liver, consumption of a small to large portion of cod liver weekly leads to an exposure 4.8- to 14.4-times the TWI in 13-year-olds and 3.1- to 9.3-times the TWI in adults. Cod roe-liver pâté used as bread-spread on one slice of bread weekly contributes with

approximately 50% of the TWI. Exposure for children, pregnant and lactating women was not calculated because the Norwegian Food Safety Authority has warnings against consumption of brown crab meat for these population groups.

Marine oils show a wide variation in concentrations of PCDD/Fs and DL-PCBs. VKM calculated the exposure from the mean concentration in cod liver oil in the data available and a concentration equal to the permitted maximal level (ML) in marine oils applicable in Norway (4.0 pg TEQ/g fat). In adults, a daily intake of 5 mL cod liver oil gives an exposure of 0.5 pg WHO_{2005}-TEQ/g fat (UB) which contributes 25% of the TWI. If the cod liver oil consumed contains PCDD/Fs and DL-PCBs equal to the ML, the weekly exposure from a daily intake of 5 mL is 1.8 pg WHO_{2005}-TEQ/g fat (UB), which is 90% of the TWI for adults. A similar consumption of marine oils in children leads to an exposure above the TWI because of the lower body weight. In 2-year-olds, daily consumption of 5 ml cod liver oil with mean concentrations of PCDD/Fs and DL-PCBs leads to exposure of 3.0 pg TEQ/kg bw per week. If the marine oil consumed in this age group has concentrations equal to the maximum level of 4 pg TEQ/g fat, the exposure will be 11 pg TEQ/kg bw per week, which is similar to the estimated mean dietary UB exposure (12 pg TEQ/kg bw/week) from the total diet for that age group.

The scenario calculations indicate that liver from pork or beef is not a major contributor to the total exposure to PCDD/Fs and DL-PCBs. The uncertainties in the estimates are high, due to estimated and not analysed concentrations in Norwegian beef and pork liver. High daily consumption of bread with liver pâté, e.g. 6 slices of bread, daily) would not make up a high contribution of the total exposure in children (below 0.2 pg TEQ/kg bw per week).

One weekly meal of reindeer meat contributes approximately 23% (UB) of the TWI for adults, whereas a weekly portion of reindeer fat contributes approximately 34% of the TWI in adults. The contribution is higher for adolescents and children due to their lower body weight, despite the lower portion size in children. However, the contribution of a weekly meal of reindeer meat or reindeer fat compared to the mean estimated dietary exposure in the respective age group is similar (for reindeer meat 5.6% in adults and 5.7% in 2-year-olds, for reindeer fat 8.5% in adults and 9.3% in 2-year-olds). Regular consumption of reindeer meat (with or without reindeer fat) is expected to replace other food. The uncertainty in the calculated exposure from reindeer is high due to few samples and that the concentration in reindeer liver was estimated due to lack of analytical data.

Consumption of one weekly portion of reindeer liver contributes more than 12-times the TWI in adults, and the exceedance of the TWI by one portion of reindeer liver is substantial also in adolescents and children. Compared to the estimated UB mean exposure in adults, a weekly meal of reindeer liver would contribute 3-times more. This indicates that reindeer liver consumption is a potential high contributor of PCDD/Fs and DL-PCBs.

Factors that can contribute to exposure reduction

VKM has not been able to identify single factors that alone can reduce the exposure to a level where all parts of the population will have exposure below the TWI for PCDD/Fs and DL-PCB. Instead, several factors may together contribute to a continued declining trend in exposure.

Food is the main source to human exposure to PCDD/Fs and DL-PCBs, and this is due to contaminated environment and/or contaminated animal feed. To reduce the environmental contamination, VKM highlights measures that reduce release, and spread of PCDD/Fs and DL-PCBs. Regarding animal feed, VKM estimated the effect of cleaning of fish oil and fish meal used in feed for farmed salmon on human exposure. The results show that cleaning of both fish oil and fish meal in the fish feed has the potential to reduce the mean total dietary exposure to PCDD/Fs and DL-PCBs by 2.7-6.5% in different age groups. If only fish oil in the feed is cleaned, the reduction in exposure is estimated to 1.8-4.3%.

Conclusion

The dietary exposure to PCDD/Fs and DL-PCBs generally exceed the TWI of 2 pg TEQ/kg bw per week. The main contributing food groups are fish (fatty), milk and dairy products, and meat. These are foods that are central in the diet to fulfil nutritional needs. It should be noted that this risk assessment addresses only the risk of dietary exposure of PCDD/Fs and DL-PCBs and does not take into consideration any beneficial health effects of foods or nutrients in food in a wider context.

VKM notes that there are many environmental and genetic factors that can lead to decreased semen quality and exposure to PCDD/Fs and DL-PCBs above the TWI is regarded as a contributing factor but not sufficient by itself to result in male infertility. The probability of a decrease in sperm concentration increase by higher exceedance of the TWI.

The scenario estimates of exposure from cod liver, whole and filled crab, and reindeer liver indicate that these are significant additional sources. Marine oil consumption may also contribute substantially to PCDD/F and DL-PCB exposure, depending on type of marine oil.

Key words: Crab, dioxin, dioxin-like PCB, DL-PCB, exposure, Norwegian Food Safety Authority, Norwegian Scientific Committee for Food and Environment, liver, marine oils, occurrence, PCDD, PCDF, reindeer, risk assessment, TEF, TEQ, VKM.

Sammendrag på norsk

Mattilsynet ba VKM om å beregne den norske befolkningens eksponering for dioksiner og DL-PCB fra kosten, og vurdere om hele eller deler av befolkningen har et spisemønster som fører til en annen eksponering enn befolkningen i Europa for øvrig (EFSA, 2018a). Videre ble VKM bedt om å vurdere risiko fra dioksiner og DL-PCB fra inntak av kosttilskudd med marine oljer, og fra inntak av reinsdyr. VKM ble bedt om å vurdere helsemessige konsekvenser av å få i seg mer dioksiner og DL-PCB enn det som er fastsatt som tålegrense (TWI; tolerabelt ukentlig inntak), både knyttet til varighet og grad av TWI-overskridelse. VKM ble også bedt om å identifisere faktorer som kan bidra til å redusere den norske befolkningens eksponering for dioksiner og DL-PCB.

Dioksiner og dioksinlignende PCB (DL-PCB) er en gruppe tungt nedbrytbare, fettløselige og svært giftige organiske miljøgifter, som hoper seg opp i næringskjeden. Dioksiner og DL-PCB omfatter 29 enkeltstoffer (kongenere), hvorav 17 er polyklorerte dibenso-p-dioksiner (PCDD) og dibensofuraner (PCDF) og 12 er polyklorerte bifenyler (DL-PCB), og de omtales som PCDD/F og DL-PCB i denne risikovurderingen. Disse 29 enkeltstoffene har samme virkningsmekanisme i kroppen (virker på samme måte) og vurderes derfor samlet. Fordi hvert av stoffene har ulikt skadepotensial, brukes vektingsfaktorer for å beskrive skadepotensialet for hvert av stoffene sammenlignet med det mest toksiske stoffet, og disse kalles toksiske ekvivalensfaktorer (TEF). Den totale mengden av dioksiner og DL-PCB oppgis som pikogram toksiske ekvivalenter (pg TEQ), som altså er et uttrykk for det samlede skadepotensialet.

I Norge og i Europa er det dokumentert en sterk nedgang i nivåene av PCDD/F og DL-PCB i blod og morsmelk siden 1980-tallet. Dette indikerer at tiltakene som har vært satt inn for å redusere miljøutslipp og eksponering fra mat har vært effektive. Fra 1986 til 2005 gikk konsentrasjonen av PCDD/F og DL-PCB i morsmelk fra førstegangsfødende i Norge ned med ca. 70 %. Målinger av konsentrasjon i mormelk fra andre Europeinske land tyder imidlertid på at den fallende trenden flater ut det siste tiåret.

Tolerabelt ukentlig inntak (TWI)

I 2018 fastsatte Den europeiske myndighet for næringsmiddeltrygghet (EFSA) en tålegrense på 2 pg TEQ/kg kroppsvekt per uke. Den nye tålegrensen ble satt på bakgrunn av at menn som ble eksponert for PCDD/F og DL-PCB i fosterlivet og i barndommen, fikk redusert konsentrasjon av sædceller. VKM brukte denne tålegrensen til å karakterisere risiko ved den estimerte eksponeringen for PCCD/F og DL-PCB fra mat i Norge.

Inntak av mat

Informasjon om kostholdet til ulike aldersgrupper i den norske befolkningen ble hentet fra fire nasjonale kostholdsundersøkelser: Norkost 3 (18-70-åringer), Ungkost 3 (4-, 9- og 13-åringer), Småbarnskost 3 (2-åringer) og Spedkost 3 (1-åringer). De nasjonale

kostholdsundersøkelsene er laget for å estimere hva et representativt utvalg av den norske befolkningen spiser. Det er brukt ulike metoder i disse kostholdsundersøkelsene.

Forekomst av PCDD/F og DL-PCB i mat

Data på innhold/forekomst av disse stoffene i egg, fisk, skalldyr, korn og kornprodukter, kosttilskudd med marine oljer, kjøtt, melk og andre meieriprodukter, og andre matvaregrupper som inkluderer sammensatte produkter, og mat for spedbarn og småbarn, ble brukt for å estimere eksponeringen. VKM brukte to datasett med forekomstdata til eksponeringsberegninger; «EFSA-datasettet», som kun inneholder forekomstdata fra europeiske land som ble rapportert av EFSA (i 2018), og «VKM-datasettet», som inneholder norske forekomstdata for fisk, skalldyr, kjøtt, egg og melk, supplert med forekomstdata for andre matvarer fra EFSA (2018).

De forekomstdataene som er tilgengelige, indikerer at konsentrasjoner av PCDD/F og DL-PCB er lavere i norskprodusert mat enn i tilsvarende matvarer rapportert til EFSA av andre europeiske land. Dette gjelder fiskearter som vanligvis spises i Norge (for eksempel oppdrettslaks, makrell, sild) og i egg, melk og kjøtt. For noen matvarer, for eksempel kjøtt, er denne konklusjonen basert på noen få prøver og har høy grad av usikkerhet, mens det for fisk, melk og egg er et større antall prøver som danner grunnlag for konklusjonen.

Konsentrasjonene av PCDD/F og DL-PCB i mat er er angitt ved «Lower bound» (LB) og «Upper bound» (UB). Ved LB settes ikke-detekterte PCDD/F og DL-PCB til 0, og ved UB settes verdien lik det som er laveste mengde som kan kvantifiseres i analysen (kvantifiseringsgrensen). Den korrekte innholdsverdien er i området mellom LB og UB. LB-estimatet er derfor et underestimat, mens UB-estimatet er et overestimat.

Mens EFSA hadde forekomstdata for PCDD/F og DL-PCB i frukt, grønnsaker og poteter, var det ingen norske analyser av disse matvarene. I og med at frukt, grønnsaker og poteter generelt har lavt fettinnhold (0,1-0,4%) og er langt nede i næringskjeden, mens PCDD/F og DL-PCB er fettløselige og akkumuleres i næringskjeden, var det uventet at det var påviste mengder av PCDD/F og DL-PCB i disse matvaregruppene. Det er ikke kjent om det skyldes lokal forurensning av de analyserte matvarene eller andre forhold. Ettersom frukt, grønnsaker og poteter spises i store mengder, vil selv et lavt innhold av PCDD/F og DL-PCB påvirke resultatet av eksponeringsberegningene. For å ta høyde for usikkerheten knyttet til forekomstdataene for frukt, grønnsaker og poteter, beregnet VKM eksponeringen både med og uten disse matvarene, men beregningen uten frukt, grønnsaker og poteter anses som mest sannsynlig riktig.

I etterkant av VKMs eksponeringsberegninger ble nye analyseresultater (2022) for epler, bananer, gulrøtter, blomkål, brokkoli, kål og poteter fra det norske markedet tilgjengelige. Disse dataene bekrefter antagelsen om at konsentrasjonene er lave, og at frukt, grønnsaker og poteter dermed ikke er store bidragsytere til den norske befolkningens eksponering for PCDD/F og DL-PCB. Nye analyseresultater på kjøtt (storfe, gris, kylling og leverpostei,

dataene er ikke inkludert i eksoneringsberegningene) bekreftet også lave konsentrasjoner i disse norskproduserte matvarene (Mattilsynet 2022, resultater tilgjengelig for VKM).

Metoder brukt for å beregne eksponering

Eksponering ble beregnet ved at data på gjennomsnittlig innhold av PCDD/F og DL-PCB i mat ble kombinert med data på konsum av de ulike matvarene.

VKM brukte tre ulike metoder for å beregne langvarig eksponering for PCDD/F og DL-PCB fra mat for ulike aldersgrupper.

Eksponeringen til hver av deltagerne i kostholdsundersøkelsene ble beregnet ut fra gjennomsnittlig inntak for observasjonsdagene ved en metode som kalles OIM, som er en forkortelse for «observed individual mean». Resultatene fra denne metoden ble brukt til å beregne ulike matvaregruppers bidrag til den totale eksponeringen for PCDD/F og DL-PCB.

Ved de to andre metodene (W-OIM, som er en forkortelse for «weighted OIM» og MM, som er en forkortelse for «Mixed model») ble det gjennomsnittlige inntaket til de ulike deltagerne i kostholdsundersøkelsene vektet for blant annet alder, kjønn og utdannelse for å oppnå bedre representativitet for den norske befolkningen. Ved bruk av MM ble også dag-til-dag variasjoner for hvert individ korrigert. Resultatene fra W-OIM ble brukt til å vise den totale langvarig eksponeringen til 1- og 2-åringer, og MM ble brukt til å beskrive den totale langvarig eksponeringen til voksne og 4-, 9- og 13-åringer.

Beregnet eksponering og vurdering av risiko

VKM beregnet gjennomsnittlig og høy eksponering for ulike aldersgrupper i den norske befolkningen. Høy eksponering ble definert som den verdien hvor 95 prosent av deltakerne har lavere eksponering enn denne verdien, mens fem prosent har høyere eksponering enn denne verdien (kalles 95 persentilen; P95).

Tabell 1 viser en oversikt over den laveste og høyeste estimerte eksponeringen fra hele kostholdet (uten frukt, grønnsaker og poteter). Ved bruk av VKM-datasettet varierte den beregnede eksponeringen fra 2,3 (gjennomsnittlig LB) til 24,2 (P95 UB) pg TEQ/kg kroppsvekt per uke. Ved bruk av EFSA-datasettet varierte den estimerte eksponeringen fra 5,8 (gjennomsnittlig LB) til 41 (P95 UB) pg TEQ/kg kroppsvekt per uke.

Tabell 1. Laveste og høyeste estimerte eksponering for PCDD/F og DL-PCB (i pg TEQ/kg kroppsvekt per uke) fra hele kosten, med unntak av frukt, grønnsaker og poteter.

Estimert eksponering	VKM datasettet		EFSA datasettet	
	Lavest eksponering	Høyest eksponering	Lavest eksponering	Høyest eksponering
Gjennomsnitt "lower bound" (LB)	2,3 13-åringer	7,3 1-åringer	5,8 Kvinner, 18-45 år	18 2-åringer

Estimert eksponering	VKM datasettet		EFSA datasettet	
	Lavest eksponering	Høyest eksponering	Lavest eksponering	Høyest eksponering
Gjennomsnitt "upper bound" (UB)	4,4 Kvinner, 18-45 år	12,3 2-åringer	7,2 Kvinner, 18-45 år	22 2-åringer
Høy eksponering (P95) "lower bound" (LB)	4,3 13-åringer	15,8 1-åringer	10 Kvinner, 18-45 år	34 2-åringer
Høy eksponering (P95) "upper bound" (UB)	7,4 Kvinner, 18-45 år	24,2 1-åringer	12 Kvinner, 18-45 år	41 1-åringer

VKM besluttet å bruke eksponeringen som er basert på VKM-datasettet og beregnet uten frukt, grønnsaker og poteter, som grunnlag for risikokarakteriseringen. Bak avgjørelsen ligger en helhetsvurdering av usikkerheten knyttet til bidraget fra frukt, grønnsaker og poteter til den totale eksponeringen, sammenligning av innholdet av PCDD/F og DL-PCB i mat produsert i Norge med innholdet i mat som er rapportert av ESFA, og graden av selvforsyning av ulike typer matvarer. Den estimerte eksponeringen for de ulike aldersgruppene, ved bruk av VKM-datasettet og uten frukt, grønnsaker og poteter, vises i tabell 2.

Tabell 2. Estimert eksponering for PCDD/F og DL-PCB (pg WHO_{2005}-TEQ/kg kroppsvekt/uke).

Aldersgruppe	PCDD/F og DL-PCB[c]			
	Gjennomsnitt		95-percentilen	
	"Lower bound"	"Upper bound"	"Lower bound"	"Upper bound"
Voksne (18-70 år)[a]	2,8	4,6	5,2	7,9
18-45 år (kvinner)[a]	2,5	4,4	4,8	7,4
13-åringer[a]	2,3	4,7	4,3	8,5
9-åringer[a]	3,2	6,6	5,6	11
4-åringer[a]	5,9	11	9,9	16
2-åringer[b]	7,1	12	15	22,4
1-åringer[b]	7,3	12	16	24,2

[a] «mixed model» metoden; [b] vektet «observed individual mean» metoden. [c] uten frukt, grønnsaker og poteter, beregnet med VKM-datasettet.

Matvaregrupper som bidrar mest til den totale eksponeringen

Ved LB-eksponering var fet fisk, kjøtt og melk og meieriprodukter hovedbidragsyterne for alle aldersgrupper. Ved UB-eksponering var matvarer gruppert i kategorien «annet» også viktige bidragsytere, spesielt for barn og ungdom i aldersgruppene 2-13 år. Denne kategorien inkluderer matoljer, drikke, søtsaker, krydder, og mat for spedbarn og småbarn.

Risikokarakterisering

I risikokarakteriseringen sammenlignes den beregnede eksponeringen med tålegrensen som er 2 pg TEQ/kg kroppsvekt per uke. Både gjennomsnitts- og P95-eksponeringen var høyere enn denne for alle aldersgrupper. Prosentandelen av befolkningen med gjennomsnittlig eksponering over tålegrensen varierte fra 55 prosent for 13-åringer til 100 prosent for 4-åringer ved LB eksponering, mens andelen med gjennomsnittlig eksponering over tålegrensen varierte fra 96 prosent for 13-åringer til 100 prosent for 2-, 4- og 9-åringer ved UB eksponering.

Scenarioer for inntak av utvalgte matvarer: beregnet eksponering og vurdering av risiko

VKM lagde scenarioer for inntak av krabbe, fiskelever, lever fra husdyr, og kjøtt, lever og fett fra reinsdyr fordi disse matvarene ikke fanges opp tilstrekkelig i de nasjonale kostholdsundersøkelsene. I tillegg ble det beregnet scenarioer for kosttilskudd med marine oljer og for leverpostei.

Tålegrensen for PCDD/F og DL-PCB ble satt på ukentlig basis (og ikke daglig eller månedlig) av EFSA. Det ble gjort for å ta hensyn til at høy eksponering på en enkelt dag ikke vil ha stor innvirkning på konsentrasjonen av disse stoffene i blodet, forutsatt at eksponeringen i løpet av en uke ikke overstiger tålegrensen. Dersom tålegrensen hadde blitt satt på månedlig basis, kunne blodkonsentrasjonen av PCDD/F og DL-PCB ved enkeltinntak av mat med høye konsentrasjoner ha økt betydelig. Dette kan føre til økt eksponering av for eksempel fosteret eller et vev på et kritisk stadium i utviklingen. Derfor har ikke VKM fordelt eksponering fra mat som spises sjeldent over en tidsperiode som er lengre enn én uke.

Én ukentlig porsjon krabbekjøtt fra hel krabbe fører til en eksponering som er 2,8 ganger høyere enn tålegrensen hos 13-åringer og 2,5 ganger høyere enn tålegrensen hos voksne. I kommersielt tilgjengelige fylte krabbeskjell er det en lavere andel av brun krabbemat enn i en hel krabbe, og inntak av ett krabbeskjell gir en eksponering som er 2,1 ganger høyere enn tålegrensen hos 13-åringer, og 1,4 ganger høyere enn tålegrensen hos voksne. Krabber utgjør ikke en stor del av kostholdet og eksponering fra krabbe kommer derfor i tillegg til eksponeringen fra det vanlige kostholdet, som allerede overstiger tålegrensen. Eksponering fra krabbekjøtt for barn, gravide, ammende mødre og kvinner i fertil alder, ble ikke beregnet fordi Mattilsynet advarer disse gruppene mot å spise brunt krabbekjøtt.

Ukentlig inntak av en liten til en stor porsjon torskelever gir en eksponering som er 4,8 til 14,4 ganger høyere enn tålegrensen hos 13-åringer, og 3,1 til 9,3 ganger høyere enn tålegrensen hos voksne. Rognleverpostei fra torsk brukt som brødpålegg på én brødskive ukentlig bidrar med ca. 50 prosent av tålegrensen. Det ble ikke beregnet eksponering for barn, gravide og ammende mødre fordi Mattilsynet advarer disse gruppene mot å spise lever fra fisk og rognleverpostei fra torsk.

Det er stor variasjon i innholdet av PCDD/F og DL-PCB i ulike marine oljer. I beregninger av tran brukte VKM gjennomsnittlig innhold, mens den gjeldende grenseverdien for hva som tillates i Norge (4,0 pg TEQ/g fett) ble brukt for marine oljer. Hos voksne er eksponeringen

fra daglig inntak av 5 ml tran 0,5 pg TEQ/kg kroppsvekt per uke, noe som utgjør 25 prosent av tålegrensen. For marine oljer som inneholder maksimalt tillatt mengde PCDD/F og DL-PCB, er eksponeringen fra daglig inntak av 5 ml på 1,8 pg TEQ/kg kroppsvekt per uke, som utgjør 90 prosent av tålegrensen for voksne. Inntak av samme mengde marine oljer hos barn fører til eksponering over tålegrensen, fordi barn har lavere kroppsvekt. Hos 2-åringer fører daglig inntak av 5 ml tran til eksponering på 3,0 pg TEQ/kg kroppsvekt per uke. Inntak av 5 ml marin olje som inneholder maksimalt tillatt mengde PCDD/F og DL-PCB, gir en eksponering på 11 pg TEQ/kg kroppsvekt per uke for 2-åringer. Denne eksponeringen er langt over tålegrensen og tilsvarer den gjennomsnittlige estimerte eksponeringen (12 pg TEQ/kg kroppsvekt) fra hele kosten for denne aldersgruppen.

Scenarioberegningene viser at lever fra svin og storfe ikke er en stor bidragsyter til den totale eksponeringen for PCDD/F og DL-PCB. Usikkerhetene i estimatene er høye, fordi det ikke var analyserte data på innholdet av PCDD/F og DL-PCB i lever, og VKM derfor måtte estimere innholdet. Høyt daglig inntak av brød med leverpostei, for eksempel seks brødskiver daglig, utgjør ikke et høyt bidrag til det totale inntaket hos barn (under 0,2 pg TEQ/kg kroppsvekt per uke).

Ett ukentlig måltid med reinsdyrkjøtt bidrar med ca. 23 prosent (UB) av tålegrensen for voksne, mens en ukentlig porsjon reinsdyrfett bidrar med ca. 34 prosent av tålegrensen hos voksne. Bidraget er høyere for ungdom og barn på grunn av lavere kroppsvekt, til tross for lavere porsjonsstørrelse hos barn. Sammenligner man eksponeringen fra et ukentlig måltid av reinsdyrkjøtt eller reinsdyrfett med den totale estimerte eksponering fra hele kosten for de ulike aldersgruppene, ser man at bidraget utgjør en like stor andel (henholdsvis 5,6 og 5,7 prosent fra reinsdyrkjøtt for voksne og 2-åringer, og 8,5 og 9,3 prosent fra reinsdyrfett for de samme aldersgruppene). Regelmessig inntak av reinsdyrkjøtt,med eller uten fett, forventes å erstatte annen mat. Inntak av én ukentlig porsjon reinsdyrlever bidrar med mer enn 12 ganger tålegrensen hos voksne, og overskridelsen av tålegrensen med én porsjon er betydelig også hos ungdom og barn. Sammenlignet med gjennomsnittlig estimert UB-eksponering fra hele kosten for voksne, vil et ukentlig måltid med reinsdyrlever bidra med tre ganger mer. Dette indikerer at reinsdyrlever er en mulig høy bidragsyter til eksponering for PCDD/F og DL-PCB. Usikkerheten i beregningene av eksponering fra reinsdyr er høy på grunn av lavt antall prøver, og fordi innholdet i lever måtte estimeres i mangel av analysedata.

Faktorer som kan bidra til å redusere eksponering

VKM har ikke klart å identifisere enkeltfaktorer som alene kan redusere eksponeringen for PCDD/F og DL-PCB til et nivå der alle deler av befolkningen vil ha eksponering under TWI. I stedet er det flere faktorer som til sammen kan bidra til at eksponeringen reduseres.

Mat er hovedkilden til eksponering for PCDD/F og DL-PCB. Det skyldes forurenset miljø og/eller forurenset dyrefôr. For å redusere miljøforurensningen er det viktig å fortsatt redusere utslipp og spredning av PCDD/F og DL-PCB. Når det gjelder dyrefôr, beregnet VKM effekten av å rense fiskeolje og fiskemel som brukes i fôr til oppdrettslaks. Resultatene viser

at rensing av både fiskeolje og fiskemel kan redusere gjennomsnittlig total eksponering for dioksiner og dioksinlignende PCB fra kosten med 2,7 - 6,5 prosent i ulike aldersgrupper. Hvis bare fiskeoljen i fôret renses, er reduksjonen i eksponering beregnet til 1,8 - 4,3 prosent.

Konklusjon

Den gjennomsnittlige eksponeringen for PCDD/F og DL-PCB fra kosten er høyere enn tålegrensen på 2 pg TEQ/kg kroppsvekt per uke. Matvaregruppene som bidrar mest er fet fisk, melk og meieriprodukter og kjøtt. Dette er matvarer som er sentrale i kostholdet for å dekke ernæringsbehov. Det påpekes at VKM i denne risikovurderingen kun har vurdert risikoen ved eksponering for PCDD/F og DL-PCB fra kosten, og at det ikke er tatt hensyn til eventuelle gunstige helseeffekter av mat eller næringsstoffer i maten.

Det påpekes at det er mange miljømessige og genetiske faktorer som kan føre til redusert sædkvalitet. Eksponering for PCDD/F og DL-PCB over tålegrensen anses som en medvirkende faktor, men dette i seg selv er ikke tilstrekkelig til å føre til mannlig infertilitet. Sannsynligheten for at spermkonsentrasjonen reduseres, øker ved høyere overskridelse av tålegrensen.

Scenarioestimatene for eksponering fra torskelever, hel og fylt krabbe og reinsdyrlever indikerer at dette er betydelige tilleggskilder. Inntak av marine oljer som tilskudd kan også bidra vesentlig til PCDD/F og DL-PCB eksponering, avhengig av typen olje.

Abbreviations and definitions

Abbreviations

AH receptor	aryl-hydrocarbon receptor
Bw	body weight
DL-PCBs	dioxin-like PCBs
DRE	dioxin responsive elements
EFSA	The European Food Safety Authority
FFQ	food frequency questionnaire
HpCDD	heptachlorodibenzo-p-dioxin
HpCDF	heptachlorodibenzofuran
HxCDD	hexachlorodibenzo-p-dioxin
HxCDF	hexachlorodibenzofuran
KBS	Norwegian food composition database and dietary survey system (in Norwegian: kostberegningssystem)
LB	lower bound
LOD	limit of detection
LOQ	limit of quantitation
ML	maximum level
MM	mixed model
NDL-PCBs	non-dioxin-like PCBs
NFSA	Norwegian Food Safety Authority
OCDD	octachlorodibenzo-p-dioxin
OCDF	octachlorodibenzofuran
OIM	observed individual mean
PCBs	polychlorinated biphenyls
PCDDs	polychlorinated dibenzo-p-dioxins
PCDD/Fs	polychlorinated dibenzo-p-dioxins and dibenzofurans
PCDFs	polychlorinated dibenzofurans
PeCDDs	pentachlorodibenzo-p-dioxins
PeCDFs	Pentachlorodibenzofurans
POPs	persistent organic pollutants
SCF	Scientific Committee on Food
TCDD	tetrachlorodibenzo-p-dioxin
TCDF	tetrachlorodibenzofuran
TEF	toxic equivalence factor
TEQ	toxic equivalent quantity
TWI	tolerable weekly intake
UB	upper bound
VKM	Norwegian Scientific Committee for Food and Environment
WHO	World Health Organization
W-OIM	Weighted observed individual means

Ww	whole weight

Definition of terms used in this risk assessment

Congeners
Congeners are chemical substances related to each other by origin, structure, or function. Chlorinated organic compound congeners share the same molecular backbone (such as biphenyls, dibenzodioxins and dibenzofurans backbones) but have a variable chlorination substitution pattern on this backbone. Examples of compound groups that each contain many congeners are polychlorinated biphenyls (PCBs, 209 congeners), polychlorinated dibenzodioxins (75 congeners), and polychlorinated dibenzofurans (135 congeners).

Consumers only
A term that refers to a calculated intake value based on data from only those who reported consumption of the specific food item.

Dioxin-like
A description used for compounds that have chemical structures, physico-chemical properties, and toxic responses similar to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).

Food
The term food includes food items, beverages and food supplements.

Food composition database and dietary survey system
In the present assessment the Norwegian food composition database and dietary survey system KBS was used (in Norwegian: KostBeregningsSystem). The KBS contains food composition databases, with descriptions of individual food items classified into food groups and broader food categories in a hierarchical food category system. FoodEx2 is the food categorisation system used by EFSA.

Food group
A food group in KBS is a collection of foods that are grouped according to traditional food categories (i.e. "Meat", "Fish and seafood", "Cereals" and so on) and share similar nutritional properties and/or have the same usage. The food groups used in this assessment are grouped according to the Norwegian food composition database and dietary survey system KBS.

Left censored data
Data below a certain value, e.g. the limit of detection or limit of quantification, for which the true value is unknown.

Limit of detection (LOD)
A limit of detection is the lowest concentration of a substance that can be detected with a certain degree of confidence using a validated analytical method.

Limit of quantification (LOQ)
The limit of quantification is the lowest concentration of a substance that can be quantified with a required certainty using a validated analytical method.

Lower bound (LB) estimate
Lower bound estimates are calculated by setting analytical results below the limit of detection (LOD) or limit of quantification (LOQ) of the analytical method to zero. The LB estimate represents an underestimate of the true value, depending on the fraction of analytical results below the LOD or LOQ.

Mixed model (MM)
The mixed model is a statistical model containing fixed and random effects. Mixed models allow estimation of day-to-day variation in the modelled exposure for each survey participant and of clustered variation between survey participants, and simulation of long-term chronic exposure. The model is used to correct for day-to-day variation in the modelled exposure for each survey participant, and for variation between survey participants.

95th percentile exposure
The 95th percentile exposure is the numeric value at which 95 percent of participants in a survey have exposure below this numeric value, whereas five percent have exposure exceeding this value. The 95th percentile is used to describe high dietary exposure level.

Observed individual means (OIMs)
Observed individual means are arithmetic mean exposures for each individual over the dietary survey days, often used as estimates of individual chronic exposure.

Weighted observed individual means (W-OIMs)
Weighted observed individual means extend the standard OIM approach. In estimating distributions, survey responses are weighted to achieve national representativity of participant characteristics.

Tolerable weekly intake (TWI)
The maximum intake of a specific contaminant or contaminant group in food that can be consumed weekly over a lifetime without risking adverse health effects.

Toxic equivalency factor (TEF)
A value representing the relative toxicity of PCDDs, PCDFs and DL-PCBs in relation to TCDD, which is the most toxic compound in this category. The TEF approach for PCDD/Fs and DL-PCBs is based on a common, receptor-mediated mechanism of action for these compounds. To include a compound in the TEF scheme, the following four criteria should be met: the compound should show structural relationship to the PCDDs and PCDFs; it should bind to the

aryl hydrocarbon receptor; it should elicit dioxin-specific biochemical and toxic responses; it should be persistent and accumulate in the food-chain (WHO, 2000).

Toxic equivalent (TEQ)
A weighted quantity measure based on the toxicity of each PCDD, PCDF and DL-PCB relative to TCDD. TEQ for each PCDD, PCDF and DL-PCB is calculated by multiplying the concentration of each congener with its corresponding TEF. The resulting concentration in TEQ for each congener can be summarised as they express TCDD-like toxicities on a common scale.

Upper bound (UB) estimate
Upper bound estimates are calculated by setting analytical results below the LOD or LOQ equal to the LOD or LOQ for the analytical method. The UB estimate represents an overestimate of the true value, depending on the fraction of analytical results below the LOD or LOQ.

Background as provided by the Norwegian Food Safety Authority

Dioxins and dioxin-like (dl-) PCBs are lipophilic environmental chemicals with long half-lives. We are exposed to dioxins and dl-PCBs mainly through consumption of foods with high fat content, like meat, dairy products and fish. Exposure over the years and accumulation in the body may be of health concern. In the newly published risk assessment from the European Food Safety Authority (EFSA, 2018) the tolerable weekly intake (TWI) was reduced from 14 to 2 pg/kg bodyweight/week. According to the EFSA report, the European population is exposed to dioxins and dl-PCBs above the new TWI, and the main food contributors of dioxins and dl-PCBs are fish, seafood, meat, egg and dairy products.

A total dietary exposure assessment of dioxins and dl-PCBs in the Norwegian population has not been conducted. The Norwegian Food Safety Authority (NFSA) asks The Norwegian Scientific Committee for Food and Environment (VKM) to perform exposure assessments of dioxins and dl-PCBs in food in Norway, and to identify sub-populations and/or food categories that require distinct assessments. NFSA will specifically ask VKM to assess the contribution of dioxins and dl-PCBs from reindeer meat. This is because NFSA was contacted by Norwegian Institute for Air Research (NILU) due to high levels of dioxins and dl-PCBs in neck muscle and kidney fat from reindeer in South-Varanger (Norway). The analytical work was conducted as part of a study on local foods in border regions of Norway, Finland and Russia in 2013, 2015 and 2016. There are no national maximum limits (MLs) set for reindeer meat, however the levels in South-Varanger are above the MLs set by the European Union (EU) in 2011 for cattle and sheep meat (4 pg TE/g fat weight). Reindeer meat is included in a regular Norwegian diet, nevertheless we expect it to be consumed relatively rarely in most populations, while some sub-populations on the other hand may consume a lot more than the average.

Furthermore, NFSA will ask VKM to perform a risk assessment on the intake of dioxins and dl-PCBs from marine oils. This request is based on the recommendations set by the Norwegian health authority regarding the intake of cod liver oils and other marine oils, which potentially could have an impact on the dioxin and dl-PCB exposure. Separately, NFSA will also ask VKM to perform a benefit-risk assessment of fish in Norway. As the exposure assessment of dioxins and dl-PCBs in fish will be a central part in both assignments, NFSA asks VKM to ensure consistency.

The toxic equivalent factor (TEF) values of dioxins and dl-PCBs will probably be revaluated by WHO. NFSA therefore requests that exposure calculations are performed using the congener specific values of dioxins and dl-PCBs, which will enable an update on exposure assessments when new TEF-values are available.

Terms of reference as provided by the Norwegian Food Safety Authority

The Norwegian Food Safety Authority (NFSA) asks VKM to

1. Perform exposure assessments of dioxins and dl-PCBs for the total Norwegian diet and assess if the Norwegian population or sub-groups of the population have different eating patterns leading to different dietary dioxin and dl-PCB exposures compared to what EFSA reported for the European population. NFSA asks VKM to assess if separate calculations are needed for sub-groups of the population or for certain food categories (beyond those already mentioned in 2. and 3. below). If yes, NFSA asks VKM to perform the necessary assessments and calculations.
2. Perform a risk assessment of dioxins and dl-PCBs in marine oils taken as food supplements.
3. Calculate how much reindeer meat (with the reported dioxin and dl-PCB values) that can be consumed before the TWI of dioxins and dl-PCBs will be exceeded. Alternatively, what is the additional contribution of dioxins and dl-PCBs from reindeer meat compared to an average diet?
4. Assess health consequences of exceeding the TWI, both related to duration and degree of TWI exceedances.
5. Identify risk-reducing factors, which could reduce dioxin and dl-PCB exposure in the population. If possible, present the risk reducing effects quantitatively.

Interpretation of the terms of reference by VKM

VKM interpreted the terms of reference as follows:

Aim

To estimate the exposure to PCDD/Fs and DL-PCBs from foods and marine oils taken as food supplements, assess possible health risk, and identify risk-reducing factors.

Sub-objectives

- Estimate the dietary exposure to PCDD/Fs and DL-PCBs for the Norwegian population.
- Identify sub-groups with dietary habits resulting in dietary PCDD/Fs and DL-PCBs exposures different from the European population (as reported in EFSA, 2018a).
- Perform separate exposure estimates for the sub-groups identified in point 2.
- Estimate the exposure to PCDD/Fs and DL-PCBs from marine oils taken as food supplements.
- Estimate the exposure to PCDD/Fs and DL-PCBs from reindeer.
- Identify factors that might reduce PCDD/Fs and DL-PCBs exposure in the Norwegian population, and if possible, give a quantitative estimate of the effect.

- Identify and describe possible health consequences resulting from an exposure exceeding the TWI, both related to duration and the degree of exceedance.

Limitations

It was clarified with the NFSA that the food/population group combinations for which the NFSA already give warnings due to the content of PCDD/Fs and DL-PCBs should not be included. An overview is shown in the table below.

Food	**Population group(s) included in the warning**	**Warning**	**Reference**
Brown crab meat	Children, pregnant women, breastfeeding mothers and women of childbearing age	These groups should not eat brown crab meat	Matportalen.no (2021a)
Fish liver and cod roe-liver pâté	Children, pregnant women, and breastfeeding mothers	These groups should not eat fish liver and cod roe-liver pâté	Matportalen.no (2021b)
Liver from self-captured fish	All population groups	Do not eat liver from self-captured fish	Matportalen.no (2021d)
Seagull eggs	Children, pregnant women, breastfeeding mothers and women of childbearing age	Do not eat seagull eggs	Matportalen.no (2021c)
Seagull eggs	The general population	Limit the intake of seagull eggs	Matportalen.no (2021c)

Assessment

1 Introduction

1.1 Dioxins, furans and dioxin-like PCBs

The group of persistent organic pollutants (POPs) often referred to as "dioxins and dioxin-like PCBs" (as in the terms of reference) is a suite of environmental contaminants that are assessed together based on their similar toxicity. At relatively low exposure they are toxic to male reproduction, disturb development, and affect thyroid hormones in newborns (EFSA, 2018a). Some of the group members are classified as carcinogenic (Group 1, carcinogenic to humans) by IARC (2012). The group refers to 29 individual substances belonging to polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs) and dioxin-like polychlorinated biphenyls (DL-PCBs) (Figure 1-1 and 1-2). Substances in each group with a similar backbone and different numbers and positions of chlorines are called congeners. The term non-ortho PCBs refers to PCB congeners with no chlorine substitution at ortho positions of the biphenyl backbone (i.e., the 2, 2', 6, and 6' positions, see Figure 1-2). Mono-ortho PCBs have one of the ortho positions chlorinated, di-ortho congeners have two, etc.

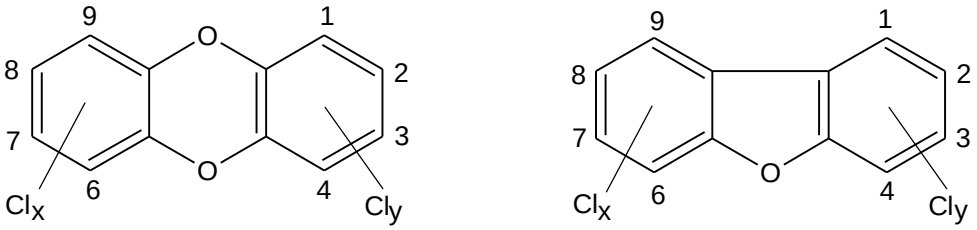

Figure 1-1. General structures of PCDDs (left) and PCDFs (right). Cl_x + Cl_y = 1-8.

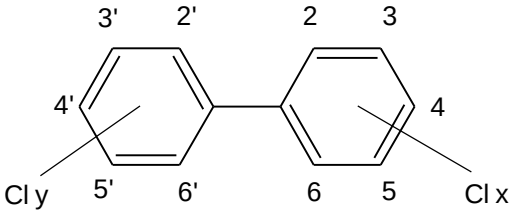

Figure 1-2. General structure of PCBs. Cl_y + Cl_x = 1-10.

Out of the 29 congeners, seven belonging to PCDDs and ten belonging to PCDFs are as a group commonly called "dioxins". However, the term "dioxins" is not used in the present assessment, in which this group of 17 substances are denoted PCDD/Fs.

While PCDDs and PCDFs are formed as by-products of industrial processes such as in bleaching of paper pulp, metallurgic industry, pesticide manufacture and waste incineration, the PCBs are synthetic chemicals that have been manufactured for various technical/industrial purposes. Once formed, these substances degrade very slowly in the environment and combined with their high fat-solubility they tend to accumulate and magnify along food-chains, leading to highest concentrations in predators that occupy the higher trophic levels of ecosystems.

Mixtures of PCDD/F and DL-PCB congeners exists in the environment and in biological material. As the 29 congeners excert toxicity by binding and activating the same receptor (called aryl hydrocarbon receptor, Ah-R), assessments are performed for the group of congeners and not for each of the single congeners. This is done by applying factors accounting for the relative toxicity of the different congeners (toxic equivalent factor (TEF) and dose addition). The chlorine substitution pattern (number and positions, Figure 1-1 and 1-2) of each congener determines their toxic potency, ranging from practically non-toxic to extremely toxic. Seven PCDDs, ten PCDFs and twelve DL-PCBs (a total of 29 different congeners) are categorized as particularly hazardous. The most toxic substance of these 29 congeners is 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD). Each of the other 28 congeners are assigned with their unique TEF relative to 2,3,7,8-TCDD, which has its TEF set as 1. These TEF factors vary over several orders of magnitude (Table 1-1). Concentration data of all individual PCDD/Fs and DL-PCBs in e.g., a food sample are obtained by specific and quantitative chemical analysis. The concentration data for each congener is then multiplied with the respective TEF value and all result values are finally summed up to provide the total dioxin toxic equivalent for the specific sample being analyzed. This sum value is termed as the total toxic equivalent quantity (TEQ) for the specific sample. This total TEQ provides an estimate of the total amount of toxic PCDD/Fs and DL-PCBs present in the sample, described by the corresponding quantity of 2,3,7,8-TCDD. The TEFs for the different congeners were decided under the auspices of the World Health Organization (WHO) in 1998. The most recent revision was in 2005 (Safe, 1990; Van den Berg et al., 1998; Van den Berg et al., 2006). The TEF values used in the present risk assessment were published in 2006 and are termed WHO_{2005}-TEFs (Table 1-1). The TEF values are currently under revision by the WHO and scheduled to be finalized by the end of 2022 (FAO/WHO, 2021).

Table 1-1. Summary of WHO_{2005}-TEF values (Van den Berg et al., 2006).

Congener	**WHO_{2005}-TEFs***
PCDDs	
2,3,7,8-TCDD	1
1,2,3,7,8-PeCDD	1
1,2,3,4,7,8-HxCDD	0.1
1,2,3,6,7,8-HxCDD	0.1

Congener	WHO_{2005}-TEFs*
1,2,3,7,8,9-HxCDD	0.1
1,2,3,4,6,7,8-HpCDD	0.01
1,2,3,4,6,7,8,9-OCDD	0.0003
PCDFs	
2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDF	0.03
2,3,4,7,8-PeCDF	0.3
1,2,3,4,7,8-HxCDF	0.1
1,2,3,6,7,8-HxCDF	0.1
2,3,4,6,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDF	0.1
1,2,3,4,6,7,8-HpCDF	0.01
1,2,3,4,7,8,9-HpCDF	0.01
1,2,3,4,6,7,8,9-OCDF	0.0003
Non-ortho PCBs	
PCB-77	0.0001
PCB-81	0.0003
PCB-126	0.1
PCB-169	0.03
Mono-ortho PCBs	
PCB-105	0.00003
PCB-114	0.00003
PCB-118	0.00003
PCB-123	0.00003
PCB-156	0.00003
PCB-157	0.00003
PCB-167	0.00003
PCB-189	0.00003

* TEF: toxic equivalence factor.

Food, and particularly food from the animal food chain, is the main source of exposure to PCDD/Fs and DL-PCBs in the general population, contributing to more than 90% of the exposure (EFSA, 2018a). The content of PCDD/Fs and DL-PCBs in various food items will practically always be present as a mixture of congeners, and these vary considerably in toxic potency. The TEF-bearing PCDDs, PCDFs and DL-PCBs normally constitute only a fraction of the total quantities of PCDD, PCDF, and PCBs that are present. Therefore, the concept of TEQs (toxic equivalents) is important for assessing toxicity risk associated with dioxin content in different foods and dietary intake (EFSA, 2018a).

1.1.1 Legislation

Dioxins and PCBs are regulated by maximum levels (MLs) in Regulation (EC) No 1881/2006 and amendments in the European Economic Area (Commission Regulation (EC) No 1881/2006). Of note, the MLs for PCDD/Fs and DL-PCBs are not health based.

Concentrations below the ML alone do not ensure that exposure is below the TWI. Based on discussions in the European Commission, MLs are set around the 90th percentile of the frequency distribution of the levels in different food classes.

All maximum levels for PCDD/Fs and DL-PCBs are set as upper bound concentrations. These are calculated on the assumption that all values of the different congeners below the limit of quantification (LOQ) are equal to the numerical value of the LOQ. Except for certain fish and fish products, liver of fish and terrestrial animals, and foods for infants and young children all MLs are given on a fat basis.

The MLs for PCDD/Fs and DL-PCBs as given in Regulation (EC) No 1881/2006 and amendments are applicable in Norway with the exception that the ML for marine oils in Norway (4 pg TEQ/g fat) is lower than that applicable in EU (6.0 pg TEQ/g fat) (Lovdata, 2015). There is no ML for brown crab meat or liver from game animals.

Action levels for these substances are set in a Commission Recommendation on reduction of the presence of dioxins and PCBs (Commission Recommendation 2013). If action levels are exceeded, work must be carried out to identify and eliminate the source of contamination. If maximum levels are exceeded, products cannot be put on the market.

1.2 Tolerable weekly intake

Based on the dioxin TEQ scheme, human health risk assessments have been conducted based on the total exposure to PCDDs, PCDFs and DL-PCBs. In 2001, the EU Scientific Committee on Food (SCF) established a tolerable weekly intake (TWI) of dioxins of 14 pg WHO-TEQ/kg body weight. In 2018, the European Food Safety Authority (EFSA) re-assessed the hazards of PCDDs, PCDFs and DL-PCBs and established a new and lower TWI at 2 pg TEQ/kg bw per week (EFSA et al., 2018a). The TWI was reduced based on new epidemiological and experimental animal data on the toxicity of these substances and more refined methods for predicting the concentrations of the substances in the human body over time.

This TWI is to be used for assessing the risk connected to exposure to PCCD/Fs and DL-PCBs in food in Norway.

The critical effect in the risk assessment by EFSA was decreased sperm concentration in men after prenatal and childhood exposure that has been observed in three cohort studies. A no observed adverse effect level (NOAEL) serum level for PCDD/Fs of 7.0 pg WHO_{2005}-TEQ/g fat at age 9 years was identified from the critical study (The Russian Children's study, Mínguez-Alarcón et al. (2017)).

The Russian Children's Study included participants in 2003-2005 in Chapaevsk in Russia when the boys were 8-9 years of age. Pubertal development was assessed in the boys by yearly examination to age 17-18, and semen samples were taken one year later. Chapaevsk is a city with former production of chlorinated pesticides that ceased in 1987, thus, 7-9 years

before the boys were born. Persistent chlorinated pesticides (HCB, βHCH and DDE) have been analysed and controlled for in the study, in addition to BMI, smoking status, alcohol consumption, season and abstinence time.

Serum concentration of 2,3,7,8-TCDD alone (p trend 0.005) and total PCDD TEQ (p trend 0.02) at age of 8–9 years was associated with a decreased sperm concentration at age 18-19 (n=133 participants delivering 256 semen samples). This was not the case for PCDF-TEQ (p-trend 0.78). However, the association was observed for the sum of PCDD/Fs (p-trend 0.04). DL-PCBs or total TEQ was not associated (p-trend 0.73 and 0.61, respectively). EFSA noted that the lack of association could be due to uncertainties connected to the TEFs, in particular for non-ortho PCBs. The TWI was based on the NOAEL concentration for the sum of PCDD/Fs but includes also the DL-PCBs.

There was a non-linear dose-response association for the sum of PCDD/Fs in the exposure quartiles. The median serum concentrations of PCDD/Fs in the quartiles were respectively 7.0, 10.9, 15.9, and 32.8 pg WHO_{2005}-TEQ/g fat. A decrease in sperm concentration of about 40% was found already in the second quartile and the sperm concentration did not decrease further.

A toxicokinetic model was used by EFSA to estimate the daily intake leading to the NOAEL serum concentration of 7.0 pg WHO_{2005}-TEQ/g fat at the age of 9 years in boys, taking into account prenatal exposure, breastfeeding for 12 months (concentration in breastmilk 5.9 pg TEQ/g fat) followed by dietary exposure until 9 years of age.

EFSA established the tolerable intake on a weekly basis (and not daily or monthly), since day-to-day variation in exposure within a week is not expected to result in a critical increase in concentrations of PCDD/Fs and DL-PCBs in the blood. This could not be assumed for extension to a longer duration of intake (e.g. tolerable intake on a monthly basis) because of the absence of studies and toxicokinetic models that can exclude that a single high dose with, e.g., half of the tolerable monthly intake, could result in a peak concentration in the blood of these substances.

It was noted in the EFSA (2018a) assessment that breastfed infants (< 12 months old) are known to have a higher exposure than toddlers (≥ 1 to < 3 years old) and children (≥ 3 to < 10 years old), and that the exposure of breastfed infants should not be compared to the TWI. These issues were taken into consideration when setting the TWI. If the mother until and during the pregnancy has had dietary intake that is lower than the TWI, then the concentration of PCDD/Fs and DL-PCBs in the breast milk will not reach a level which can increase the risk of health effects in the breastfed child. EFSA took into consideration in the modelling of the tolerable intake that children (≥ 3 to < 10 years old), due to their higher energy demands relative to the body weight, have a factor two times higher exposure to dioxins and DL-PCBs from food than adults. However, children also accumulate PCDD/Fs more slowly than adults because of dilution by increasing body weight. Because the higher exposure during childhood was taken into account in the modelling, children can have a dietary exposure to PCDD/Fs and DL-PCBs two times the TWI after being breastfed for 12

months before the concentration in blood will approach the critical level identified by EFSA at age 9 years. Therefore, exposure two times higher than the TWI (i.e. 4 pg TEQ/kg bw per week) can be considered safe for children of 1 to 9 years of age.

Other effects than the decrease in sperm concentration that were also considered causal by EFSA (2018a) due to PCDD/F exposure were chloracne and other dermal effects, lower sex ratio at birth (boys:girls), developmental effects on teeth and increased thyroid-stimulating hormone (TSH) in newborns (EFSA, 2018a). The effect on development of teeth (enamel hypomineralization) after exposure via breastmilk was estimated by EFSA to be associated with a concentration in breast milk of around 9.2 pg PCDD/F-TEQ/g fat. DL-PCBs were not considered in the epidemiological studies addressing these effects. The other effects considered causally related occur at substantially higher exposure levels and are not relevant at current exposure estimated by EFSA (2018a).

1.2.1 Possible impact of a change in the TEFs

TEFs are internationally agreed weighted values that are based on animal studies and supported by *in vitro* studies. TEFs are used to enable expressing the toxicities of PCDD/Fs and DL-PCBs on a common scale, relative to 2,3,7,8-TCDD. When setting TEFs, the underlying relative effect potencies that are determined for each congener show a large range of values, due to factors like animal species/strain, measured endpoint and duration of exposure. The most recent TEFs (WHO-TEF_{2005}) are rounded based on a log scale, and each value as such presents an order of magnitude in different potencies (see values in table 1-1). TEFs are thus not a precise estimate of the toxic potency of a congener, and this may affect the interpretation of both human and animal studies. In particular, the TEF of PCB-126 was discussed in the EFSA opinion in 2018, and EFSA referred to studies indicating lower potencies in humans than in rodents, which are presently the major basis for the PCB-126 TEF.

Since the TEF of PCB-126 is relatively high compared to the TEFs for the other DL-PCBs, it has high impact on the total TEQ concentration in food or in blood. Of note, the TEFs are updated at irregular intervals based on new scientific information. The TEFs set by WHO in 2005 as published in Van den Berg et al (2006) are currently under revision by WHO and scheduled to be finalized by the end of 2022 (FAO/WHO, 2021).

As the hazard characterization done by EFSA is based on serum concentrations of the sum of PCDD/Fs in the critical study, and extended to include DL-PCBs, a change in TEFs for DL-PCBs will not affect the TWI set by EFSA in 2018. However, a change in TEFs for DL-PCBs will make it necessary to update the exposure assessment to the sum of PCDD/Fs and DL-PCBs based on new TEFs. If the revision of TEFs by WHO result in changes in TEFs for PCDDs or PCDFs that are major contributors to serum levels, also a revision of the TWI might become necessary.

1.3 Previous dietary exposure assessments of PCDD/Fs and DL-PCBs in Norway

Dietary exposure to PCDD/Fs and DL-PCBs in Norway has previously been assessed for participants in the Norwegian Fish and Game study (NFG-study) (Kvalem et al., 2009b) and in pregnant women in the Norwegian Mother, Father and Child Cohort Study (MoBa) (Caspersen et al., 2013; Kvalem et al., 2009b). Both papers used the same database on concentrations of PCDD/Fs and DL-PCBs in food and the same validated food frequency questionnaire (FFQ), with the exception that while participants in MoBa were asked about their average food consumption during pregnancy, the NFG-study participants were asked to report their average food consumption during the previous year.

From a regional survey in 27 selected inland and coastal municipalities with good access to hunting and fishing locations, the Norwegian Fish and Game study recruited 73 randomly selected participants (representative consumers) and 111 high consumers of seafood and game. The estimated lower bound (LB) median intakes of sum PCDD/PCDFs and DL-PCBs of the representative and high consumers were 5.46 and 8.75 pg TEQ_{2005}/kg bw per week, respectively. Fatty fish was the major source of PCDD/PCDFs and DL-PCBs in both high and representative consumers (Kvalem et al., 2009b).

Caspersen et al. (2013) assessed dietary exposure during the first half of pregnancy in 83524 pregnant women (FFQ in gestational week 22) in MoBa during the years 2002 to 2008. The median LB intake was 3.92 pg TEQ_{2005}/kg bw per week. The exposure was thus lower than in the Norwegian Fish and Game study (Kvalem et al., 2009b). This may be ascribed to lower fish consumption among the pregnant women in MoBa. The women reported approximately half the amount of fish and seafood consumed compared to the NFG-study. Caspersen et al. (2013) investigated the contributing dietary sources among women with consumption below and above 14 pg TEQ/kg bw per week (the previous TWI for PCDD/Fs and DL-PCBs). Fatty fish including salmon/trout, cereals and milk, contributed 63% of total intake in women with intake below 14 pg TEQ/kg bw/week. Among women with intake above 14 pg TEQ/kg bw per week (2.3% of the participants), cod roe-liver pâté and seagull eggs, which were consumed by 80% of these women, were the major contributors to the total intake of PCDD/Fs and DL-PCBs.

VKM (2014) reported the mean intake of PCDD/Fs and DL-PCBs from fish only based on consumption in adults in Norkost 3 and occurrence data in fish in Norway to be 1.4 pg TEQ/kg bw/week (LB) and 1.7 pg TEQ/kg bw/week (upper bound (UB)). Fatty fish contributed 76% of the mean intake from fish. However, the contribution from other food was not calculated, so the proportion of the total exposure coming from fish could not be estimated.

1.4 Human biomonitoring data and time trends in Europe and Norway

PCDD/Fs and DL-PCBs are commonly measured in blood or breast milk, and concentrations are generally expressed per unit of fat in the sample, reflecting that these substances are highly fat soluble. Since concentrations of these substances in the body lipids are quite similar, other tissue (e.g., fat tissue) can also provide information on contaminant exposure levels in humans.

In Norway as well as in Europe there is a downward trend in the levels of PCDD/Fs and DL-PCBs in blood and breast milk. From 1986 to 2005 the concentration of PCDD/Fs, DL-PCBs and NDL-PCBs in breastmilk from first-time mothers in Norway decreased by approximately 70% (VKM, 2013). In pooled blood sampled in 2007-2009 and 2019 from women in the Northern Norway mother-and-child contaminant cohort study (MISA) a significant decrease in PCDD/Fs and DL-PCBs (only no-PCBs, mo-PCBs were not analysed) was reported (Xu et al., 2022).

Breast milk samples from Swedish mothers in Stockholm have been analysed for PCDD/Fs and DL-PCBs since 1972 and show a considerable decrease from a concentration of around 82 pg total WHO_{2005}-TEQ/g fat in the 1970s to around 8.0 pg total WHO_{2005}-TEQ/g fat in 2011 (Fång et al., 2013). In Swedish mothers in Uppsala the mean concentration was 5 pg total WHO_{2005}-TEQ/g in 2017 (Gyllenhammar, 2021).

EFSA (2018a) summarized exposure in Europe based on results from the WHO coordinated breast milk surveys as well as other biomonitoring data from Europe. The WHO coordinated breast milk surveys have been conducted regularly since 1987. The Norwegian breast milk data referred to in VKM (2013) are part of the WHO database, but Norway has not participated in the later WHO-surveys. In samples reported by Norway in 2005 the concentration was 7.8 pg WHO_{2005}-TEQ/g fat for the sum of PCDD/Fs and DL-PCBs, of which 4.6 pg TEQ came from PCDD/Fs (reported as data from 2006 in EFSA 2018a). The more recent data from Europe were summarized like this (EFSA, 2018a):

"In summary, the occurrence data from the past few years as reported by individual studies as well as by the WHO field studies, indicate that the current median PCDD/F and DL-PCB levels in human milk from European mothers are generally below 10 pg WHO-TEQ/g fat. Pooled human milk samples collected as part of the WHO field studies across European countries in 2014/2015 revealed levels of 2.4-5.7 pg WHO_{2005}-TEQ/g fat and 4.8–9.6 pg WHO_{2005}-TEQ/g fat for PCDD/Fs and for the sum of PCDD/Fs and DL-PCBs, respectively. The data indicate a substantial global decline of PCDD/F and DL-PCB levels in human milk since the first measured samples collected in the early 1980s. This may be an indication that the measures to decrease the environmental release were effective where applied (UNEP, 2013). However, available results from the last decade are quite similar. Whether this is an indication that the concentrations of PCDD/Fs and DL-PCBs in human milk are levelling off can only be answered if data from future years of monitoring are available."

Decreasing trend in human blood indicates decreasing environmental levels and may therefore indicate decreasing levels in food, leading to lower dietary exposure, provided there are no major dietary changes. A recent report from Institute of Marine Research, Norway, reported a decrease in the level of PCDD/Fs and DL-PCBs in cod liver from the Barents Sea between 2006 and 2019 (Frantzen et al., 2022).

Although the levels in breast milk have decrease the downward trend might have levelled off the last decade (EFSA et al., 2018a; Gyllenhammar et al., 2021; Niedersächsisches Landesamt für Verbrauterschutz und Lebensmittelsicherheit, 2021). Despite efforts to remove known exposure sources there is still background exposure from diffuse or non-identified sources.

Continuous time trend data on concentrations of PCDD/Fs and DL-PCBs for the period 1996 to 2017 in breast milk samples from first-time mothers in Uppsala, Sweden (Gyllenhammar et al., 2021), are shown in Figure 1.4-1. The authors indicated that if the decreasing time trend in total TEQ continue as it has been calculated for the entire time period 1996-2017 (-5.7% per year), 97.5% of first-time mothers in Uppsala area will have body burdens below the estimated safe level (a total TEQ in breast milk resulting from a maternal steady state intake equal to the TWI; 5.9 pg TEQ/g fat) in year 2022. However, if the decreasing tend is following the trend calculated for the years after 2008-2017 (-1.6% per year), this will not be achieved until 2045.

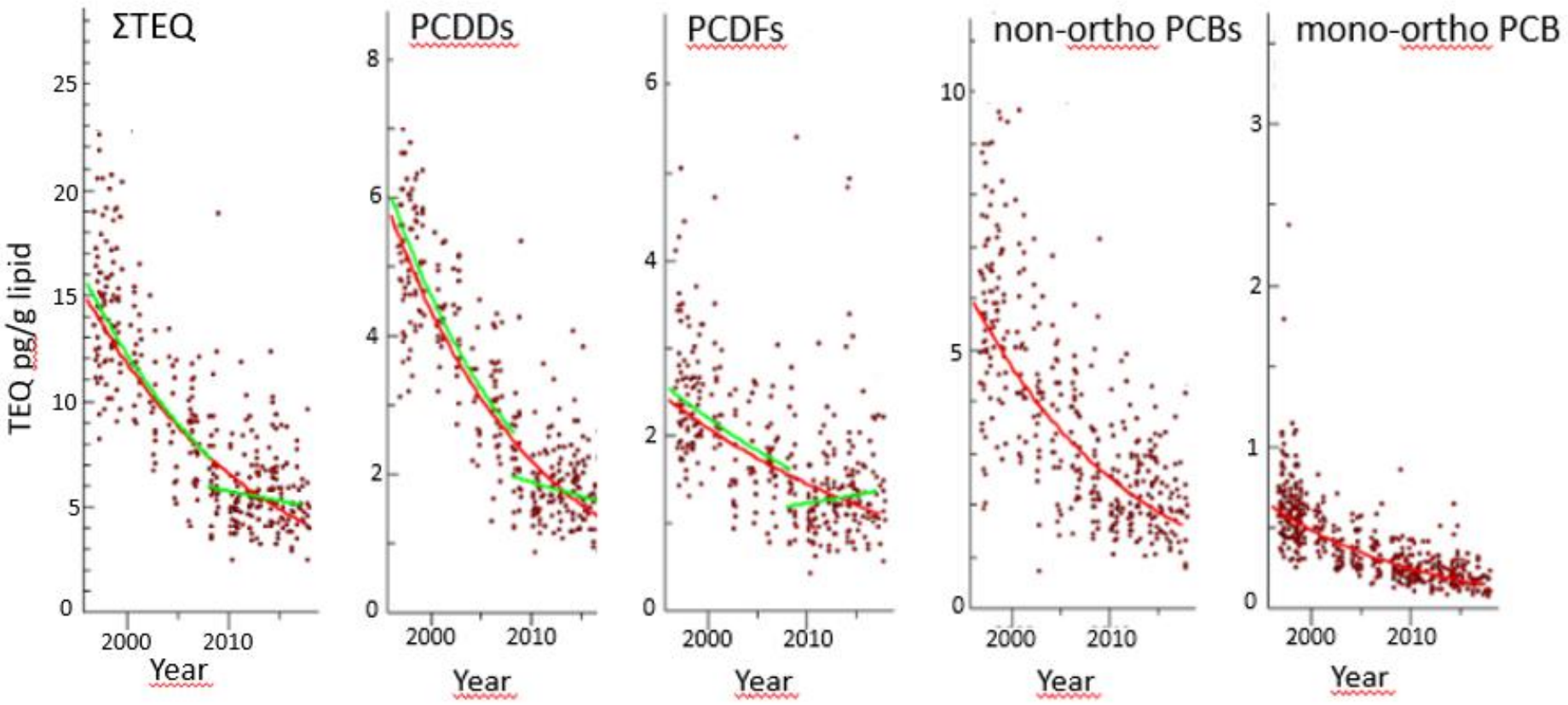

Figure 1.4-1. Concentrations of PCDD/Fs and DL-PCBs from 1996 to 2017 in breast milk samples from first-time mothers in Uppsala, Sweden. The figure is modified from Gyllenhammar et al. (2021). Red line = temporal trend, green line = temporal trend before or after change point year. The authors indicated that the decrease levelled off at around year 2008-2009, which is defined as the change point year.

1.5 EFSA's characterisation of risk from exposure in Europe

EFSA (2018a) estimated chronic LB and UB exposure to PCDD/Fs and DL-PCBs across 35 dietary surveys from Europe covering all age classes. Exposure was estimated for the sum of PCDD/Fs and DL-PCBs (29 congeners) and for PCDD/Fs separately (17 congeners) (Table 1.5-1). The latter exposure assessment was done to estimate the impact of a potentially lower future TEF of PCB-126 (see Chapter 1.2.1). The ratios between the LB and UB estimates across all age classes were small (UB/LB ratio generally <1.5), indicating low uncertainties in the occurrence data. If a large proportion of the analytical results would have been below the LOQ, the UB/LB ratio would have been higher.

Table 1.5-1. Range of chronic dietary intake of PCDD/Fs and DL-PCBs (pg WHO_{2005}-TEQ/kg bw per week) across 35 surveys and age classes, based on data in EFSA (2018a).

	Mean range (pg WHO_{2005}-TEQ/kg bw per week)	95th percentile range (pg WHO_{2005}-TEQ/kg bw per week)
PCDD/Fs and DL-PCBs (29 congeners)	LB: 0.2 to 15 UB: 2.7 to 18	LB: 5.3 to 42 UB: 6.5 to 46
PCDD/Fs (17 congeners)	LB: 0.8 to 6.4 UB: 1.2 to 9.0	LB: 2.1 to 12 UB: 3.0 to 17

The highest exposures were estimated for the age classes toddlers (≥ 1 to < 3 years old) and children (≥ 3 to < 10 years old) and was about two-times higher than in adolescents (≥ 10 to < 18 years old) and all adult age groups (≥18 years).

PCB-126 contributed most to the average mean LB total exposure to PCDD/Fs and DL-PCBs (pg WHO_{2005}-TEQ/kg bw, 29 congeners), followed by 2,3,4,7,8- PeCDF, 1,2,3,7,8-PeCDD, 2,3,7,8-TCDF, PCB-169 and 2,3,7,8-TCDD. As a group, the non-ortho PCBs showed the highest contribution (59%), followed by the PCDFs (23%), PCDDs (14%) and mono-ortho PCBs (5%). Considering only the sum of PCDDs and PCDFs (17 congeners), the PCDFs contributed 62%.

The exposure ranges were compared to the TWI of 2 pg WHO_{2005}-TEQ/kg bw per week. EFSA concluded that the mean exposure to PCDD/Fs and DL-PCBs of adolescents (≥ 10 to < 18 years old), adults (≥ 18 to < 65 years old), elderly (≥ 65 to < 75 years old) and very elderly (≥ 75 years old), exceeded the TWI up to five-times (highest UB). At the 95th percentile (P95), the exceedance ranged from 3 to 15-times. Toddlers (≥ 1 to < 3 years old) and children (≥ 3 to < 10 years old) had a two-times higher exceedance than older age groups.

Regarding PCDD/Fs only, the mean exposure of adolescents and adults were up to two-times higher than the TWI (highest UB), and up to six-times higher than the TWI at the P95.

2 Methods

2.1 Dietary surveys used for the exposure estimates

Dietary assessment data used for the exposure estimates are based on four dietary surveys (Table 2.1-1).

Table 2.1-1. The Norwegian national dietary surveys used for the exposure estimations.

Dietary survey	Year of data collection	Age groups (years)	Survey respondenst (number)	Participation rate (%)	Method used
Norkost 3	2010-2011	18-70	1787	37	Two 24-hour recalls by telephone
			1453	30	Food propensity questionnaire
Ungkost 3	2015/2016	4	399	20	Web-based food diary (4 consecutive days)
		9	636	55	
		13	687	53	
Småbarnskost 3	2019	2	1413	47	Food frequency questionnaire
Spedkost 3	2019	1	1957	66	Food frequency questionnaire

Norkost 3 is a national dietary survey in which food consumption data were collected in 2010-2011 (Totland et al., 2012), carried out by the University of Oslo, the Norwegian Food Safety Authority, the Norwegian Directorate of Health, and the Norwegian Institute of Public Health. Norkost 3 was based on two 24-hour recalls by telephone at least one month apart, and food amounts were presented in household measures or estimated by photographs. Most survey respondents in Norkost 3 also filled in a food propensity questionnaire. The propensity questionnaire consisted of 216 frequency questions of different foods, drinks, dishes, and supplements.

Ungkost 3 is a national dietary survey including 4-, 9- and 13-year-old children (Hansen et al., 2015), carried out by the University of Oslo, the Norwegian Food Safety Authority, the Norwegian Directorate of Health, and the Norwegian Institute of Public Health in 2015 (9- and 13-year-olds) and 2016 (4-year-olds). The dietary assessment tool was a 3-4-days validated web-based food diary.

Småbarnskost 3 is a national dietary survey in which food consumption data for 2-year-olds were collected in 2019 (Astrup et al., 2020), carried out by the University of Oslo and the Norwegian Institute of Public Health. This dietary survey was based on a semi-quantitative food frequency questionnaire (FFQ). The caretaker was asked to have the last two weeks in

mind when answering the questionnaire. In addition to predefined household units, food amounts were also estimated from photographs.

Spedkost 3 is a national dietary survey in which food consumption data for 1-year-olds were collected in 2019 (Paulsen et al., 2020), carried out by the University of Oslo and the Norwegian Institute of Public Health. The dietary survey was based on a semi-quantitative FFQ. The caretaker was asked to have the last two weeks in mind when answering the questionnaire. In addition to predefined household units (eggs, slices of bread, decilitres etc.), food amounts were also estimated from photographs.

2.2 Approaches used for exposure estimation

Exposure to PCDD/Fs and DL-PCBs was estimated by combining reported food consumption (person-day observations) with the mean concentrations of PCDD/Fs and DL-PCBs in food (see Chapter 3.1) for each response-day for the survey respondent. These exposures were divided by individual body weights, as reported in the dietary surveys, and multiplied by seven, to reflect exposure per kilogram of body weight on a weekly basis. The approaches used to estimate exposure distributions are introduced in this chapter. Technical details are presented in the Appendix I (Chapter 10).

The main goal of the chronic exposure estimation is to arrive at a characterisation of the long-term mean exposure for individuals. In assessing chronic exposure to PCDD/Fs and DL-PCBs at the population level in Norway, two main challenges were faced: (i) estimation of habitual intake based on the limited number of days with food consumption data for each dietary survey respondent, and (ii) generalisation from survey respondents to the Norwegian population. If all survey responses are treated as equally representative in the approaches used for exposure estimation, the estimated exposure distributions describe the variation across survey respondents and not necessarily variations in the population as a whole.

The first challenge was addressed by adopting the observed individual means approach (OIM, see Chapter 2.2.1) and the mixed model approach (MM, see Chapter 2.2.3). To address the second challenge, the survey responses were weighted for respondents' demographic characteristics (W-OIM and MM) as described in Chapter 2.2.2.

2.2.1 Observed individual means (OIMs)

The OIM is the mean intake over the survey days for each individual respondent in the dietary surveys. The number of survey days varies by the dietary survey (as presented Chapter 2.1). Classical approaches to exposure analysis and estimation usually consists of summary statistics (mean, percentiles, median etc.) for OIMs.

For Spedkost 3 and Småbarnskost 3, with just one observation per individual, FFQ-data were assumed to average out the day-to-day variation in exposure by design, as the caretaker was asked to cover the intake over the previous two weeks. For Ungkost 3 and Norkost 3,

the daily values were averaged over the survey days at the level of each respondent. Averaging over the survey days reduces informativeness of the survey responses, but at the same time smooths the data somewhat at the level of the individual and produces narrower distribution estimates.

Norkost 3 and Ungkost 3 have two and 3-4 survey days of data per respondent, respectively. This is too little to accurately capture the habitual intake, particularly for food that is not consumed daily or in varying quantities from day to day, potentially resulting in non-representative averages over a small number of days. The standard error of the estimate (a measure of accuracy with which all these individual means can be estimated) is inversely related to the number of days for which intake data are available. First, this implies that, regardless of approach, a lower number of days will lead to an overestimation of the variation between individuals, since there will be more statistical noise in the estimated means for each individual. Second, since most exposure distributions are skewed with long right tails, using OIMs will particularly lead to an overestimation of the upper intake percentiles. For instance, a Norkost 3 participant reporting fish consumption on both survey days does not necessarily consume fish every day. Similarly, participants not reporting fish consumption on either survey day are not necessarily non-consumers of fish.

In this risk assessment, results obtained with the OIM approach are used to calculate the contribution from different food groups to the total PCDD/F and DL-PCB (29 congeners) exposure (Chapter 3.3). In addition, the results obtained using the OIM approach is compared with the result in the EFSA opinion (Chapter 3.4), in which a similar approach is used for many dietary surveys in Europe (EFSA, 2018a).

2.2.2 Population representativity

No survey is perfectly representative of the population it is meant to represent. Some survey respondents are relatively overrepresented, as measured by their demographic characteristics, while others are underrepresented.

One approach to increase population representativity of the estimated distributions is to give the survey respondents different weights based on their demographic characteristics. This was performed adopting the procedure called "raking" (otherwise known as iterative proportional fitting and sample-balancing). Gender, education, age, and geographic regions were chosen as the demographic characteristics used for weighting of surveys, which is in line with the standard choice of characteristics used for survey weighting (Pew Research Center et al., 2018).

The data used in estimation of weights for individual survey respondents were obtained from microdata.no. Microdata.no combines several Norwegian registries including the Norwegian Tax Administration (*Skatteetaten*) and the Norwegian State Educational Loan Fund (*Lånekassen*). The database covers all individuals registered in Norway in the past.

The information from microdata.no was collected as of January 1, 2018, and included the following variables:

- Age group (Norkost 3 only): Age was transformed into five age groups: 18–29, 30–39, 40–49, 50–59, 60+. For Ungkost 3, Småbarnskost 3, and Spedkost 3, each age cohort was analysed separately, and therefore there was no need to weight by age.
- County of registration: Counties were used to assign individuals geographically to regions. There were 7 regions (in Norwegian: landsdeler; as defined by the SSB, Statistics Norway) in Norway as of January 1, 2018.
- Education level: The education levels were set to 1 for respondents with a college degree and above and 0 otherwise. For Ungkost 3, Småbarnskost 3, and Spedkost 3, information on parental education was used, setting the variable to 1 if at least one parent had a college degree.
- Gender

This approach resulted in 140 groups for adults ($2 * 2 * 5 * 7$): two genders, two education levels, five age groups, and seven regions. For 1-, 2-, 4-, 9-, and 13-year-olds, there were 28 groups ($2 * 2 * 7$): two genders, two education levels, and seven regions.

The sizes of the corresponding 140 groups for adults and the 28 groups for 1-, 2-, 4-, 9-, and 13-year-olds were collected from microdata.no. This allowed to compute relative group sizes or, equivalently, group frequencies. Population group frequencies were compared to the corresponding dietary survey group frequencies, and weights were calculated in such a way that the weighted survey frequencies were equal to the population frequencies. These calculated weights were based purely on demographic characteristics and were used for W-OIM estimates for 1- and 2-year-olds and in constructing distributions based on the MM estimates, as described in Chapter 10.3 (Appendix I).

2.2.3 **Weighted observed individual means (W-OIMs)**

By adopting the weights described in Chapter 2.2.2, distributions of person-day observations were made representative for the demographic characteristics. The raw person-day observations were used directly together with the corresponding weights to compute W-OIM distributions, means, standard deviations and percentiles.

2.2.4 Mixed models (MMs)

Food reported eaten during the dietary survey days may not be representative for an individual's consumption of foods not eaten on a daily basis, such as fish. As a consequence, both high and low consumption quantities may be non-representative for the long-term intake for the person. To address this, a modelling approach known as mixed model (MM),

based on Bayesian estimation, was used (for technical details, see Appendix I (Chapter 10.3)).

Using the MM approach in this risk assessment allows us to

- Quantify day-to-day variation within individuals
- Simulate long-term averages for chronic exposure levels
- Translate the chronic exposure levels to distributions representative for the general population within a given age group applying the weights described in Chapter 2.2.2.
- Estimation of confidence intervals around the average distribution of exposure

A general description of the model is presented in Chapter 10.3. For more technical details, please see Chapter 10.4.

In applying the MM, the simulated chronic exposure averages out the day-to-day variation for each survey participant, while weighting for demographic representativity makes the estimated distributions representative. This approach was considered to give the best estimate of the exposure distribution for total exposure for adults, 4-, 9-, and 13-year-olds. As the MM cannot be used for surveys with just one observation per participant and exposure was averaged over a longer observation period, the W-OIM approach was considered to give the best estimate of the exposure distribution for 1- and 2-year-olds.

3 Exposure assessment

3.1 Occurrence data for PCDD/Fs and DL-PCBs in foods

Information on occurrence of PCDD/Fs and DL-PCBs in food was available from several sources.

Concentrations in food reported to EFSA (EFSA database)

The EFSA opinion (2018a) offers the data on occurrence of PCDD/Fs and DL-PCBs in food that were submitted to EFSA (available in Annex B, EFSA, 2018a). Annex B, Tables 2A and 2B include all occurrence data, while Tables 3A and 3B in the same Annex gives an overview of the occurrence data that were used for the exposure assessment by EFSA in their opinion (EFSA, 2018a).

The occurrence data that EFSA used for exposure assessment contained 20,273 values for occurrence of PCDD/Fs (17 congeners) and 19,965 values for DL-PCBs (12 congeners) from the period 2010-2016, which were submitted by 23 European countries, including Norway (concentrations in 2,128 fish samples).

Norwegian occurrence data (VKM database)
Norwegian occurrence data (analytical results on food produced in Norway) were obtained for fish, other seafood, fish roe and liver, marine oils (supplement), egg, meat, and milk. An overview of the data and the data providers, are presented in Table 3.1-1.

A more detailed overview of the Norwegian occurrence data used in the exposure estimation, and examples on EFSA occurrence data for similar foods, is given in Table 3.1-2. Both upper bound (UB) and lower bound (LB) concentrations are shown. Since the final concentration values are the sums of the individual analytical results of the 29 PCDD/F and DL-PCB congeners or the 17 PCDD/F congeners, the percentage of values below LOQ (left-censored data) among the food categories is not considered informative. This is also in line with how EFSA presented the food occurrence data.

Table 3.1-1. An overview of sources of occurrence data in Norwegian food used in the present risk assessment.

Food group	**Food**	**Provider of data**	**Analysed by**	**Year of sampling**	**Reference**
Lean fish (<5% fat)	Atlantic cod, plaice, rose fish, saithe, and wolfish	IMR	IMR	2010-2020	Data received directly from IMR
Fatty fish (≥5% fat)	Farmed Atlantic salmon, farmed trout, mackerel, herring, Atlantic halibut	IMR	IMR	2010-2020	Data received directly from IMR
Other seafood	Crab, brown meat	VKM	NIFES	Before 2010***	NIFES (2004)
Cod roe and liver	Liver	IMR	IMR	2010-2020	Data received directly from IMR
	Roe	Not given	Not given	2005***	Kvalem et al., 2009a
	Cod roe-liver pâté*	NFSA	NIFES	Before 2010***	Julshamn and Frantzen (2009)
Marine oils (supplement)	Cod liver oil and fish oil	NFSA	NIFES and IMR	2013-2018	Nilsen and Måge (2014); Nilsen and Måge (2015); Nilsen and Måge (2016); Nilsen and Måge (2017); Nilsen and Sanden (2018); Nilsen and Sanden (2019).
Egg	Egg, chicken	NFSA	NIFES	2016	NIFES (2016)
Meat	Meat, cattle	NFSA	FERA	2018	Data received directly from NFSA
	Meat, chicken	NFSA	FERA	2014-2017	Data received directly from NFSA
	Liver pâté	NIPH	Not given	2003-2004***	Kvalem et al., 2009a
	Pork	NFSA	FERA	2018	Data received directly from NFSA
	Reindeer	NIPH	NIPH****	2012, 2018	Bremnes et al. (2012); Bremnes and Thomsen (2018)
	Reindeer	NILU	NILU**	2013, 2015, 2016	NILU (2017)
	Reindeer	NFSA	FERA	2018	Data received directly from NFSA
	Sheep	NFSA	FERA	2018	Data received directly from NFSA
	Sheep	NIPH	NIPH****	2021	Bremnes et al. (2022)

Food group	**Food**	**Provider of data**	**Analysed by**	**Year of sampling**	**Reference**
Milk	Milk, cow	NFSA	FERA	2014-2017	FERA et al. (2018) *Errors in the reported unit for single congeners were corrected by VKM (specified in an e-mail from FERA to NFSA)*
		Tine SA	Eurofins	2020	Unpublished data received from Tine SA

IMR: Institute of Marine Research; NFSA: Norwegian Food Safety Authority; NILU: Norwegian Institute for Air Research; NIFES: National institute of Nutrition and Seafood Research; NIPH: National Institute of Public Health.

*Cod roe-liver pâté, a canned bread spread made mainly of cod roe and cod liver (38% roe, 24% liver).

** Mono-ortho PCBs were available for 6 of the 15 samples.

*** The samples were taken before 2010 (see deviations from the protocol).

**** Consensus value, analysed by multiple laboratories as part of a ring test.

Table 3.1-2. Mean concentration (pg WHO_{2005}-TEQ/g) of PCDD/Fs and DL-PCBs in foods and food groups used in this assessment.

Food	**pg/g ww[c] or fat**	**Sum of PCDD/Fs (17 congeners)**					**Sum of PCDD/ Fs and DL-PCBs (29 congeners)**					**Data source[b]**
		n	Mean (LB)	Mean (UB)	P95[a] (LB)	P95[a] (UB)	n	Mean (LB)	Mean (UB)	P95[a] (LB)	P95[a] (UB)	
Lean fish (<5% fat)												
Atlantic cod	ww	60	0.003	0.04	0.01	0.06	60	0.028	0.064	0.066	0.117	VKM
Cod and whiting	ww	384	0.04	0.08	0.09	0.21	375	0.171	0.284	0.48	0.88	EFSA
Plaice	ww	54	0.119	0.120	0.263[d]	0.315[d]	54	0.441	0.521	0.943[d]	1.031[d]	VKM
Plaice	ww	63	0.19	0.218	0.709	0.736	61	0.459	0.505	1.534	1.617	EFSA
Rose fish	ww	746	0.133	0.207	0.381	0.436	746	0.520	0.594	1.528	1.604	VKM
Saithe	ww	51	0.005	0.03	0.01[d]	0.05[d]	51	0.066	0.093	0.133[d]	0.162[d]	VKM
Wolffish	ww	38	0.018	0.048	0.054[d]	0.081[d]	38	0.059	0.090	0.144[d]	0.192[d]	VKM
Sea catfish and wolf-fish	ww	69	0.059	0.087	0.485	0.491	69	0.126	0.155	0.626	0.643	EFSA

Food	pg/g ww[c] or fat	Sum of PCDD/Fs (17 congeners)					Sum of PCDD/ Fs and DL-PCBs (29 congeners)					Data source[b]
		n	Mean (LB)	Mean (UB)	P95[a] (LB)	P95[a] (UB)	n	Mean (LB)	Mean (UB)	P95[a] (LB)	P95[a] (UB)	
Fatty fish (≥5% fat)												
Farmed Atlantic salmon	ww	1074	0.079	0.242	0.180	0.360	1074	0.391	0.555	0.651	0.829	VKM
Trout, farmed	ww	24	0.042	0.234	0.118[d]	0.325[d]	24	0.295	0.488	0.550[d]	0.682[d]	VKM
Salmon and trout	ww	907	0.27	0.33	1.93	1.95	857	0.88	0.94	5.82	5.82	EFSA
Mackerel (autumn)	ww	541	0.221	0.389	0.863	1.011	541	0.831	1.002	2.869	2.905	VKM
Mackerel	ww	322	0.37	0.43	1.23	1.24	317	1.36	1.44	4.72	4.78	EFSA
Herring	ww	150	0.376	0.465	0.650	0.713	150	0.805	0.895	1.342	1.404	VKM
Herring	ww	401	1.22	1.25	1.93	1.95	399	2.34	2.39	6.36	6.36	EFSA
Atlantic halibut	ww	389	0.328	0.375	0.975	0.991	389	1.345	1.392	3.430	3.449	VKM
Halibut	ww	466	0.31	0.35	0.92	0.94	466	1.12	1.16	3.32	3.36	EFSA
Other seafood												
Crab, brown meat	ww	435	1.910	2.057	4.454	4.774	435	3.470	3.617	8.041	8.063	VKM
Crab, brown and white	ww	275	0.62	0.63	2.28	2.28	274	1.26	1.27	4.18	4.18	EFSA
Roe and liver												
Cod liver	ww	1207	2.37	3.30	6.50	7.06	1207	15.16	16.09	37.49	38.31	VKM
Fish offal	ww	911	4.33	4.89	12.7	13.1	911	21.7	22.0	60.3	60.5	EFSA
Cod roe-liver pâté	ww	2	3.0	3.1	na	na	2	0.46	0.55	na	na	VKM
Cod roe[e]	ww	4	0.074	na	na	na	4	0.321	na	na	na	VKM
Marine oils (supplement)												
Cod liver oil	fat	12	0.025	0.247	0.117[d]	0.502[d]	12	0.845	1.080	3.903[d]	4.066[d]	VKM
Cod liver oil	fat	7	0.553	0.631	na	na	7	3.014	3.093	na	na	EFSA
Fish oil	fat	25	0.951	1.130	4.503[d]	4.552[d]	25	4.943	5.135	22.267[d]	23.192[d]	VKM
Fish oil	fat	21	0.130	0.244	na	na	21	1.220	1.336	na	na	EFSA
Meat												
Cattle	fat	19	0.157	0.232	0.395[d]	0.417[d]	19	0.306	0.387	0.695[d]	0.804[d]	VKM
Cattle	fat	869	0.53	0.61	1.68	1.68	866	2.14	2.23	6.08	6.08	EFSA

Food	pg/g ww[c] or fat	Sum of PCDD/Fs (17 congeners)					Sum of PCDD/ Fs and DL-PCBs (29 congeners)					Data source[b]
		n	Mean (LB)	Mean (UB)	P95[a] (LB)	P95[a] (UB)	n	Mean (LB)	Mean (UB)	P95[a] (LB)	P95[a] (UB)	
Beef liver	ww	183	0.06	0.07	0.18	0.19	181	0.15	0.15	0.41	0.41	EFSA
Chicken	fat	5	0	0.333	na	na	5	0.095	0.576	na	na	VKM
Chicken	fat	573	0.14	0.26	0.45	0.58	565	0.30	0.43	0.93	1.09	EFSA
Pork	fat	7	0.009	0.130	0.016[d]	0.160[d]	7	0.024	0.173	0.044[d]	0.227[d]	VKM
Pork	fat	459	0.08	0.162	0.33	0.36	454	0.139	0.236	0.45	0.52	EFSA
Liver pâté[e]	fat	3	0.249	na	na	na	3	0.398	na	na	na	VKM
Pâté, pork liver	fat	24	0.22	0.27	na	na	24	0.25	0.30	na	na	EFSA
Pork liver	ww	5	0.134	0.140	na	na	55	0.12	0.13	na	na	EFSA
Reindeer[f]	fat	19	na	3.10	na	na	19	na	6.89	na	na	VKM
Sheep	fat	7	0.251	0.365	0.533[d]	0.696[d]	7	0.478	0.592	0.813[d]	0.964[d]	VKM
Sheep	fat	241	0.50	0.57	1.43	1.43	240	0.95	1.05	2.55	2.56	EFSA
Milk												
Cow milk	fat	62	0.071	0.279	0.175	0.632	60	0.176	0.413	0.434	0.786	VKM
Cow milk	fat	948	0.278	0.449	0.90	0.98	935	0.747	0.916	1.79	2.01	EFSA
Egg												
Whole egg, chicken	fat	146	0.139	0.468	0.447	1.251	143	0.249	0.579	0.779	1.359	VKM
Whole egg, chicken	fat	2328	0.472	0.582	1.79	1.79	2312	1.18	1.31	4.32	4.32	EFSA
Grain												
Wheat bread and rolls	ww	2	0.007	0.018	na	na	0	na	na	na	na	EFSA
Fruit, vegetables and potatoes												
Apple	ww	3	0	0.151	na	na	3	<0.001	0.160	na	na	EFSA
Brussel sprouts	ww	1	0.005	0.008	na	na	1	0.009	0.012	na	na	EFSA
Courgettes	ww	12	0.010	0.018	na	na	5	0.014	0.019	na	na	EFSA
Tomatoes	ww	2	<0.001	0.019	na	na	2	0.002	0.023	na	na	EFSA
Main crop potatoes	ww	1	0.004	0.005	na	na	1	0.003	0.005	na	na	EFSA

Food	pg/g ww[c] or fat	Sum of PCDD/Fs (17 congeners)					Sum of PCDD/ Fs and DL-PCBs (29 congeners)					Data source[b]
		n	Mean (LB)	Mean (UB)	P95[a] (LB)	P95[a] (UB)	n	Mean (LB)	Mean (UB)	P95[a] (LB)	P95[a] (UB)	
Vegetables and vegetable products	ww	164	0.02	0.05	0.12	0.21	136	0.05	0.08	0.26	0.28	EFSA
Other food groups[g]												
Olive oil	fat	43	0.040	0.105	na	na	43	0.061	0.172	na	na	EFSA
Rapeseed oil	fat	15	0.003	0.055	na	na	15	0.008	0.063	na	na	EFSA
Sunflower oil	fat	88	0.074	0.131	na	na	88	0.086	0.158	na	na	EFSA
Foods for infants and young children	ww	500	0.00	0.01	0.02	0.04	472	0.01	0.02	0.04	0.07	EFSA

na: Not available.

[a] P95: 95th percentile.

[b] EFSA: data from EFSA (2018a); VKM: analytical results in foods from Norway.

[c] Whole weight.

[d] The number of samples is low, and gives more uncertainty to the P95 values.

[e] Cod roe, and liver pâté from Kvalem et al., 2009a. Liver pâté concentrations were calculated from ww based on 22.1% fat as given by the authors.

[f] Mono ortho-PCB missing in 9 samples.

[g] Other food groups are composite foods that are not assigned any other category in the KBS, food oils (except for marine oils and butter), drinks, sweets, spices, and food for infants and young children.

3.1.1 Basis for use of national and EFSA occurrence data for exposure assessment

The occurrence data in Table 3.1-2 indicate that concentrations of PCDD/Fs and DL-PCBs are lower in fish species commonly consumed in Norway (e.g. farmed salmon, mackerel, herring) and in eggs, milk and meat sampled in Norway compared to the concentrations in similar products submitted by European countries. For some foods (e.g. meat) this conclusion is based on only a few samples and has high degree of uncertainty, whereas for fish, milk and eggs a larger number of samples form basis for the conclusion. Most of the data in the EFSA database (in total 20,273 samples) were submitted by Germany (6,550 samples), France (5,188 samples), and Norway (2,128 samples). The Norwegian samples, all on fish, constituted approximately 30% of the fish samples in the EFSA database. A reason for the generally lower concentration in Norwegian fish samples might be that the occurrence data submitted to EFSA included fish from the Baltic area. EFSA reported that higher levels of PCDD/Fs and DL-PCBs were found in samples of herring, salmon and trout of possible Baltic origin than in those of non-Baltic origin (EFSA, 2018a).

Reasons for lower occurrence levels in Norwegian eggs, meat and milk is not known. However, studies on PCDD/Fs and POPs in background soil and air indicate that there is a north -south gradient in level of contamination reflected by proximity to source regions (Halse et al., 2011; Hassanin et al., 2005; Meijer et al., 2003; Schuster et al., 2011; Wagrowski and Hites, 2000). There may be lower environmental contamination level in Norway than in central Europe due to lower degree of industrialization and population density, which is reflected in lower concentration in farm animals. Regarding feed for Norwegian livestock, there is a high share of Norwegian produced concentrates and roughage, and a high share of Norwegian raw materials in the feed production. According to Animalia (2020a), the percentage of Norwegian raw materials in the total feed (roughage and concentrates) was 82% for cattle in milk production, 86% for bovine meat production, 96% for sheep and lambs, 71% for pigs, 40% for chicken and 54% for chicken egg production. No occurrence data of PCDD/Fs and DL-PCBs in Norwegian feed material has been identified, however, lower levels of PCDD/Fs and POPs in background soil and air may also indicate lower levels in Norwegian produced concentrates and roughage than in feed produced in the central Europe.

The occurrence data available from Norway alone are not sufficient to form basis for exposure assessments covering the total diet, as data on important food groups are missing. When evaluating which occurrence data that should be used for exposure assessment, the results from food sampled in Norway and results from food sampled in the Europe as available in the EFSA database were compared, and also the degree of self-sufficiency in Norway was taken into consideration. The overall self-sufficiency is around 43% on energy basis but is higher for foods known to contribute substantially to PCDD/Fs and DL-PCB exposure. In 2018, self-sufficiency for fish was 80%, for meat 95%, egg 99%, milk, cream and sour cream 100%, yoghurt 87%, cheese 84% and butter 98%, whereas it was 20% for

margarine and other fat (NIBIO, 2021). For other foods generally not considered as major sources of PCDD/Fs and DL-PCBs, the degree of self-sufficiency was variable. For grains and flour the self-sufficiency was 21%, for potatoes 79%, vegetables 46%, and for fruits and berries 6%. Based on the high degree of self-sufficiency for important food groups, the Norwegian occurrence data are considered more relevant for the exposure assessment for these food groups than the occurrence data submitted to EFSA. For other food groups the concentrations in the data submitted to EFSA are equally relevant as the Norwegian ones.

The number of samples from Norway is low (except from fish) (Table 3.1-2) compared to the number of samples in the EFSA database. As the uncertainty in the results increase with a low number of samples, it was decided to perform two exposure assessments; one based on concentration data from EFSA only (termed EFSA dataset), and one based on Norwegian occurrence data for fish, meat, egg, and dairy products in combination with data from EFSA (2018a) for other foods (termed VKM dataset). This also provides an opportunity to compare the exposure based on Norwegian consumption data using the EFSA dataset to exposures estimates obtained by EFSA for numerous European dietary surveys.

VKM notes that the two different datasets have some values occurring in both datasets, since data on fish from Norway was reported to EFSA and was part of the EFSA database together with fish from other countries (EFSA, 2018a).

The occurrence data for fruits, vegetables and potatoes

VKM noted the detected PCDD/Fs and DL-PCBs levels in fruits, vegetables and potatoes in the EFSA database (Table 3.1-2). EFSA indicated that the uncertainty related to reported occurrence levels in samples of plant origin was high, due to the low number of samples in most categories. As PCDD/Fs and DL-PCBs are lipid soluble and accumulate in the food chain, whereas fruits, vegetables and potatoes generally have low fat content (0.1-0.4%) and are low in the food chain, the reported concentrations are hard to explain. One possibility could be that these fruits, vegetables and potatoes were contaminated locally, e.g., by remnants of contaminated earth or deposits from local air pollution (it is not known whether the samples were washed before analysis). For food groups consumed in high quantities (as measured in grams), even low concentrations will influence the total intake. To account for these uncertainties, VKM decided to calculate the exposure both with and without fruits, vegetables and potatoes.

3.1.2 Preparation of occurrence data for exposure assessment

The occurrence data from farm animals (except liver) were reported on a fat weight basis. The data on all other foods were reported per whole weight (ww) basis.

Occurrence data reported as fat weight

For each food item from farm animals (except liver) reported eaten in the dietary surveys, a PCDD/F and DL-PCB concentration value on whole weight basis was calculated from the concentration on fat weight basis. This was done based on concentration in the given food

per gram of fat (Table 3.1-2) and the fat percentage in the given food in the Norwegian food composition database and the dietary survey system KBS. The fat percentage in food used in the KBS can also be found in the Norwegian food composition database "Matvaretabellen" (www.matvaretabellen.no). For example, for the VKM dataset, the PCDD/F and DL-PCB concentration in milk fat was multiplied by the fat percentage in different cheeses to find the PCDD/F and DL-PCB concentrations in the cheeses on whole weight basis. The PCDD/F and DL-PCB concentrations in whole weight, derived from fat weight, were used in the exposure estimates. Whole weight concentrations in food items from farmed animals are not reported in this risk assessment.

Occurrence data reported as whole weight

In food groups for which the concentrations of PCDD/F and DL-PCB are given in whole weight (e.g., fish, grain, and grain products), the mean concentrations were used directly for exposure assessment.

Matching of FoodEx and KBS food names

In the present exposure assessment, the Norwegian food composition database and the dietary survey system KBS was used for the categorisation of consumption data from the dietary surveys. The KBS contains food composition databases, with descriptions of individual food items classified into food groups and broader food categories in a hierarchical food category system. The food categorisation system used by EFSA (FoodEx) were used in the EFSA opinion (2018a). Both KBS and FoodEx consists of descriptions of many individual food items aggregated into food groups and broader food categories in a hierarchical parent-child relationship. Matching of the KBS and the FoodEx food names was done by VKM based on food knowledge and expert judgement.

Grouping of foods into different levels in the food categorisation system

EFSA retained occurrence data at the most detailed classification level (FoodEx level 3) if more than six samples were available. If less than six samples were available, the occurrence data were compared to occurrence data for similar foods belonging to other categories at similar FoodEx level. If the occurrence levels were similar, these food categories were grouped together into a new category. If not, the samples reported at this FoodEx level could be merged with a less detailed FoodEx level, if the levels for the less detailed category was well-presented by the available categories. If the levels reported were very different from the broader food category, the samples were excluded. In this exposure assessment by VKM, the approach is different from EFSA. Food categories in the EFSA database with less than six occurrence data were included in the exposure estimation when there was high similarity between the food codes in the KBS and FoodEx. For example, in the FoodEx level 1 group "Grains and grain-based products", "Corn grain" (n=20) was the only FoodEx level 3 category with occurrence data for more than 6 samples. Instead of estimating exposure based on occurrence data in the less detailed (FoodEx level 1) category "Grains and grain-based products", VKM used the available occurrence data from the EFSA database at the more detailed level (FoodEx level 3) such as "Wheat bread and rolls" (n=2), "Muslie" (n=1), and "Pastries and cakes" (n=2).

Fruits, vegetables and potatoes

EFSA did not include the food group "Fruit and fruit products" in their exposure assessment due to few samples. VKM included occurrence data for the food items "Strawberries" (n=5), "Table grapes" (n=2), "Avocados" (n=1), "Peaches" (n=3), and "Apples" (n=3) that matched well between FoodEx and the KBS. Other fruits were not included in the exposure assessment by VKM due to lack of data.

The EFSA FoodEx level 1 food group "Vegetables and vegetable products" consisted of 40 different FoodEx level 3 food groups (Annex Table 2A (EFSA, 2018a)). To be consistent with the practice described above, "Brussels sprouts" (n=1), "Courgettes" (n=5), "Tomatoes" (n=2) are examples of vegetables with occurrence values at FoodEx level 3. Vegetables at FoodEx level 3 with particularly high occurrence levels, such as "Head cabbage" (n=3) and "Cucumbers" (n=1), were not used by the VKM (for further explanation, see Chapter 3.1.1). Instead, linking categories within the FoodEx level 1 occurrence data for "Vegetables and vegetable products" were used for exposure assessment, "Root vegetables" and "Fruiting vegetables", respectively.

The EFSA FoodEx level 1 food group "Starchy roots and tubers" consisted of 3 different FoodEx level 3 food groups. "Main-crop potatoes" (n=1) has an occurrence value at FoodEx level 3, which was used in the present assessment.

Fish, meat, egg and milk

For the food groups for fish, meat, eggs and milk, occurrence data for more than six samples were available in each case. The EFSA occurrence data were used in the present assessment, to the extent possible, identically to the occurrence data use in EFSA (2018a).

Composite dishes

Fish is eaten in many forms: raw, cooked, baked, and grilled, as a fillet or as an ingredient in composite fish dishes and products. An important share of the fish intake in Norway comes from composite fish dishes. As occurrence values for PCDD/Fs and DL-PCBs are available for raw fish fillets, to estimate exposure from fish intake, the amounts of fish in composite fish dishes and fish products were converted into raw fish-fillet equivalents. For details of the conversion procedure used for fish and other composite dishes, see Chapter 10.2.

The most common fish species used as ingredients in composite fish dishes were identified and used in the recipes and recalculations. Several fish dishes are frequently eaten and merit particular attention: fish cakes, fish pudding, fish balls, and mackerel in tomato sauce (the latter used as a bread spread). For fish cakes, fish pudding and fish balls, the main fish ingredients are Alaska pollock and smelt (Aakre et al., 2019). As there were no available occurrence data, the PCDD/F and DL-PCB values for haddock were used for both Alaska pollock and smelt. Intake of mackerel in tomato sauce was converted to raw mackerel fillet. Based on information from the manufacturers of mackerel in tomato sauce, autumn mackerel is the main type of mackerel used in this product. Therefore, the PCDD/F and DL-PCB values from autumn mackerel were used in the present assessment.

For roe-liver pâté 100 g of the composite dish was based on the information on the product label converted to 50 g of cod liver, 39 g of cod roe, and 11 g og other ingredients. The contribution from the other ingredients was not considered. For further details on recipe estimations see Chapter 10.2 (Appendix I).

3.2 The estimated exposure

As explained in Chapter 3.1.1, VKM decided to present exposure assessments based on two different occurrence datasets; the EFSA dataset (containing only occurrence data from European countries as reported by EFSA (2018a)) and the VKM dataset (Norwegian occurrence data for fish, meat, eggs, and dairy products combined with data from EFSA for other foods). As also explained in 3.1.1, the exposure was estimated both with and without fruits, vegetables and potatoes. Exposure estimates were done at both LB and UB for PCDD/Fs and for the sum of PCDD/Fs and DL-PCBs. This resulted in multiple different exposure calculations for each dietary survey, as illustrated in Table 3.2-1.

Table 3.2-1. The different exposure calculations.

	PCDD/Fs and DL-PCBs	PCDD/Fs
EFSA dataset with fruit and vegetables	UB and LB	UB and LB
EFSA dataset without fruit and vegetables	UB and LB	UB and LB
VKM dataset with fruit and vegetables	UB and LB	UB and LB
VKM dataset without fruit and vegetables	UB and LB	UB and LB

The estimated exposure, obtained using the MM approach for the age groups 4-, 9- and 13-year-olds and adults, and the W-OIM approach for 1-and 2-year-olds, are presented in Chapter 3.2.1. The contribution from different food groups to the mean total PCDD/F and DL-PCB exposure was estimated from the mean exposures obtained using the OIM approach (Chapter 3.3).

3.2.1 The estimated exposure from the total diet

The estimated mean and 95th percentile exposure excluding fruits, vegetables and potatoes, for the VKM and the EFSA datasets, is given in Table 3.2.1-1 and 3.2.1-2, respectively. The estimated mean and 95th percentile exposure including fruits, vegetables and potatoes, for the VKM and the EFSA datasets, is given in Table 12-1 and 12-2.

An overview of the mean estimated exposure to the sum of PCDD/Fs and DL-PCBs for all age groups, with and without fruits, vegetables and potatoes, is shown in Figure 3.2.1-1. In addition to the exposure for adults aged 18-70 years, the exposure for women aged 18-45 year is shown to indicate exposure in women of childbearing age. Calculated exposures at different percentiles, with and without fruits, vegetables and potatoes, is given in Appendix III (Chapter 12). The results in figure 3.2.1-1 illustrate that fruit and vegetables seems to be a major contributor to the total exposure. This is particularly visible at the UB exposures in children in all age groups. Noting the high uncertainty in the occurrence data for fruits, vegetables and potatoes (see 3.1.1), including the low fat content in addition to being low in

the food chain, making these foods unlikely to be major contributors, VKM decided to present exposures including fruits, vegetables and potatoes in Appendix III (Chapter 12) and to present data without fruits, vegetables and potatoes in the main body of the text.

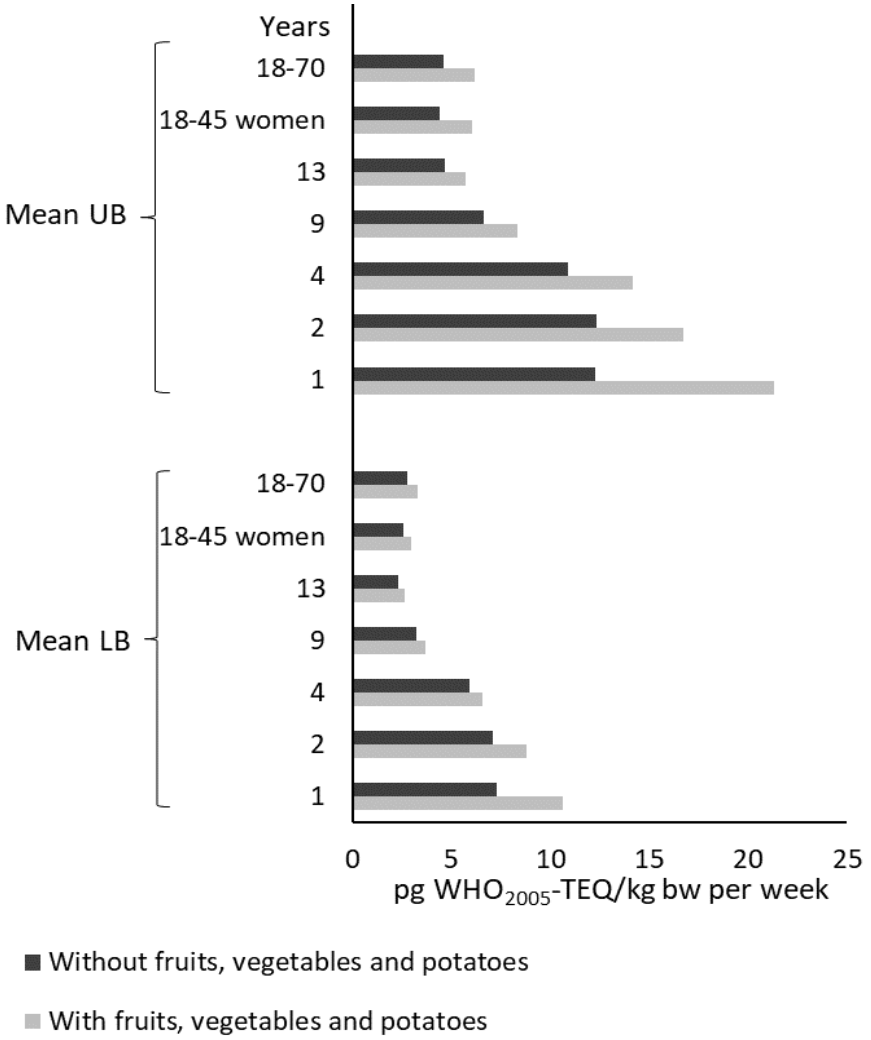

Figure 3.2.1-1. The estimated exposure to the sum of PCDD/Fs and DL-PCBs, with and without fruits, vegetables and potatoes and for different age groups, using the VKM dataset (Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods). The weighted observed individual means approach was applied for 1-and 2-year-olds, the mixed model approach was applied for the other age groups.

The LB mean estimated exposures using the EFSA dataset are 2.0 to 2.8-times higher than the results obtained using the VKM dataset. The EFSA dataset UB mean estimated exposures are 1.5 to 1.8-times higher than the results obtained using the VKM dataset. The EFSA dataset LB P95 estimated exposures are 2.1 to 2.8-times higher than the results obtained using the VKM dataset, whereas the UB P95 estimated exposures are 1.4 to 1.8-times higher than the results obtained using the VKM dataset.

Women aged 18-45 years and 12-13-year-olds have a lower exposure than adults aged 18-70 years, whereas 1-, 2- and 4-year-olds have higher exposure than adults aged 18-70 years.

The mean UB/LB ratio for the sum of exposure to PCDD/Fs and DL-PCBs was 1.2 to 1.3 in different age groups using the EFSA dataset and 1.7 to 2.1 using the VKM dataset. This reflects a slightly higher uncertainty in the Norwegian data. However, a higher UB/LB ratio can also be expected when the concentrations are lower in food groups contributing to the exposure (e.g. milk and meat). The exposure estimates based on the VKM dataset show that there is higher difference between UB and LB for the sum of PCDD/Fs than for DL-PCBs, which can be explained by higher detection rate for some of the DL-PCBs than for PCDD/Fs.

Table 3.2.1-1. Estimated exposure to PCDD/Fs and the sum of PCDD/Fs and DL-PCBs (pg WHO_{2005}-TEQ/kg bw/week), without fruits, vegetables and potatoes, obtained using the VKM dataset. The mixed model approach is applied for all population groups, except for 1-and 2-year-olds, for which weighted observed individual means are shown.

Age group	Sum of PCDD/Fs and DL-PCBs				PCDD/Fs			
	Mean		95-percentile		Mean		95-percentile	
	LB	UB	LB	UB	LB	UB	LB	UB
Adults (18-70 years)	2.78	4.62	5.17	7.88	1.02	2.61	1.71	4.26
18-45 years (women)	2.54	4.40	4.75	7.47	0.97	2.55	1.69	4.15
13-year-olds	2.32	4.67	4.28	8.52	1.04	2.98	1.79	5.28
9-year-olds	3.24	6.61	5.58	10.8	1.46	4.24	2.39	6.74
4-year-olds	5.90	10.9	9.88	16.3	2.33	6.67	3.55	9.53
2-year-olds	7.11	12.3	14.5	22.4	2.60	7.24	4.76	12.5
1-year-olds	7.25	12.2	15.8	24.2	2.56	7.06	5.13	13.2

Table 3.2.1-2. Estimated exposure to PCDD/Fs and the sum of PCDD/Fs and DL-PCBs (pg WHO_{2005}-TEQ/kg bw/week), without fruits, vegetables and potatoes, obtained using the EFSA dataset. The mixed model approach is applied for all population groups, except for 1-and 2-year-olds, for which weighted observed individual means are shown.

Age group	Sum of PCDD/Fs and DL-PCBs				PCDD/Fs			
	Mean		95-percentile		Mean		95-percentile	
	LB	UB	LB	UB	LB	UB	LB	UB
Adults (18-70 years)	6.49	7.97	11.5	13.7	2.28	3.37	3.89	5.53
18-45 years (women)	5.77	7.19	10.3	12.5	2.05	3.14	3.57	5.21
13-year-olds	6.59	8.51	11.7	14.8	2.28	3.79	3.96	6.54
9-year-olds	9.09	11.9	14.8	18.8	3.23	5.40	5.18	8.48
4-year-olds	15.1	19.2	23.8	28.8	5.15	8.39	7.58	11.9
2-year-olds	17.8	21.9	34.0	40.0	6.03	9.36	10.8	16.1
1-year-olds	16.0	20.3	33.5	40.7	5.27	8.77	10.4	16.2

3.2.2 Contribution of the individual congeners and congener families to the total dietary exposure

The contribution of the 17 PCDD/Fs and the 12 DL-PCBs to the total estimated mean exposure without fruits, vegetables and potatoes, using the VKM dataset for all age groups, is shown in Figure 3.2.2-1. DL-PCBs contributed 61 to 65% of the total TEQ from PCDD/Fs and DL-PCBs at the LB and 36 to 44 % at the UB. This is in line with findings in EFSA (2018a), in which it was reported that 63.2% of the LB mean exposure in adults came from

DL-PCBs. According to EFSA 2018, the mean contribution from PCB-126 was about 55% of the total LB estimated exposure and thus 87% of the exposure to DL-PCBs (in TEQ) (EFSA 2018a).

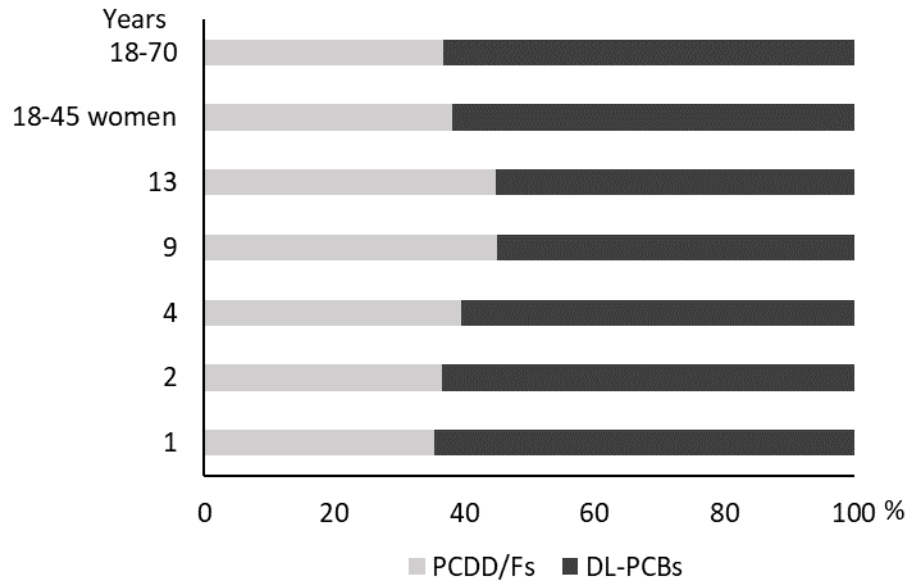

Figure 3.2.2-1. The contribution of PCDD/Fs and DL-PCBs to the mean estimated exposure without fruits, vegetables and potatoes, using the VKM dataset. The mixed model approach is applied for all population groups, except for 1-and 2-year-olds, for which weighted observed individual means are shown.

3.3 Contribution from food groups to total PCDD/F and DL-PCB exposure

The contribution of different food groups to the total PCDD/F and DL-PCB exposure (29 congeners) was estimated from the mean exposure obtained using the OIM approach and the VKM dataset. The contribution is shown for all foods grouped into fish (lean and fatty fish, including fish liver and roe), shellfish (crustaceans and bivalves), meat (farm animals), dairy (milk and dairy products), egg, grain (grain and grain products), other, and marine oil supplements. The category "Other" is comprised of composite foods that are not assigned any other category in the KBS, food oils (except for marine oils and butter), drinks, sweets, spices, and food for infants and young children.

The contribution from different food groups in pg TEQ/kg bw per week is illustrated in Figure 3.3-1 (LB). The numerical values are available in Appendix III (Chapter 12.4). The contribution in percent is shown in Figures 3.3-2 (LB) and 3.3-3 (UB) and Table 3.3-1.

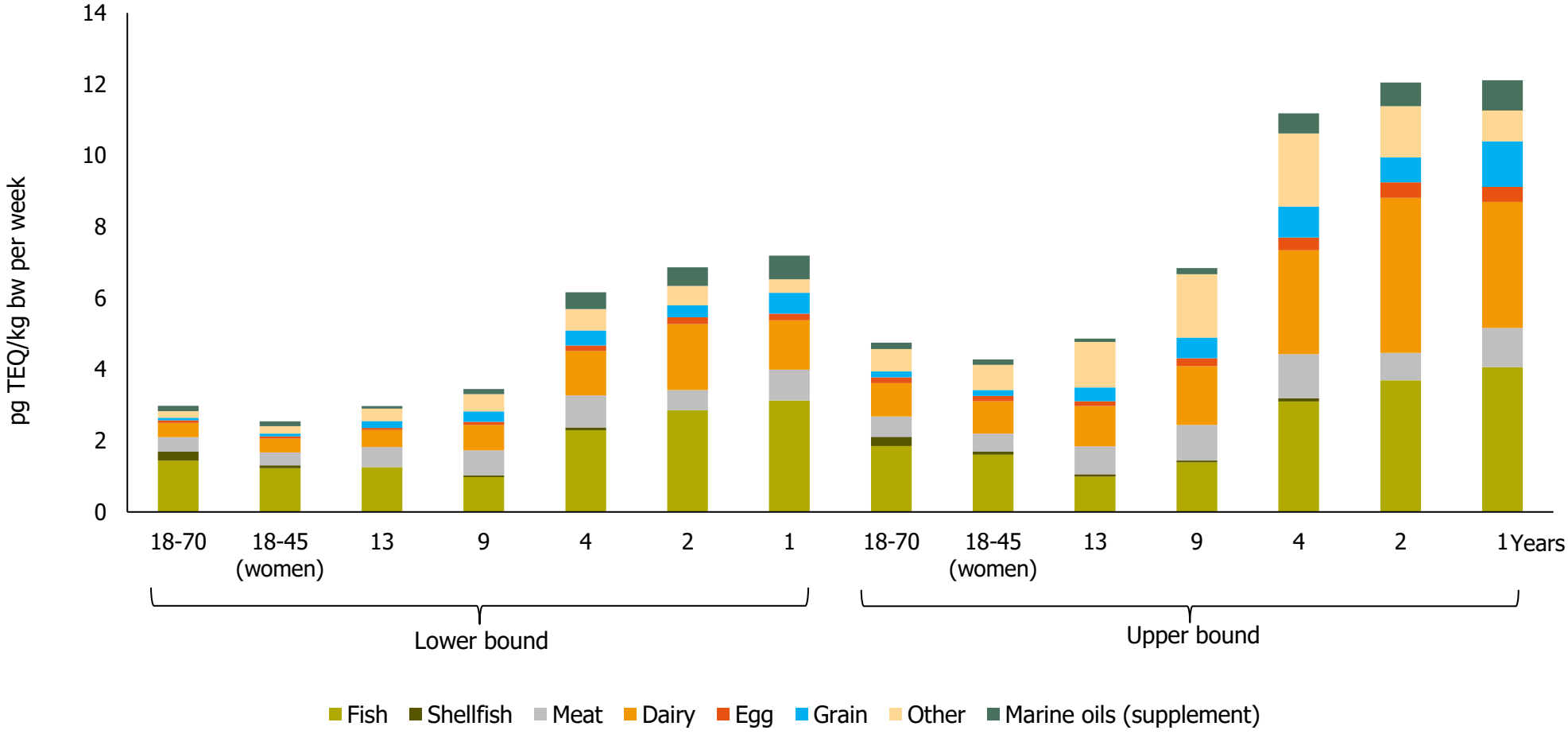

Figure 3.3-1. Contribution in pg WHO_{2005}-TEQ/kg bw per week of different food groups to mean total exposure to PCDD/Fs and DL-PCBs for different age groups, based on observed individual means and the VKM dataset.

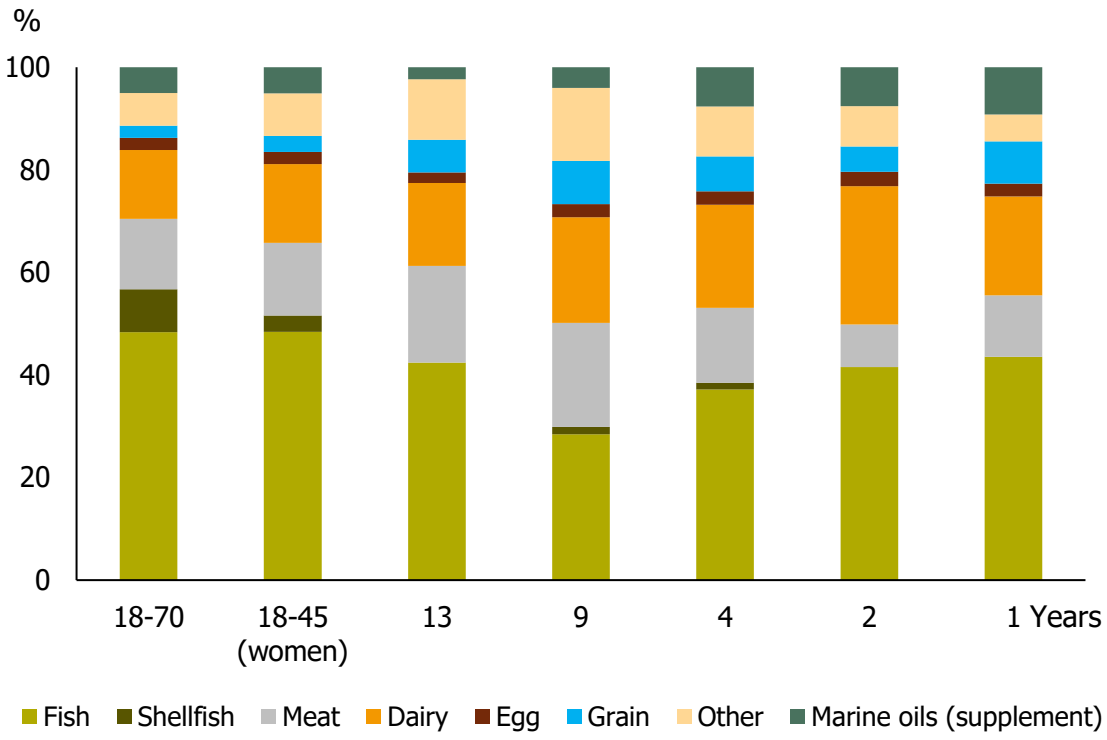

Figure 3.3-2. Contribution in percent (lower bound) of different food groups to mean total to PCDD/Fs and DL-PCBs, based on observed individual means and the VKM dataset.

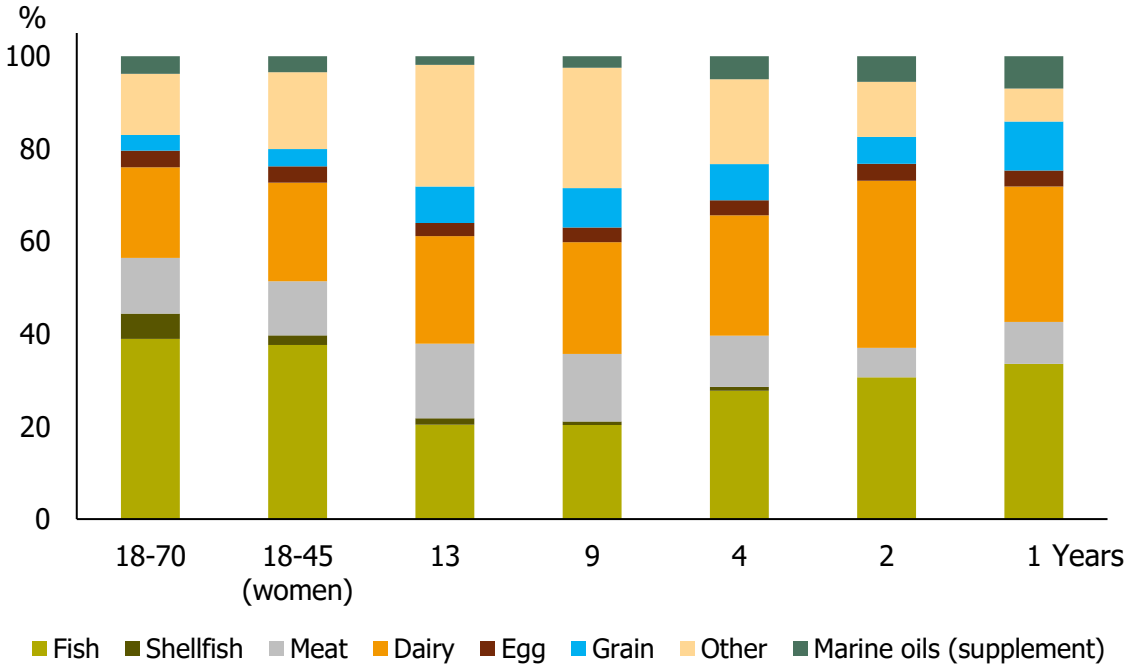

Figure 3.3-3. Contribution in percent (upper bound) of different food groups to mean total exposure to PCDD/Fs and DL-PCBs, based on observed individual means and the VKM dataset.

Table 3.3-1. The contribution of food groups, in percent (rounded), to the mean total lower bound and upper bound PCDD/F and DL-PCB exposure.

	Adults (18-70 years)		Women (18-45 years)		13-year-olds		9-year-olds		4-year-olds		2-year-olds		1-year-olds	
	LB	UB	LB	UB	LB	UB	LB	UB	LB	UB	LB	UB	LB	UB
Fish	48	39	49	38	28	20	29	20	37	28	40	30	43	33
Shellfish	8.4	5.5	3.3	2.1	2.7	1.4	1.4	0.7	1.4	0.8	0	0	0	0
Meat	14	12	14	12	23	16	20	15	15	11	8.1	6.2	12	9.1
Dairy	13	20	15	21	19	23	21	24	20	26	26	35	19	29
Egg	2.4	3.5	2.5	3.5	2.4	2.9	2.7	3.2	2.5	3.2	2.7	3.6	2.5	3.5
Grain	2.5	3.3	3.0	3.8	7.5	7.8	8.3	8.5	6.9	7.8	4.8	5.7	8.2	11
Other	6.3	13	8.1	17	14	26	14	26	9.7	18	7.5	12	5.2	7.1
Marine oils (supplement)	5.0	3.8	5.1	3.5	2.8	1.8	4.0	2.5	7.6	5.0	11	7.6	9.8	7.4

At the LB exposure, fish, meat and dairy products were the main contributors in all age groups. At the UB exposure, also foods grouped into "other" were important contributors in particular for children and adolescents in the age groups 2-13 years of age.

The contribution to the mean total PCDD/F and DL-PCB exposure for fish and shellfish was estimated from the mean exposure obtained using the OIM approach and the VKM dataset. The seafood was grouped into lean fish (all), salmon and trout, mackerel, other fatty fish (halibut, herring, and char), roe and liver, and shellfish. Contribution in pg WHO_{2005}-TEQ/kg bw per week is shown in Figure 3.3-5 (LB) and in Appendix III (Chapter 12.4). Contribution in percent is shown in Figure 3.3-6 (LB) and Table 3.3-3 (LB).

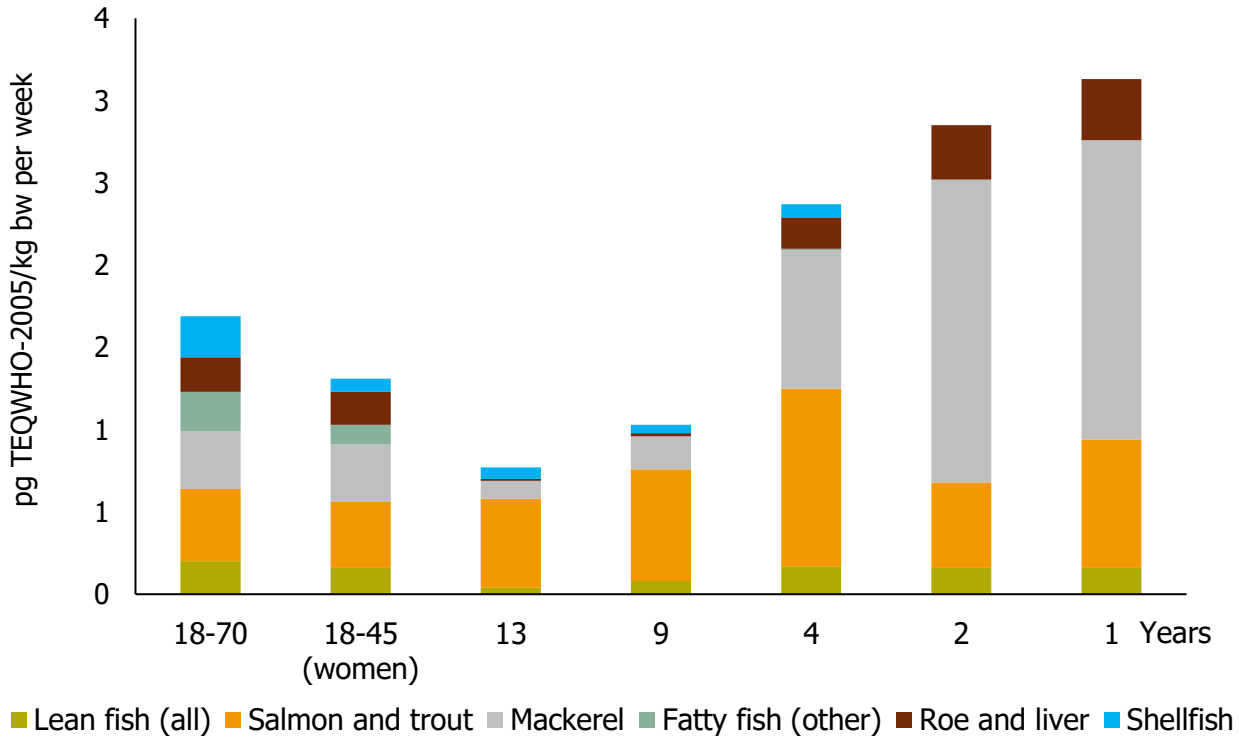

Figure 3.3-5. Contribution in pg WHO_{2005}-TEQ/kg bw per week (lower bound) to mean total to PCDD/F and DL-PCB exposure from fish and shellfish, based on observed individual means and the VKM dataset. Fatty fish (other) includes halibut, herring, and char.

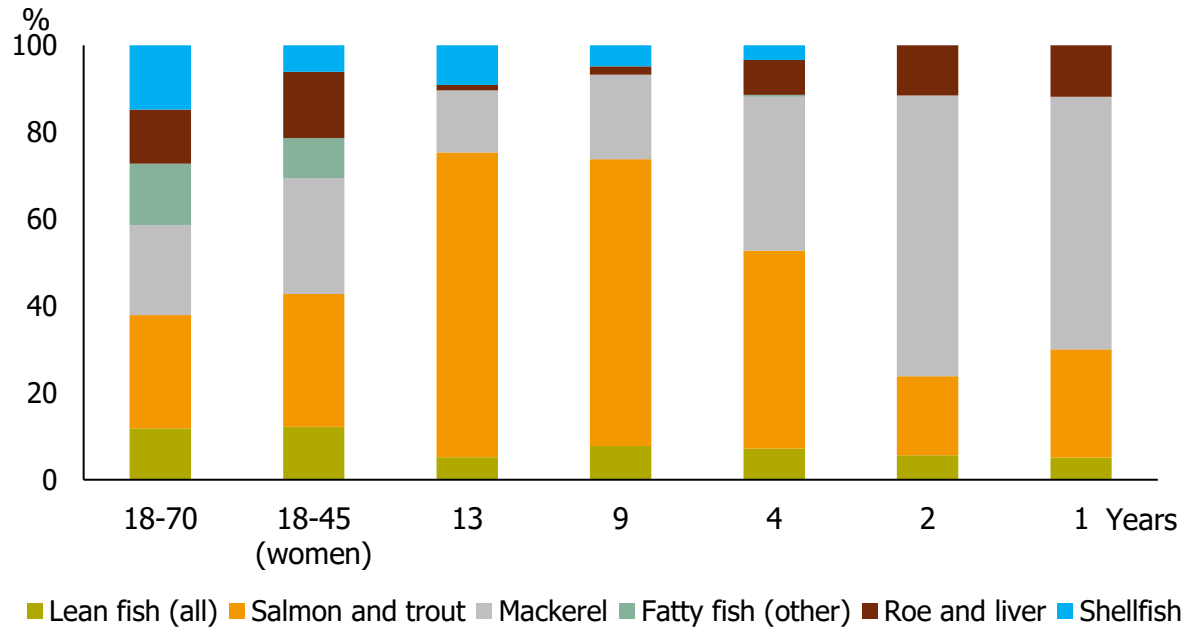

Figure 3.3-6. Contribution in percent (lower bound) to mean total to PCDD/F and DL-PCB exposure, based on observed individual means and the VKM dataset. Fatty fish (other) includes halibut, herring, and char.

Table 3.3-3. Contribution in percent to total lower bound PCDD/F and DL-PCB exposure from fish and shellfish. Fatty fish (other) includes halibut, herring, and char.

	Adults (18-70 years)	Women (18-45 years)	13-year-olds	9-year-olds	4-year-olds	2-year-olds	1-year-olds
Lean fish	12	12	5.1	8.1	7.2	5.8	5.1
Salmon	26	30	70	66	46	18	25
Mackerel	21	27	14	19	35	65	58
Other fatty fish	14	9.0	0.0	0.0	0.5	0.0	0.0
Roe and liver	13	16	1.4	2.1	7.8	12	12
Shellfish	15	6.3	8.7	4.6	3.5	0.0	0.0

Fatty fish species had the highest contribution in all age groups. The fatty fish species salmon and trout and mackerel contribute approximately equally in adults, and the

contribution from other fatty fish is also substantial. In the youngest children (1- and 2-year-olds), mackerel contributes the most.

In both adults and children, fish liver and roe contribute substantially to the mean exposure. The number of respondents reporting consumption of fish liver or fish-liver containing products (cod roe-liver pâté) was low for all age groups. The number of fish liver consumers among 1-, 2-, 4-, 9-, and 13-year-olds was 18, 9, 1, 0, and 0, respectively. Only 21 adults reported consuming fish liver, of whom 4 were women aged 18-45. On the other hand, a substantial part of the participants in the dietary surveys consumed cod roe in the form of the bread spread "Kaviar". As example, consumption of "Kaviar" was recorded in 45% of 2-year-olds and 31% of 4-year-olds. Given the high concentration of PCDD/F and DL-PCB in fish liver, even a small number of consumers adds up to a large relative contribution, as shown in Table 3.3-4.

Analysis of the exposure in the 10% with the highest estimated exposures confirmed that fish and shellfish, and in particular fish liver is a major contributor to the exposure (Appendix III, Chapter 12.4.7).

The exposure from fish liver and products containing liver is further addressed by scenario calculations in 3.5.2.

Table 3.3-4. Percent contribution from fish liver and roe to total lower bound PCDD/F and DL-PCB exposure from fish liver and roe.

	Adults (18-70 years)	Women (18-45 years)	13-year-olds	9-year-olds	4-year-olds	2-year-olds	1-year-olds
Liver	82	93	0	0	11	40	66
Roe	18	7	100	100	89	60	35

3.4 Comparisons of exposure in Norwegian surveys with results from surveys from other European countries included by EFSA (2018a)

The approach used by EFSA for chronic exposure assessment for all dietary surveys from European populations available to EFSA is similar to the OIM approach used in the present risk assessment. EFSA (2018a) included 17 European surveys for adults (≥18 to < 65 years old). The results for adults in EFSA (2018a) are summarized in Table 3.4-1 together with the results for adults (18-70 years) in Norkost 3 from the present assessment using the EFSA dataset and the VKM dataset. Estimated exposure both including and excluding fruits, vegetables and potatoes are shown for adults in Norkost 3. The food group "Fruit and fruit products" was not included in the EFSA exposure assessment. Since the summary data from EFSA cover quite wide ranges of age groups in children (≥ 36 months to < 10 years old) and adolescents (≥ 10 years to < 18 years old), comparison of exposures in these age groups was not considered meaningful by VKM. Using the EFSA dataset and the VKM dataset, the estimated exposures without fruits, vegetables and potatoes are between minimum and maximum exposures reported by EFSA for the included surveys. When fruits, vegetables and

potatoes are included, the exposures based on the VKM dataset are within the EFSA range, whereas using the EFSA dataset leads to mean and P95 UB exposures that are slightly higher than the highest exposure reported by EFSA across European dietary surveys. The differences can be explained by a high fish consumption in Norway, and that, as explained in 3.1.1, in particular the concentrations in fish in the EFSA database are not representative of the concentration in Norwegian fish.

Overall, the results indicate that the exposure estimates done by EFSA and by VKM are quite similar.

Table 3.4-1. Minimum, median and maximum mean and 95th-percentile exposure to PCDD/Fs and DL-PCBs in adults (≥18 to < 65 years old) in 17 dietary surveys in adults (≥18 to < 65 years old) from Europe (EFSA, 2018a), and mean and 95th-percentile exposure in adults in Norkost 3 (18 – 70 years old) based on the EFSA dataset and the VKM dataset (pg WHO_{2005}-TEQ/kg bw per week)

	Dietary surveys in EFSA (2018a)						**Norkost 3 OIM**			
	Min LB	Med LB	Max LB	Min UB	Med UB	Max UB	VKM LB	VKM UB	EFSA LB	EFSA UB
Adults mean	2.9	4.5	7.8	3.4	5.3	9.1	3.0 *3.4**	4.8 *6.3**	6.7 *7.0**	8.0 *9.5**
Adults P95	6.6	13.6	20.1	8.3	14.6	21.8	7.1 *10.3**	10.0 *15.3**	17.3 *19.9**	19.5 *24.1**

*Intakes including fruits, vegetables and potatoes.

3.5 Estimated exposure to PCDD/Fs and DL-PCBs based on scenarios for consumption of selected food items

The Norwegian national dietary surveys Norkost 3, Ungkost 3, Spedkost 3 and Småbarnskost 3 do not supply sufficient information about consumption of rarely consumed food, due to the assessment methods used. Therefore, to estimate exposure to PCDD/Fs and DL-PCBs from liver from livestock animals, liver from fish, brown crab meat, reindeer meat and fat, consumption scenarios were used. In addition, consumption scenarios for marine oil supplements and liver pâté were used. The scenarios were designed as portions and indicate the contribution of one portion of these foods to the PCDD/Fs and DL-PCB exposure. Varying with the type of food in the scenarios, the exposure was calculated by a daily (marine oil supplement) or weekly portion (liver from livestock animals, liver from fish, brown crab meat, reindeer meat and fat). Where appropriate, different scenarios were used for different age groups. To calculate the exposure on a body weight basis, mean body weights from the national dietary surveys were used: adults (Norkost 3): 77.7 kg; 13-year-olds (Ungkost 3): 50.3 kg; 9-year-olds (Ungkost 3): 39.2 kg; 4-year-olds (Ungkost 3): 17.5 kg; 2-year-olds (Småbarnkost 3): 12.8 kg; 1-year-olds (Spedkost 3): 10.0 kg.

The Norwegian Food Safety Authority (NFSA) has issued warnings for certain foods containing high levels of PCDD/Fs and DL-PCBs for certain population groups, e.g. they state

that children, pregnant and breastfeeding women, and women of childbearing age should not eat brown crab meat. When such warning is issued, no scenarios are created for these age groups. An overview of relevant warnings is given in the interpretations of the terms of reference.

3.5.1 Crabs (brown and white meat)

The autumn is the main crab-eating season for private catchers, whereas commercially filled crab shells are available the whole year. The edible part of crabs (*Cancer pagurus*) includes both brown and white meat. The white meat is found in the claws, legs and shoulder (in Norwegian "støet"), whereas the brown meat is found in the carapace. All brown meat is edible except the crab stomach (in Norwegian "paven") which is removed prior to consumption, but after cooking. The organ that functions both as liver and pancreas in the crab, the hepatopancreas, is present in the crab brown meat after cooking.

The relative amount of white and brown meat is different in whole crab and commercially available filled crab shells. It is considered that commercially available filled crab shells contain 33% brown meat and 67% white meat, whereas whole crabs contain 67% brown meat and 33% white meat. The composition of brown and white crab meat is based on information in the previous VKM risk assessment (2010) and information from a producer of filled crab shells (HitraMat A/S (2022), personal communication).

In Norkost 3, 15 out of 1787 participants reported eating crab on one or both consumption days and the intakes were between 17.5 and 150 gram per day (Totland et al., 2012). In the food propensity questionnaire, 44% reported eating crab/lobster one to five times per year, and 13 persons reported eating crab/lobster 4 times or more per month.

For the crab scenarios, it is assumed that both adolescents and adults consume one whole crab or one filled crab shell as a portion, and 150 g is used as portion size for both. This is based on the previous VKM risk assessment (2010), where the edible meat in one whole crab was considered to be 160 g and the edible meat in one filled crab shell was 150 g.

Concentration data for PCDD/Fs and DL-PCBs were only available for brown crab meat. It is known from previous analyses that white crab meat contains substantially lower PCDD/F and DL-PCB concentrations than brown meat, which can be explained by lower fat content in addition to absence of specific PCDD/F binding proteins that are present in the crab hepatopancreas (which is present in brown meat). To obtain concentrations for PCDD/Fs and DL-PCBs for whole crab and for filled crab shells, VKM estimated the concentration in white crab meat based on the concentration in brown crab meat and the brown meat/white meat concentration ratio of 12 from a previous VKM risk assessment (2010). It should be noted that the data from the previous risk assessment were calculated in TEQ based on WHO-TEF from 1998, and that for some congeners these TEF factors were higher than the TEF factors from 2005 (Van den Berg et al., 2006). The PCDD/F and DL-PCB concentrations used in the

scenarios for whole crab and filled crab shells are shown in Table 3.5.1-1. A detailed overview of the estimation of these concentrations is available in Appendix II (Chapter 11.1).

Table 3.5.1-1. The estimated PCDD/Fs and DL-PCBs concentrations used in the scenarios for exposure from consumption of crab.

Food	Sum of PCDD/Fs and DL-PCBs (pg WHO_{2005}-TEQ/g whole weight)	
	Mean LB	Mean UB
Crab, brown meat	3.47	3.62
Crab white meat (estimated)	0.289	0.301
Whole crab (67% brown meat, 33% white meat) (estimated)	2.42	2.52
Filled crab shell (33% brown meat, 67% white meat) (estimated)	1.33	1.39

The exposure was calculated based on the suggested portion sizes and the estimated concentrations (Table 3.5.1-1), and the results are shown in Table 3.5.1-2. Children, pregnant women, breastfeeding mothers, and women of childbearing age are not included in the scenario as a warning not to eat brown crab meat due to the content of PCDD/Fs and DL-PCBs is given for these groups by the NFSA.

Table 3.5.1-2. The estimated exposure to PCDD/Fs and DL-PCBs from consumption of whole crab and filled crab shell (pg WHO_{2005}-TEQ/kg bw).

Age group	Whole crab, one portion (á 150 g)		Filled crab shell, one portion (á 150 g)	
	LB	UB	LB	UB
18-70-year-olds	4.7	4.9	2.6	2.7
13-year-olds	7.2	7.5	4.0	4.2

3.5.2 Fish liver

Fish liver is part of traditional fish meals made of cod or saithe. The traditional meal "mølje" consists of cooked cod, liver and roe, and the season for this meal is February to April. Fish liver can also be used as a side dish to cooked saithe, and the season for this dish is June to August. There are regional variations in fish liver consumption, and fish liver may also be consumed outside the abovementioned seasons. Fish liver is also consumed in the form of cod roe-liver pâté (in Norwegian: Rognleverpostei), which may be used as sandwich spread. The proportion of cod liver in cod roe-liver pâté is approximately 25%, whereas cod roe constitutes approximately 38%.

In Norkost 3 (n=1787), three persons reported eating fish liver and the intakes were between 20 and 39 grams (Totland et al., 2012).

In the food propensity questionnaire in Norkost 3, 295 persons (20%) reported eating fish liver one to five times per year, 31 persons (2%) reported eating fish liver 6-11 times per year, 22 persons (2%) reported eating fish liver once per month, seven persons reported eating fish liver 2-3 times per month, and one person reported eating fish liver 4-5 times per month.

In Norkost 3, cod roe-liver pâté was reported eaten by 18 persons (1%). In the food propensity questionnaire, 10% reported eating such pâté at least once a month. In children aged 4 years, one out of 399 participants reported liver consumption and none reported cod roe-liver pâté consumption. In children aged 2 years, none reported fish liver consumption and 9 out of 1413 participants reported cod roe-liver pâté consumption.

As the serving size for cod liver is highly variable, VKM decided to use tablespoons of about 15 g as a size measure, where a small portion may correspond to approximately two tablespoons (30 g) and a large portion may correspond to six tablespoons (90 g).

The portion size used for cod roe-liver pâté is similar to the estimated portion size for liver pâté which is 20 g per slice of bread, according to the Norwegian "weights, measures and portion sizes for foods table" (Dalane et al., 2015).

The mean LB/UB concentration of PCDD/Fs and DL-PCBs in cod liver and in cod roe-liver pâté presented in Chapter 3.1 were used in the scenarios (Table 3.5.2-1).

Table 3.5.2-1. The PCDD/Fs and DL-PCBs concentrations used in the scenarios for exposure from consumption of cod liver and cod roe-liver pâté.

Food	Sum of PCDD/Fs and DL-PCBs (pg WHO_{2005}-TEQ/g whole weight)	
	Mean LB	Mean UB
Cod liver	15	16
Cod roe-liver pâté	3.0	3.1

The exposure was calculated based on the suggested portion sizes and the estimated concentrations (Table 3.5.2-1), and the results are shown in Table 3.5.2-2. Children, pregnant women, and breastfeeding mothers are not included in the scenario as a warning not to eat fish liver and cod roe-liver pâté due to the content of PCDD/Fs and DL-PCBs is given for these groups by the NFSA.

Table 3.5.2-2. Cod liver and cod roe-liver pâté; estimated exposure to PCDD/Fs and DL-PCBs (pg WHO_{2005}-TEQ/kg bw).

Age group	Cod liver, small portion (two tablespoons á 15 g)		Cod liver, large portion (six tablespoons á 15 g)		Cod roe-liver pâté, one portion (á 20 g)	
	LB	UB	LB	UB	LB	UB
18-70-year-olds	5.9	6.2	17.6	18.6	0.8	0.8
13-year-olds	9.0	9.6	27.1	28.8	1.2	1.2

3.5.3 Marine oils

According to Spedkost 3, 31% of 1-year-olds consumed cod liver oil as a supplement, and the average intake among the users was 4 ml. For 2-years-olds, 30% consumed cod liver oil, and the average intake among the users was 4.2 ml (Småbarnskost 3). Spedkost 3 and Småbarnskost 3 have no information on consumption of fish oil. According to Ungkost 3, 28% of 4-year-olds consumed cod liver oil and 8% consumed an omega 3 supplement at least one of the 3-4 food registration days. Among 9-year-olds and 13-year-olds, 20% and 17%, respectively, reported consumption of cod liver oil, and 8% and 11 %, respectively, reported consumption of omega-3 supplements. For adults, 47% men and 58% women reported consumption of dietary supplements with marine oils or containing long chain n-3 fatty acids (Norkost 3). Supplements with cod liver oil were most commonly used.

There are several fish oil and cod liver oil products on the Norwegian market. In addition, other marine oils are available, which include oils produced from whale, seal, sharks, algae, krill and Calanus (Nilsen and Måge, 2014; Nilsen and Måge, 2015; Nilsen and Måge, 2016; Nilsen and Måge, 2017; Nilsen and Sanden, 2018; Nilsen and Sanden, 2019). The reported concentrations in these marine oils are highly variable, the sum of PCDD/Fs and DL-PCBs range from 0.13 to 49 pg TEQ/g fat (upper bound) as illustrated in Table 3.5.3-1.

Table 3.5.3-1. The PCDD/Fs and DL-PCBs concentrations (pg TEQ/g fat, UB) in different marine oil categories (based on Nilsen and Måge (2014); Nilsen and Måge (2015); Nilsen and Måge (2016); Nilsen and Måge (2017); Nilsen and Sanden (2018); Nilsen and Sanden (2019).

Product	n	mean	min	max
Cod liver oil	12	1.1	0.15	5.4
Fish oil[a]	15	1.4	0.24	4.0
Shark or ratfish oil[b]	10	11	1.1	49
Algal oil	3	0.51	0.26	1.0
Krill or calanus oil	4	0.78	0.32	1.2
Seal oil	7	1.2	0.32	4.6
Whale oil	1	17	na	na
Mixed fish and vegetable oil	8	0.33	0.19	0.46

[a] Anchovy, sardines, salmon, tobis/sandeel, herring.
[b] Includes oils from liver and meat.
na: Not available.

The marine oil supplements are available both in liquid form and as capsules, and the recommended doses of marine oils vary between products. The most common dose of cod liver oil supplements is 5 mL, which is used in the scenarios for cod liver oil. The same amount was used for other marine oils. The density of marine oil (0.92 kg/m^3) was not taken into consideration in the calculation. This can lead to a small overestimation of the exposure from marine oils, but the uncertainty is small compared with the uncertainty and variability in volume that is consumed as one portion of marine oil.

VKM calculated scenario exposure to cod liver oil using the mean concentration from 12 cod liver oils of 1.1 pg TEQ/g fat (UB) for the sum of PCDD/Fs and DL-PCBs. In addition, VKM calculated the exposure from marine oil with concentration equal to the maximum level (ML; 4.0 pg TEQ/g fat) for marine oils in Norway. The exposure was estimated based on 5 mL oil per day regardless og age, since 5 mL is recommended above age 1 year on the product label. The results are shown in Table 3.5.3-2. VKM notes that cod liver oil is not recommended as vitamin D supplement for infants younger than one year of age.

Table 3.5.3-2. Marine oils by daily use; estimated exposure to PCDD/Fs and DL-PCBs (pg WHO_{2005}-TEQ/kg bw per week).

Age group	Cod liver oil, one daily portion (5 mL) containing 1.1 pg WHO_{2005}-TEQ/g fat (UB)	Marine oil, one daily portion (5 mL) containing 4 pg WHO_{2005}-TEQ /g fat (UB)
18-70-year-olds	0.50	1.8
13-year-olds	0.77	2.9
9-year-olds	0.98	3.6
4-year-olds	2.2	8.0
2-year-olds	3.0	11
1-year-olds	3.9	14

3.5.4 Liver from livestock animals

Liver from most livestock animals is commercially available. Whereas beef and pork liver is available in grocery stores and in selected butcher shops, liver from sheep is of limited availability and can usually only be purchased during the slaughter season.

According to the national dietary survey Norkost 3, adult consumption of offal (blood, liver and kidney) from livestock animals and game is low. For men, the mean consumption is 1 g/day, for women the mean consumption is <0.1 g/day.

In the food propensity questionnaire in Norkost 3, 2.3% reported eating beef liver more than 6 times per year, and 4 persons (0.3%) reported eating beef liver once per week. In Ungkost 3, only one person reported eating liver.

As liver pâté is frequently consumed, exposure to PCDD/Fs and DL-PCBs from liver pâté is included in the exposure estimates in Chapter 3.3. A mean daily intake of 11 g liver pâté for 1 year old children was reported in Spedkost 3. As liver pâté is frequently eaten by the youngest children, portion scenarios for intake are included for 1-, 2-, and 4-year-old children in addition to scenarios for exposure from beef and pork liver consumption for all age groups.

An estimated portion size for liver is not given in the Norwegian "weights, measures and portion sizes for foods table" (Dalane et al., 2015). The portion size for meat is 150 to 200 g. VKM decided to use 175 g, the mean value for the meat portion sizes, in the portion scenarios for consumption of liver from livestock animal for both adolescents and adults, and 80 g per portion for children and toddlers (< 10 year).

According to the Norwegian "weights, measures and portion sizes for foods table" (Dalane et al., 2015), the estimated portion size for liver pâté is 20 g per slice of bread.

No Norwegian occurrence data for liver were identified. Based on the limited occurrence data for Norwegian meat, it seems that mean PCDD/Fs and DL-PCB concentrations are lower than the mean values used by EFSA (2018a). Consequently, using EFSA concentration data for Norwegian beef and pork liver would represent an overestimate. In lack of occurrence data, VKM used the ratio between liver and meat based on EFSA's concentration data (2018a) to estimate PCDD/Fs and DL-PCB concentrations for Norwegian beef and pork liver based on the concentration in kidney tallow from Norwegian beef and pork. The PCDD/F and DL-PCB concentrations used in the scenarios for liver from beef and pork is shown in Table 3.5.4-1. A detailed overview of the calculation of these concentrations is available in Chapter 11.2 (Appendix).

Table 3.5.4-1. The estimated PCDD/Fs and DL-PCBs concentrations in liver from Norwegian beef and pork used in the scenarios for exposure from consumption of beef and pork liver.

Food	Sum of PCDD/Fs and DL-PCBs (pg WHO_{2005}-TEQ/g whole weight)	
	Mean LB	Mean UB
Beef liver	0.021	0.027
Pork liver	0.021	0.095
Liver pâté[a]	0.088	Na

[a] Liver pâté values from Kvalem et al. (2009a).

The exposure was calculated based on the suggested portion sizes and the estimated concentration (Table 3.5.4-1), and the results are shown in Table 3.5.4-2.

Table 3.5.4-2. Beef liver, pork liver, and liver pâté; estimated exposure to the sum of PCDD/Fs and DL-PCBs (pg WHO_{2005}-TEQ/kg bw) from one portion (175 g in adults and 13-year-olds, 80g in children 1-9 years).

Age group	Beef liver, one portion		Pork liver, one portion		Liver pâté, one portion (á 20 g)	
	LB	UB	LB	UB	LB	UB
18-70-year-olds	0.05	0.06	0.05	0.21	Not estimated	Not estimated
13-year-olds	0.07	0.09	0.07	0.33	Not estimated	Not estimated
9-year-olds	0.09	0.12	0.09	0.42	Not estimated	Not estimated
4-year-olds	0.21	0.27	0.21	0.95	0.003	0.004
2-year-olds	0.29	0.37	0.29	1.30	0.005	0.005
1-year-olds	0.37	0.47	0.37	1.66	0.006	0.007

High consumption of liver pâté in children of 4 year age was selected as 6 daily portions. The exposure from liver pâté would be 0.13 pg TEQ/kg bw per week at LB and 0.17 pg TEQ/kg bw per week at UB.

3.5.5 Reindeer meat, liver, and fat

Some groups in the Norwegian population have high consumption of reindeer meat, and consume reindeer liver and fat in addition to the meat. VKM therefore made scenarios to estimate the contribution to total exposure for people consuming other parts of reindeer than the lean meat.

Traditional reindeer herders may use all edible parts of the reindeer, and a family of reindeer herders may consume approximately 10 animals a year (personal communication, J. A. Lifjell). The slaughter weight of the reindeer varies from 23 kg for a calf to 40 kg for a cow, and 60 kg for a buck (Matprat, 2021). This gives an average slaughter weight of 41 kg. Depending on the size and how well fed the animal is, 19 – 24% of the weight is bone. The fat content in reindeers will depend on the feeding, thus, there are variations in the fat content. It is especially the fat around the stomach and intestines that is used as food. On a well-fed reindeer there can be about 200 g fat per animal (personal communication, Lifjell).

VKM decided to use the same portion sizes for reindeer meat as for liver from livestock animals. The parameters used in the exposure estimates is 175 g reindeer meat per portion for adults and adolescents and 80 g per portion for children (< 10 year) and toddlers.

The amount of reindeer fat used for cooking varies with cooking method and type of dish. The portion size of 5 g for a slice of bread is given for butter and margarine in "weights, measures and portion sizes for foods table" (Dalane et al., 2015), and was used as a basis for estimating portion sizes for reindeer fat: 10 g fat per portion is used for adults and adolescents, 5 g per portion is used for children and toddlers.

Previous data from 2002 indicated that reindeer meat and kidney tallow from reindeer in Sør-Varanger contain higher levels of PCDD/Fs and non-ortho PCBs than reindeer further west in the Finnmark county (Haug et al., Unpublished). A report from the NFSA on

occurrence of contaminants in food on the Norwegian market in 2003-2005 reported 6-times higher concentration of PCDD/Fs and DL-PCBs in one sample of reindeer from Svanvik than in four reindeer from other areas (Kvalem et al., 2009a). More recently, a summary report from NILU to "Fylkesmannen i Finnmark" including concentration of PCDD/Fs and non-ortho-PCBs in nine reindeer (meat) from different areas in Finnmark sampled between 2013 and 2015 and six animals (kidney tallow) from 2016 in which also mono-ortho-PCBs were analysed, did not provide sufficient evidence to conclude that the concentrations vary across Finnmark (NILU, 2017). The contribution of mono-ortho-PCBs to total TEQ was low in these samples (<1.5% of total TEQ) (NILU 2017). The mean concentration of PCDD/Fs and DL-PCBs in the available reindeer samples was 6.89 pg TEQ/g fat (Table 3.1-2).

A study on reindeer in Finland found that the concentration of PCDD/Fs and DL-PCBs per gram fat are quite equally distributed in different parts of the animal, with the exception of liver, which like in other animals contain higher concentrations per gram fat (Holma-Suutari et al., 2016). This publication also indicated large variation in concentration depending on life-stages of the animals, with highest concentrations in calves, likely due to lactational transfer.

There are no concentration data for PCDD/Fs and DL-PCBs in reindeer liver from Norway. Therefore, the concentration in reindeer liver was estimated based on the ratio for PCDD/Fs and DL-PCBs in reindeer liver and fat in a study from Finland (Holma Suutari et al., 2014). Muscle and liver samples from five reindeer hinds and two calves were analysed for PCDD/Fs and DL-PCBs. Median ratio between liver and muscle for the sum of PCDD/Fs and DL-PCBs from the seven animals, based on lipid concentration, was 35 (see Chapter 11.3 (Appendix) for background information).

In total 19 samples were available on concentration of PCDD/Fs and DL-PCBs in reindeer, all from Finnmark County. As indicated above, concentrations in 15 animals were presented in reports to "Fylkesmannen i Finnmark" NILU (2017), of which 6 samples had information on all 29 congeners. In addition, two samples of reindeer (meat and fat) from Finnmark that were analysed as part of the Interlaboratory Comparison of Persistent Organic Pollutants in food (ILC POPs) at NIPH were available (Bremnes et al., 2012; Bremnes and Thomsen, 2018). Furthermore, 2 samples (kidney tallow) were analysed by FERA for NFSA (data received directly from NFSA). The mean concentration in these samples was 6.9 pg TEQ/g fat (UB). Only UB concentration has been used in scenario calculation (LB was not reported for all samples).

The concentration of PCDD/Fs and DL-PCBs on whole weight basis in reindeer meat, kidney fat, and liver were calculated based on the mean value in the available samples (6.89 pg TEQ/g fat) using 2.9% fat in meat, 4.5% fat in liver (4.5%, Holma Suutari et al., 2014), and 77.5% fat in kidney tallow (the mean in six kidney tallow samples (NILU, 2017)) and the ratio of 35 between liver and muscle of sum PCDD/F and DL-PCBs, based on lipid concentration. The resulting concentrations in whole weight used in the scenarios are presented in Table 3.5.5-1.

Table 3.5.5-1. Estimated PCDD/Fs and DL-PCBs concentrations used in the scenarios for exposure from consumption of reindeer meat and reindeer fat.

Food	Sum of PCDD/Fs and DL-PCBs, mean UB (pg WHO_{2005}-TEQ/g whole weight)
Reindeer meat	0.200
Reindeer fat	5.27
Reindeer liver	10.9

The exposure was calculated based on the suggested portion sizes and the estimated concentration (Table 3.5.5-1), and the results are shown in Table 3.5.5-2.

Table 3.5.5-2. The estimated exposure to PCDD/Fs and DL-PCBs (upper bound) from consumption of reindeer meat, liver, and fat (pg WHO_{2005}-TEQ/kg bw).

Age group	Reindeer meat, one portion*	Reindeer fat, one portion**	Reindeer liver, one portion*
18-70-year-olds	0.45	0.68	25
13-year-olds	0.70	1.1	38
9-year-olds	0.41	0.67	22
4-year-olds	0.91	1.5	50
2-year-olds	1.3	2.1	68
1-year-olds	1.6	2.6	87

*175 g/portion for adults and adolescents, 80 g/portion for children 1-9 years.
**10 g/portion adults/adolescents, 5 g/portion children 1-9 years.

An adult with body weight of 77.7 kg (mean in Norkost) will have exposure equal to the TWI of 2 pg TEQ/kg bw per week by consuming 777 g reindeer meat weekly (4.4 portions of 175 g) containing 0.2 pg TEQ/g meat (whole weight). For adolescents and children the amount of meat would be smaller, because of their lower body weight. However, exposure from other food will come in addition.

3.5.6 Summary of the scenarios

An overview of the exposure estimates for the scenarios (Chapter 3.5.1 to 3.5.5) are given in Table 3.5.6-1.

Table 3.5.6-1. An overview of the estimated exposure in the scenarios. Open fields: exposure is not estimated.

	18-70-year-olds		13-year-olds		9-year-olds		4-year-olds		2-year-olds		1-year-olds	
	LB	UB	LB	UB	LB	UB	LB	UB	LB	UB	LB	UB
Whole crab*, one portion (á 150 g)	4.7	4.9	7.2	7.5	Not estimated as an advice not to eat brown crab meat due to the content of PCDD/Fs and DL-PCBs is given for these groups by the NFSA.							
Filled crab shell*, one portion (á 150 g)	2.6	2.7	4.0	4.2								
Cod liver**, small portion (á 30 g)	5.9	6.2	9.0	9.6	Not estimated as an advice not to eat fish liver and cod roe-liver pâté due to the content of PCDD/Fs and DL-PCBs is given for these groups by the NFSA.							
Cod liver**, large portion (á 90 g)	18	19	27	29								
Cod roe-liver pâté**, one portion (á 20 g)	0.8	0.8	1.2	1.2								
Beef liver, one portion***	0.05	0.06	0.07	0.09	0.09	0.12	0.21	0.27	0.29	0.37	0.37	0.47
Pork liver, one portion***	0.05	0.21	0.07	0.33	0.09	0.42	0.21	0.95	0.29	1.30	0.37	1.66
Liver pâté, one portion (á 20 g)							0.003	0.004	0.005	0.005	0.006	0.007
Reindeer meat, one portion***		0.45		0.70		0.41		0.91		1.3		1.6
Reindeer liver, one portion***		25		38		22		50		68		87
Reindeer fat, one portion****		0.68		1.1		0.67		1.5		2.1		2.6
Cod liver oil, supplement, one daily portion (5 mL)		0.50		0.77		0.98		2.2		3.0		3.9
Marine oil, supplement, one daily portion (5 mL)		1.8		2.9		3.6		8.0		11		14

* Pregnant women, breastfeeding mothers, and women of childbearing age are not included as an advice not to eat this food item due to the content of PCDD/Fs and DL-PCBs is given by the NFSA.

** Pregnant women and breastfeeding mothers are not included as an advice not to eat this food item due to the content of PCDD/Fs and DL-PCBs is given by the NFSA.

*** 175 g in adults and 13-year-olds, 80 g in children 1-9 years.

**** 10 g for adults and adolescents, 5 g for children 1-9 years.

4 Risk characterisation

VKM decided to use the exposure estimates based on the VKM dataset (Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods) without fruit and vegetables as basis for the risk characterisation. This was selected because it is considered most likely to represent the true exposure in Norway, based on expert judgement as explained in 3.1.1 and 3.4. Analytical results from recent (2022) samples of apples, banana, carrots, cauliflower, broccoli, cabbage, and potatoes on the Norwegian market became available after the exposure was calculated by VKM. These results confirm that the concentrations are low and that fruit, vegetables and potatoes are not major contributors to exposure in Norway. New analytical results on meat (cattle, pig, chicken and liver pâté, not included in the exposure assessment) also confirmed low concentrations (NFSA 2022, results made available to VKM).

In the risk characterisation, the dietary exposures presented in Chapter 3 were compared to the TWI of 2 pg TEQ/kg bw per week for PCDD/Fs and DL-PCBs as given by EFSA (2018a).

The WHO_{2005} TEF-values (Van den Berg et al., 2006) are under revision, and there are indications that the relative potency of the most contributing DL-PCB (PCB-126) may be much lower in humans than suggested by its current TEF of 0.1. Such a decrease/reduction in the TEF factor for PCB-126, which with the present TEF-factors contributes 87% of the mean intake of DL-PCBs TEQ at LB (see 3.2.2) will reduce the contribution from DL-PCBs TEQ substantially. Therefore, VKM (like EFSA) find it relevant to compare the TEQ intake of PCDD/Fs only to the TWI, in addition to the intake from the sum of PCDD/Fs and DL-PCBs.

4.1 Applicability of the TWI for infants and children below 9 years of age

As indicated by EFSA, the TWI is not applicable for infants. The TWI was set to prevent a level in breastmilk that would result in infant exposure associated with adverse effects. Breastmilk from mothers with an exposure at the TWI result in infant exposure much higher than the TWI, however, the resulting serum concentrations are not associated with adverse effects. Therefore, the TWI set by EFSA (2018a) is not applicable for infants. Like EFSA, VKM therefore did not include 1-year-olds in the risk characterisation.

After being breastfed for 12 months with milk from a mother with exposure equal to the TWI, the dietary exposure to PCDD/Fs and DL-PCBs can be two-times the TWI for children of 1 to 9 years of age before the concentration in blood will approach the critical level in children at 9 years of age (EFSA, 2018a). Therefore, in the risk characterisation, the exposure estimates for 2-, 4- and 9-year-olds are compared to the TWI (2 pg TEQ/kg bw per week) and the value two-times the TWI (4 pg TEQ/kg bw per week).

4.2 Risk from dietary exposure in the general population

The estimated dietary exposures in different age groups relative to the TWI based on the VKM dataset without fruits, vegetables and potatoes is illustrated in Figures 4.2-1 (PCDD/Fs and DL-PCBs) and 4.2-2 (PCDD/Fs). The percent of the population with exposure above the TWI, and the times above the TWI, is shown in Tables 4.2-1 and 4.2-2.

The mean LB exposure in adults 18-70 years and women 18-45 years exceed the TWI for the sum of PCDD/Fs and DL-PCBs. At LB estimates, 71% of adults and 63% of women of childbearing age had exposures above the TWI, whereas almost all (98% and 97%, respectively) had an exposure above the TWI at the UB estimate (Table 4.2-1).

In adults (18-70 years), the mean LB and UB values were 1.4 and 2.3-times the TWI, and the UB value in adults at 95th-percentile exposure was 2.6 and 3.9-times the TWI, respectively. A similar overview of the results including fruits, vegetables and potatoes, is given in Table 12-3.

In other age groups (except 13-year-olds) the proportion of the population exceeding the TWI was higher than for adults and was 100% for children at UB estimates. The degree of exceedance was approximately two-times higher in 4-year-olds compared to adults. For 2-year-olds the exceedance was higher, whereas it was lower for 9-year-olds. Therefore, VKM considers that the degree of exceedance is of similar concern in adults and children, since a 2-times higher dietary intake in children than in adults was already accounted for in the modelling done by EFSA.

Considering the exposure to PCDD/Fs alone, 2% of the adults exceeded the TWI at the LB level, whereas 74% of adults (18-70 years) and 71% of women of fertile age exceeded the TWI at the UB level (Table 4.2-2). The 95th-percentile UB exposure was 2.1-times the TWI in adults. A similar overview for the results for PCDD/Fs alone including fruits, vegetables and potatoes is given in Table 12-4.

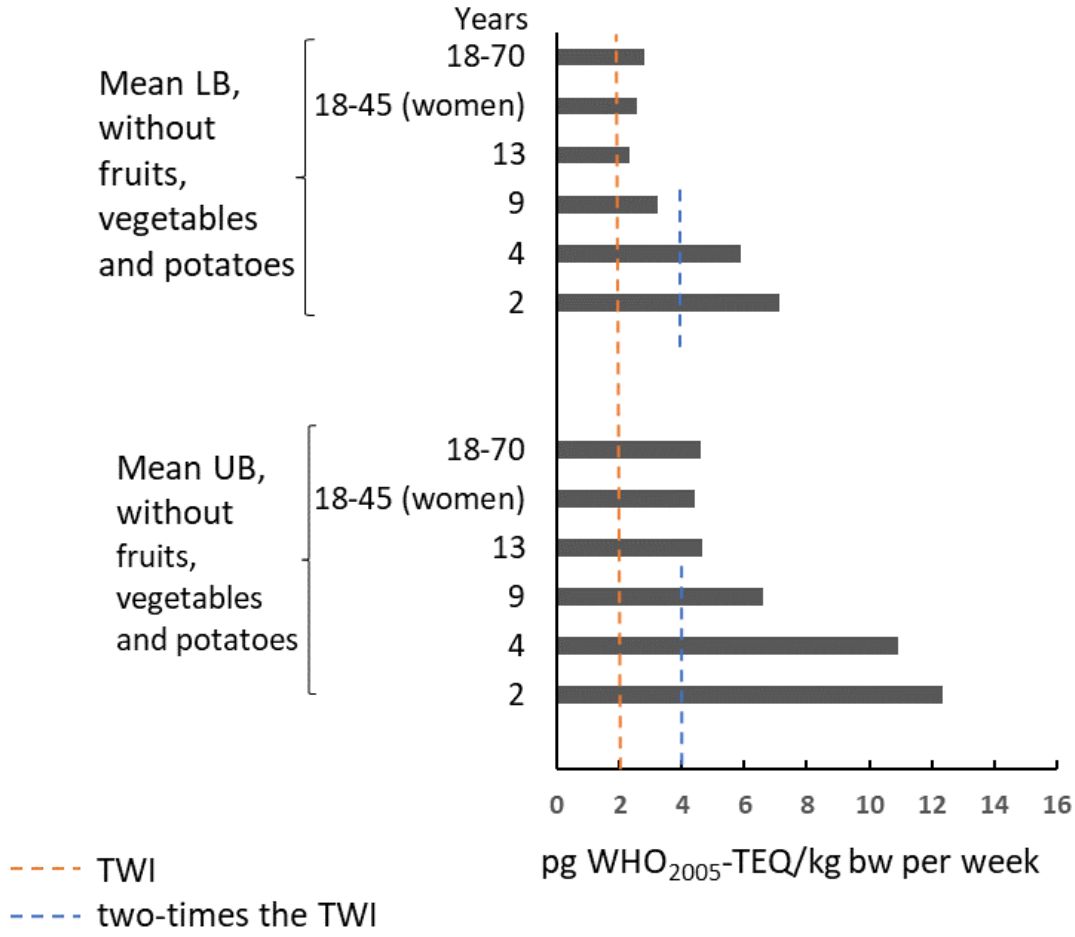

Figure 4.2-1. Estimated mean exposure to the sum of PCDD/Fs and DL-PCBs, not including fruits, vegetables and potatoes, obtained using the VKM dataset (Norwegian occurrence data for fish, meat, eggs, and dairy products combined with data from EFSA for other foods). Mixed model method applied for all age groups except 2-year-olds, for which the weighted observed individual means approach was applied. LB: lower bound; UB: upper bound.

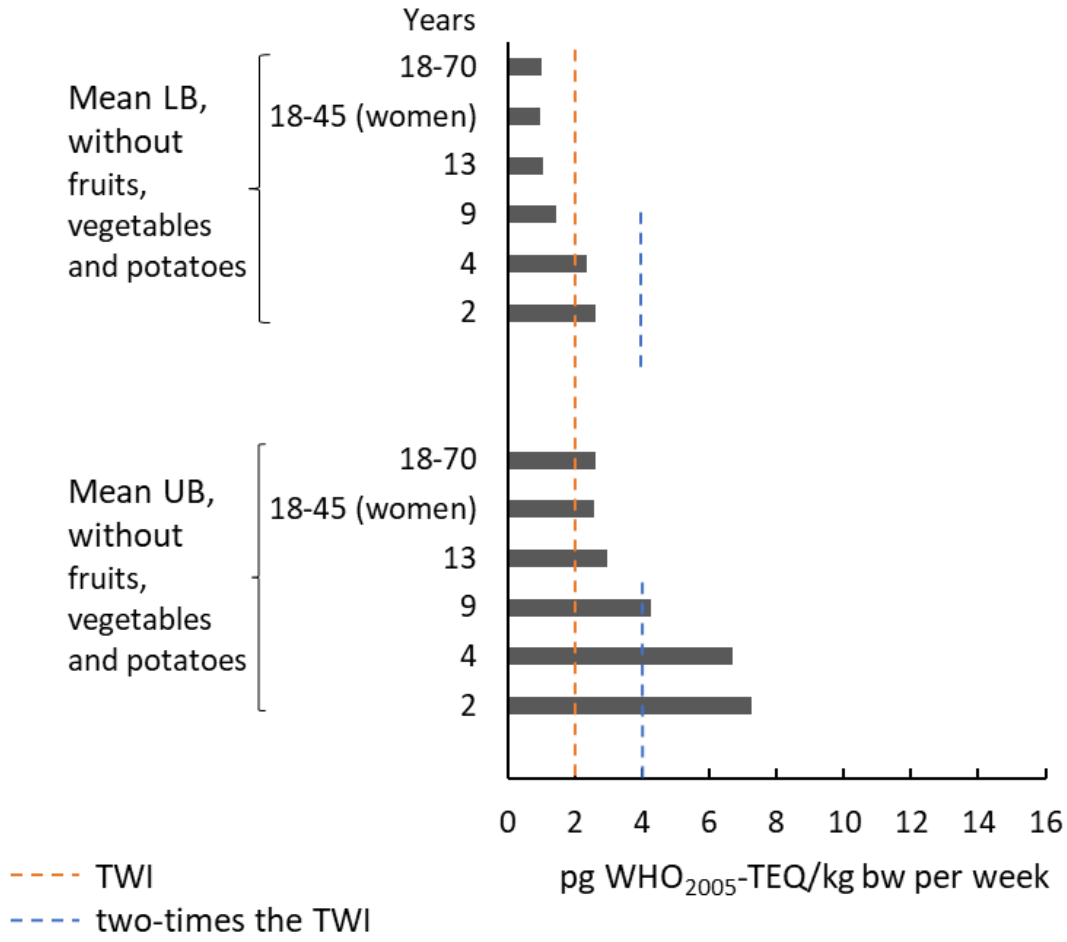

Figure 4.2-2. Estimated mean exposure to PCDD/Fs obtained using the VKM dataset (Norwegian occurrence data for fish, meat, eggs, and dairy products combined with data from EFSA for other foods). Mixed model method applied for all age groups except 2-year-olds, for which the weighted observed individual means approach was applied. LB: lower bound; UB: upper bound.

Table 4.2-1. Comparison between the TWI and the estimated exposure to the sum of PCDD/Fs and DL-PCBs, without fruits, vegetables and potatoes, obtained using the VKM dataset[a].

	18-70 years	18-45 years (women)	13-year-olds	9-year-olds	4-year-olds	2-year-olds
Percent of the population with exposure above the TWI (LB exposure)	71%	63%	55%	85%	100%	99%
Times above the TWI (mean LB exposure)	1.4	1.3	1.2	1.6	3.0	3.6
Times above the TWI (P95 LB exposure)	2.6	2.4	2.1	2.8	4.9	7.3

	18-70 years	18-45 years (women)	13-year-olds	9-year-olds	4-year-olds	2-year-olds
Percent of the population with exposure above the TWI (UB exposure)	98%	97%	96%	100%	100%	100%
Times above the TWI (mean UB exposure)	2.3	2.2	2.3	3.3	5.5	6.2
Times above the TWI (P95 UB exposure)	3.9	3.8	4.3	5.4	8.2	11.2

[a] VKM dataset: Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods.

Table 4.2-2. Comparison between the TWI and the estimated exposure to PCDD/Fs, without fruits, vegetables and potatoes, obtained using the VKM dataset[a].

	18-70 years	18-45 years (women)	13-year-olds	9-year-olds	4-year-olds	2-year-olds
Percent of the population with exposure above the TWI (LB exposure)	2%	2%	2%	14%	66%	66%
Times above the TWI (mean LB exposure)	0.5	0.5	0.5	0.7	1.2	1.3
Times above the TWI (P95 LB exposure)	0.9	0.9	0.9	1.2	1.8	2.4
Percent of the population with exposure above the TWI (UB exposure)	74%	71%	78%	98%	100%	100%
Times above the TWI (mean UB exposure)	1.3	1.3	1.5	2.1	3.3	3.6
Times above the TWI (P95 UB exposure)	2.1	2.1	2.6	3.4	4.8	6.2

[a] VKM dataset: Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods.

The food groups contributing most to dietary exposure were fish, meat and dairy in adults and children (see Chapter 3.3). While mackerel contributes most in children, salmon/trout and mackerel contribute equally in adults, and the contribution from other fatty fish is also substantial. In both adults and children, fish liver and roe contribute substantially to the mean exposure. The category "roe and liver" contributed from about 8 to 16% to the total exposure from fish. Fish liver contributed most for adults and 1-year-olds, whereas fish roe contributed most for 2-, 4-, 9- and 13-year-olds.

As fish liver is only consumed by a limited number of participants on the survey days and the exposure estimation from the total diet includes consumers and non-consumers, this implies

that the actual contribution of fish liver to the total exposure is higher for the consumers than what is shown in Chapter 3.3. Analysis of exposure in the 10% of respondents with the highest estimated total exposure to PCDD/Fs and DL-PCBs (in the corresponding age group) confirmed that fish and shellfish, and in particular fish liver, are major contributors. To estimate the exposure from one portion of fish liver for consumers only, scenario estimations were performed (see 3.5.2 and 4.2.2).

The critical effect for PCDD/Fs and DL-PCBs is reduced sperm concentration in boys following pre- and postnatal exposure. EFSA (2018a) stated that the available evidence indicates that there may be a postnatal period of sensitivity that might expand into puberty. The exposure for women from birth, during childhood and up to childbearing age (18-45) as well as exposure during childhood for boys, is therefore of particular relevance since the exposure in utero, via breastmilk and via food in childhood into puberty affect the blood concentration in young boys. Therefore, exposure in women 18-45 years is presented separately. It should be noted that the same women are also included in the conclusions for adults 18-70 years.

The dietary exposure to PCDD/Fs and DL-PCBs generally exceeds the TWI and this is of concern as it might reduce semen quality. Exposure to PCDD/F and DL-PCB may, therefore, be one of several factors that contribute to a lower semen quality (EFSA, 2018a). The probability of a decrease in sperm concentration increases with higher exceedance of the TWI.

In the critical study identified by EFSA (Minguez-Alarcon et al., 2017), there was a non-linear dose-response association for the sum of PCDD/Fs. A decrease in sperm concentration of about 40% was observed already in the second exposure quartile and the sperm concentration did not decrease further at higher exposure. A similar maximal effect size was observed in a cohort from Seveso and also in animal studies (EFSA, 2018a). Based on the current knowledge the decrease in sperm concentration is not expected to be more than approximately 40%, even at high exceedance of the TWI.

VKM notes that there are many environmental and genetic factors that can lead to decreased semen quality and exposure to PCDD/Fs and DL-PCBs above the TWI of 2 pg TEQ/kg bw per week is regarded as a contributing factor but not sufficient by itself to result in male infertility.

With the the range of estimated exposure to PCDD/Fs and DL-PCBs in Norway, other effects, such as postnatal developmental tooth enamel defects is not expected to occur.

The exposure to PCDD/Fs and DL-PCBs has shown a strong decline since the 1980s. From 1986 to 2005 the concentration of PCDD/Fs, DL-PCBs and NDL-PCBs in breastmilk from first-time mothers in Norway decreased by approximately 70% (VKM, 2013). The decline worldwide has been documented by WHO-coordinated monitoring of pooled breast milk samples from first-time mothers (EFSA et al., 2018a; van den Berg et al., 2017). Recent data from both WHO and Swedish mothers indicate that the decrease may be levelling off (EFSA 2018a; Gyllenhammar et al., 2021). According to EFSA, the pooled samples collected by WHO across European countries in 2014/2015 had concentrations of 2.4-5.7 pg WHO_{2005}-

TEQ/g fat for PCDD/Fs and 4.8–9.6 pg WHO2005-TEQ/g fat or the sum of PCDD/Fs and DL-PCBs (EFSA, 2018a). Since the dietary exposure to PCDD/Fs and DL-PCBs in Norway are estimated to be in similar range as in Europe, it is reasonable to believe that also the concentrations in breastmilk are in a similar range. The concentration in breastmilk associated with chronic exposure equal to the TWI was 5.9 pg TEQ/g fat (EFSA, 2018a). This again indicates that parts of the population have exposures exceeding the TWI.

4.3 Risk from exposure based on scenario consumption of particular food items

VKM identified some rarely consumed foods (crabs, fish liver, liver from livestock animals, reindeer meat, liver, and fat) that can have higher concentrations of PCDD/Fs and DL-PCBs than regularly consumed foods. The impact of consumption of such food on the total dietary exposure has been estimated by calculating the exposure per portion in different age groups. VKM did not make such scenario estimates for population groups for which there are warnings from the NFSA against consumption because of risk of too high exposure to PCDD/Fs and DL-PCBs.

Furthermore, although exposure from marine oil consumption is assessed by the national dietary surveys, VKM made scenario calculations for exposure to PCDD/Fs and PCBs from consumption of marine oils as food supplements. This was done to show the impact of the variability in occurrence levels in such oils supplement on exposure. Furthermore, this scenario calculation replaced a calculation of exposure from marine oils in "consumers only" in the national dietary surveys.

For 1-, 2-, and 4-year-olds, exposure from consumption of liver pâté used as bread spread was estimated in scenarios, since this could be a potential large single source.

The tolerable intake of PCDD/Fs and DL-PCBs was set on a weekly (and not daily or monthly) basis by EFSA to account for the fact that higher intake on a single day may not have high impact on the concentration of these substances in the blood provided that the intake over a week is not exceeding the TWI. If the tolerable intake would have been set on a monthly basis, the blood concentration of PCDD/Fs and DL-PCBs could have been increased substantially by occasional consumption of food with high concentrations. This could lead to elevated exposure of the foetus or sensitive tissues during a critical window. VKM has therefore not averaged exposure from seldomly consumed foods over a longer time period than one week.

4.3.1 Crabs

Crabs contain brown crab meat, which has considerably higher concentrations of PCDD/Fs and DL-PCBs than the white meat. The scenario intakes indicate that one portion of whole crab meat leads to an exposure 2.8-times the TWI in 13-years-olds and 2.5-times the TWI in adults. Eating filled crab shells that are commercially available leads to exposure that is 2.1-

times the TWI in 13-years-olds and 1.4-times the TWI in adults. Crabs are not a major part of the diet and the exposure from crab meat consumption therefore comes in addition to that from the regular diet, which already exceeds the TWI.

4.3.2 Fish liver

Consumption of a small to large portion of cod liver weekly leads to an exposure 4.8- to 14.4-times the TWI in 13-years-olds and 3.1- to 9.3-times the TWI in adults. Cod roe-liver pâté used as bread-spread on one slice of bread weekly contributes with approximately 50% of the TWI.

VKM noted that the fish liver and cod roe-liver pâté consumption is low in the dietary surveys, however, this low consumption constitute from 8-16% (LB) of the mean exposure in the included age groups, including both consumers and non-consumers. In adults, less than 3% report fish liver consumption monthly or more often and less than 10% report consumption of roe-liver pâté monthly or more often. In children, less than 1% at 2 and 4 years of age reported consumption. Fish liver contributed most for adults and 1-year-olds, whereas fish roe contributed most for 2-, 4-, 9- and 13-year-olds.

4.3.3 Marine oils

In adults, a daily intake of cod liver oil contributes with 0.50 pg TEQ (UB) of PCDD/Fs and DL-PCBs, which is 25% of the TWI. If the cod liver oil consumed contains PCDD/Fs and DL-PCBs equal to the ML applicable in Norway (4.0 pg TEQ/kg bw per week), the weekly exposure will be 1.8 pg TEQ/kg bw, which is 90% of the TWI. A similar consumption of marine oils in children contributes to more of the TWI due to their lower body weight. In 2-year-olds, daily consumption of 5 ml cod liver oil leads to exposure of 3.0 pg TEQ/kg bw per week, and if the marine oil consumed in this age group has concentrations equal to the ML of 4 pg TEQ/g fat the exposure will be 11 pg TEQ/kg bw per week, which is similar to the estimated mean UB exposure (12 pg TEQ/kg bw/week) from the total diet for that age group.

The highest mean UB exposure from marine oil supplements based on dietary surveys, including both consumers and non-consumers, was calculated for 4-year-olds (0.56 pg TEQ/kg bw per week). According to the dietary survey Ungkost 3, 28% of 4-year-olds consumed cod liver oil and 8% consumed an omega 3 supplement at least one of the 3-4 food registration days. Although a high proportion are consumers, the mean exposure is influenced by the mixing with non-consumers and thus, the actual exposure of the consumers is higher as shown by the scenario. The scenario calculations indicate that whereas marine oil supplement intake accounts for only 1.9 to 7.0% of the mean weekly (UB) exposure for different age groups (see 3.3) marine oil supplements can contribute a much larger proportion among daily consumers of such supplements, depending on type and brand.

4.3.4 Liver from livestock animals

One meal of liver from beef or pork contributes 2.5% (LB) to 11% (UB) of the TWI in adults, which is 1.8% (LB) or 4.5% (UB) of the estimated mean exposure in adults (based on exposure without potatoes, fruit and vegetables, Table 3.2.1-4). In 2-year-olds, the exposure from one portion of beef or pork liver corresponds to 4.1% (LB) to 5.9% (UB) of the mean total intake in the age group (Table 3.2.1-4). The scenario calculations indicate that liver from pork or beef is not a major contributor to the total exposure PCDD/Fs and DL-PCBs. The uncertainties in the estimates are high, due to estimated and not analysed concentrations in Norwegian beef and pork liver.

The occurrence value used for exposure from liver pâté consumption is higher than the occurrence value in liver itself and is based on three samples from 2003-2004. Considering the low number of samples and declining time-trend this occurrence value is therefore highly uncertain. The estimated exposure from a portion of liver pâté is low in the scenarios in children. High consumption (e.g. 6 slices of bread with liver pâté daily) would not make up a high contribution of the total exposure in these age groups.

4.3.5 Reindeer meat, liver and fat

One weekly meal of reindeer meat contributes approximately 23% (UB) of the TWI for adults, whereas a weekly portion of reindeer fat contributes approximately 34% of the TWI in adults. The contribution is higher for adolescents and children due to their lower body weight, despite the lower portion size in children. However, the relative contribution of a weekly meal of reindeer meat or reindeer fat compared to the mean estimated dietary exposure in the respective age group is similar (for reindeer meat 5.6% in adults and 5.7% in 2-year-olds, for reindeer fat 8.5% in adults and 9.3% in 2-year-olds). Regular consumption of reindeer meat (with or without reindeer fat) is expected to replace other food.

Consumption of one weekly portion of reindeer liver contributes more than 12-times the TWI in adults, and the exceedance of the TWI by one portion of reindeer liver is substantial also in adolescents and children. Compared to the estimated UB mean exposure in adults, a weekly meal of reindeer liver would contribute 3-times more. This indicates that reindeer liver consumption is a potential high contributor of PCDD/Fs and DL-PCBs.

The uncertainty in the concentrations in reindeer liver in Norway is high, since it is estimated based on meat and kidney tallow concentration using the ratio between liver and fat in reindeer samples from Finland. However, the concentrations in reindeer liver used for the calculations (10.9 pg TEQ/g ww) is higher than concentrations reported in liver from seven reindeer in Finland (0.3 to 5.9 pg TEQ/g ww, calculated from concentrations given in lipid weight and mean fat % in liver reported in Holma Suutari et al., 2014).

5 Factors that can contribute to exposure reduction

Food is the main source to human exposure to PCDD/Fs and DL-PCBs, and this is due to contaminated environment and/or contaminated animal feed. Farmed animals, including fish, are mainly exposed to PCDD/Fs and DL-PCBs though their feed. The feed may be contaminated by accident such as under the Belgian dioxin and PCB incident (van Larebeke et al., 2001) or by use of contaminated ingredients (Hoogenboom et al., 2020). Furthermore, marine oils with relatively high concentrations of PCDD/Fs and DL-PCBs (although still below regulatory limits) may be used in feed for farmed fish. In addition, food from farmed animals foraging outdoor on contaminated sites may have elevated levels of PCDD/Fs and DL-PCBs (Heres et al., 2010; Hoogenboom et al., 2016; Hoogenboom et al., 2010; Hoogenboom et al., 2015). In the European Economic Area, the level of PCDD/Fs and DL-PCBs in feed and feed material are regulated through Directive 2002/32/EC (2002) and amendments.

5.1 Cleaning of PCDD/Fs and DL-PCBs from fish feed and impact on human exposure

In the "Benefit-risk assessment of fish and fish products in the Norwegian diet – an update", VKM described how changes in the feed composition has led to reduced levels of PCDD/Fs and DL-PCBs in farmed Atlantic salmon (VKM, 2014). The substitution of fish oil with vegetable oils have decreased the level of PCDD/Fs and DL-PCBs in feed and subsequently in Atlantic salmon (VKM, 2014, and references therein). In 2013, marine oils constituted 10.9% of the feed, and in 2016, the inclusion had decreased somewhat to 10.4% (Aas et al., 2019). For comparison, in 2000, the marine oils constituted 31.1% of the feed (Aas et al., 2019). The inclusion of marine oils in feed for Atlantic salmon seems to have stabilised at around 10-11%, in consideration of the welfare of the fish. A way to reduce the level of PCDD/Fs and DL-PCBs in feed and farmed fish further could be to reduce the levels in fish oil and fish meal. VKM has earlier described how cleaning of fish oil leads to reduced levels of PCDD/Fs, DL-PCBs and other POPs in farmed Atlantic salmon (VKM, 2014).

The VKM decided to estimate the effect of cleaning of fish oil and fish meal on the concentration of PCDD/Fs and DL-PCBs in fillet of farmed Atlantic salmon and on human exposure.

5.1.1 Impact of cleaning of farmed fish feed on the concentrations in Atlantic salmon

A validated, congener specific model describing the transfer of PCCD/Fs and DL-PCBs from feed to fillet (Berntssen et al, 2016) was used. Three scenarios were used:

1) No cleaning of fish oil and fish meal
2) Cleaning of fish oil

3) Cleaning of fish oil and fish meal

For each scenario the current (2016) feed composition was used, i.e. 14.5% (of feed) fish meal, 10.4% fish oil, 40.3% plant protein, 10.6% carbohydrate sources, 20.2% plant oil and 4.0% micronutrients (Aas et al, 2019).

The degree of decontamination was set as a mean percentage decrease in concentration based on results from the industry provided to EFSA for their assessment of the decontamination processes. The results from the most efficient decontamination processes were used, and the degree of decontamination was assumed to be similar among all PCDD/Fs (17 congeners) and DL-PCBs (12 congeners). This is a simplification of the real situation, but congener-specific data were not available from the EFSA assessments.

EFSA has assessed four methods on decontamination of PCDD/Fs and DL-PCBs from fish oil and/or fish meal for use in fish feed (EFSA et al., 2017a; EFSA et al., 2018b; EFSA et al., 2017b; EFSA et al., 2018c). For fish oil the most efficient decontamination process assessed by EFSA is an adsorption with active carbon followed by physical filtration (EFSA et al., 2017b). The process removes up to 95% pf the PCCD/Fs and up to 45% of the DL-PCBs (EFSA et al., 2017b). For fish meal, the most effective decontamination process assessed by EFSA is an extraction of the fish oil from the fish meal followed by a cleaning of the fish oil using active carbon and physical filtration. The cleaned fish oil is then added back to the residual material to reconstitute the fish meal (EFSA et al., 2018b). The process removes 97% of the PCCD/FS and 93% of the DL-PCBs (EFSA et al., 2018b)..

Data on the concentrations of PCDD/Fs and DL-PCBs in feed ingredients were taken from the feed surveillance program run by the Institute of Marine Research (IMR) on behalf of the NFSA. Data for fish oil and fish meal were from 2020 (IMR, unpublished data) while data for plant protein, plant oil and premix (micronutrients) were from 2010-2012 (surveillance program run by Institute of Marine Research on behalf of the NFSA). Only UB values were used in the model estimations. When cleaning of oil or meal leads to concentrations below LOQ, the concentration value is substituted by the LOQ. If LB values had been used, many data points would have to be set as 0. The uncertainty in the LB estimates would be high, and the difference between UB and LB estimates would increase, making the impact of cleaning hard to interpret in the LB estimates.

The estimated concentrations for feed and Atlantic salmon fillet are shown in table 5.1.1-1. Cleaning of fish oil (scenario 2) led to a decrease in the concentration of PCDD/Fs and DL-PCBs in feed and fillet. The cleaning of fish meal (scenario 3) led to a further reduction in the concentrations in feed and fillet. Looking at the sum of the concentrations of PCDD/Fs and DL-PCBs the concentration in fillet was reduced from 0.51 pg WHO_{2005}-TEQ/g ww to 0.34 pg WHO_{2005}-TEQ/g ww when the fish oil was cleaned (33% reduction), and further reduced to 0.26 pg WHO_{2005}-TEQ/g ww when both fish oil and fish meal were cleaned (49%). The significance of these reduced concentrations in Atlantic salmon fillet on the dietary exposure to PCDD/Fs and DL-PCBs are described in Chapter 5.1.2.

The concentrations predicted for scenario 1 (no cleaning of fish oil and fish meal) are comparable to concentrations of PCDD/Fs and DL-PCBs found in commercial Atlantic salmon in 2019. Surveillance data for 2019 showed mean concentrations of 0.25, 0.27 and 0.51 pg WHO_{2005}-TEQ/g ww for PCDD/Fs, DL-PCBs and ∑PCDD/FS and DL-PCBs, respectively. The model estimated concentrations were 0.28, 0.23 and 0.51 pg WHO_{2005}-TEQ/g ww for PCDD/Fs, DL-PCBs and ∑PCDD/FS and DL-PCBs, respectively. This confirms the validity of the model used.

Table 5.1.1-1. Estimated concentrations of PCDD/Fs and DL-PCBs in fish feed and salmon fillet by scenarios without cleaning, cleaning of fish oil and cleaning of fish oil and meal (upper bound, in pg WHO_{2005}-TEQ/g ww).

	Without cleaning (scenario 1)	**Cleaning of fish oil (scenario 2)**	**Cleaning of fish oil and meal (scenario 3)**
Feed			
PCDD/Fs	0.36	0.26	0.20
DL-PCBs	0.24	0.19	0.11
∑PCDD/FS and DL-PCBs	0.60	0.44	0.31
Fillet			
PCDD/Fs	0.28	0.20	0.15
DL-PCBs	0.23	0.15	0.11
∑PCDD/FS and DL-PCBs	0.51	0.34	0.26

The contribution (in percentage) of the feed ingredients to the total amount of PCDD/Fs and DL-PCBs in feed are shown in Table 5.1.1-2. In the feed with non-decontaminated fish oil and fish meal (scenario 1), fish oil is the major (38%) contributor to the total amount of PCDD/Fs and DL-PCBs in the feed, while fish meal and plant protein (including carbohydrate sources) contribute with 24% each. When fish oil is cleaned (scenario 2), the contribution from fish oil to the total amount of PCDD/Fs and DL-PCBs in feed is reduced to 16%, and the major contributors are fish meal and plant protein (including carbohydrate sources) each contributing with 33%. In the feed with cleaned fish oil and fish meal, the major (48%) contributor to the total amount of PCDD/Fs and DL-PCBs in feed is plant protein and carbohydrate sources. Fish meal and fish oil contribute with 24% and 25%, respectively.

Table 5.1.1-2. Contribution (in %) of different feed ingredients to the total amount of PCDD/Fs and DL-PCBs in feed by scenarios without cleaning, cleaning of fish oil and cleaning of fish oil and meal.

	Without cleaning (scenario 1)*	**Cleaning of fish oil (scenario 2)***	**Cleaning of fish oil and meal (scenario 3)***
Fish oil	38	16	24
Fish meal	24	33	3
Plant protein and carbohydrate sources	24	33	48
Plant oil	13	17	25

	Without cleaning (scenario 1)*	Cleaning of fish oil (scenario 2)*	Cleaning of fish oil and meal (scenario 3)*
Micronutrients	2	2	3

* Does not sum to exactly 100% due to rounding.

5.1.2 Impact of cleaning of Atlantic salmon feed on human exposure

In order to assess effects of the cleaning scenarios on the total exposure, we used total exposure estimates based on the VKM dataset, upper bound, without fruits, vegetables, and potatoes. In assessing the scenarios, we assumed that the dioxin values in Table 5.1.1-1 apply to both farmed salmon and farmed trout. The effects of the cleaning scenarios are shown in Table 5.1.2-1 (PCDD/Fs and DL-PCBs, 29 congeners) and 5.1.2-2 (PCDD/Fs, 17 congeners)

Table 5.1.2-1. Total dietary exposure (upper bound, in pg WHO_{2005}-TEQ/kg bw per week) to PCDD/Fs and DL-PCBs when occurrence in salmon is substituted with the concentrations resulting from cleaning of fish feed. Based on the mixed model approach is applied for all population groups, except for 1-and 2-year-olds, for which weighted observed individual means are shown.

Age group	Exposure	Without cleaning (Scenario 1)	Cleaning of fish oil (Scenario 2)	Change (%), scenario 2 vs scenario 1	Cleaning of fish oil and meal (Scenario 3)	Change (%), scenario 3 vs scenario 1
Adults (18-70 years)	Mean	4.60	4.42	-3.9	4.34	-5.7
	P95	7.86	7.51	-4.5	7.34	-6.6
Women (18-45 years)	Mean	4.37	4.20	-3.9	4.11	-6.0
	P95	7.42	7.04	-5.1	6.85	-7.7
13-year-olds	Mean	4.62	4.42	-4.3	4.32	-6.5
	P95	8.36	7.79	-6.8	7.52	-10.1
9-year-olds	Mean	6.54	6.29	-3.8	6.17	-5.7
	P95	10.6	10.1	-5.2	9.83	-7.6
4-year-olds	Mean	10.8	10.4	-3.9	10.2	-5.7
	P95	16.4	15.8	-3.7	15.5	-5.3
2-year-olds	Mean	12.3	12.1	-1.8	12.0	-2.7
	P95	22.4	22.1	-1.2	21.9	-1.9
1-year-olds	Mean	12.2	11.8	-2.9	11.7	-4.2
	P95	24.1	23.5	-2.8	23.3	-3.4

Table 5.1.2-2. Total dietary exposure (UB, in pg WHO_{2005}-TEQ/kg bw per week) to PCDD/Fs when occurrence in salmon is substituted with the concentrations resulting from cleaning of fish feed, based on the mixed model approach is applied for all population groups, except for 1-and 2-year-olds, for which weighted observed individual means are shown.

Age group	Exposure	Without cleaning (Scenario 1)	Cleaning of fish oil (Scenario 2)	Change (%), scenario 2 vs scenario 1	Cleaning of fish oil and meal (Scenario 3)	Change (%), scenario 3 vs scenario 1
Adults (18-70 years)	Mean	2.65	2.57	-3.02	2.52	-4.91
	P95	4.35	4.18	-3.91	4.08	-6.21
Women (18-45 years)	Mean	2.58	2.52	-2.33	2.46	-4.65
	P95	4.22	4.05	-4.03	3.94	-6.64
13-year-olds	Mean	3.00	2.92	-2.67	2.86	-4.67
	P95	5.34	5.09	-4.68	4.93	-7.68
9-year-olds	Mean	4.29	4.19	-2.33	4.11	-4.20
	P95	6.90	6.67	-3.33	6.50	-5.80
4-year-olds	Mean	6.74	6.56	-2.67	6.44	-4.45
	P95	9.55	9.38	-1.78	9.24	-3.25
2-year-olds	Mean	7.30	7.19	-1.51	7.13	-2.33
	P95	12.6	12.5	-0.80	12.4	-1.03
1-year-olds	Mean	7.14	6.97	-2.38	6.87	-3.78
	P95	13.4	13.0	-2.99	12.9	-3.81

The results show that cleaning of fish oil or fish feed has the potential to reduce the mean total dietary exposure to PCDD/Fs and DL-PCBs by 2.7-6.5% for scenario 3 (1.8-4.3% for scenario 2). The relative reductions for the 17 PCDD/F congeners are lower, with 2.3-4.9% and 1.5-3% reductions, respectively. This is because the concentrations of PCDD/Fs are approaching the detection limits by decontamination and no further decrease is quantifiable. The relative reduction to the 95th percentile is greater than the reduction to the mean for all groups except children of 1, 2, and 4 years.

5.2 PCDD/Fs and DL-PCBs in chicken eggs from hens with outdoor access

The term free-range eggs may include eggs both from birds with and without access to the outdoor environment. Hens must have access to outdoor environment in ecological farming. Around 7% of the egg producing flocks in Norway are under ecological farming. There are also hens with outdoor access (free land) that are not produced by ecological farming (Animalia, 2020b). The importance of feed as source of contamination of eggs is exemplified by the Belgian dioxin crisis (Covaci et al., 2008), but also contaminated soil may cause elevated levels of PCDD/Fs and PCBs in eggs. The levels of PCDD/Fs and DL-PCBs appear to be higher in eggs from hens with access to the outdoors (outdoor/open-air growing conditions) than in eggs from hens kept indoor (Schoeters and Hoogenboom (2006); EFSA, 2018a). In case studies, contaminated land due to pollution (Lambiase et al., 2017) and old building materials (Piskorska-Pliszczynska et al., 2014; Winkler, 2015) as well as local waste

burning (Hoogenboom et al., 2016) have been identified as sources of PCDD/Fs and DL-PCBs in eggs from free-range hens. Increased levels in eggs were particularly pronounced at sites near municipal waste incinerators, but elevated levels are also observed in eggs from sites with low soil contamination. In some areas in Europe measures have been taken to remove point sources at farms (Weber et al., 2018). For free-range hens with outdoor access, it is important to control the environment, i.e., do not keep hens on contaminated soil/land and remove remains of old building material.

5.3 Measures to reduce contamination of PCDD/Fs and DL-PCBs in soil and sediments

Anthropogenic PCDD/Fs are primarily formed during combustion of municipal solid waste items that contain chlorinated substances. PCDD/Fs are also formed in various chemical manufacturing processes and in the mining industry (IOM, 2003).

Emission of PCBs reached a peak in the 1970s and 1980s and major sources of exposure are so-called reservoir sources due to past spread of contaminants (Breivik et al., 2002; Dopico and Gómez, 2015; IOM, 2003). PCBs have been extensively used in various products, such as in paints, in transformers and capacitors, windows and various building materials (e.g. Tanabe (1988)). PCBs have been extensively used in various products, such as in paints, in transformers and capacitors, windows and various building materials (e.g. Tanabe (1988)). This has led to considerable environmental spread and pollution. Particularly, at waste disposal sites and harbors, there are areas with very high levels of these substances (Cornelissen et al., 2012; Weber et al., 2011). At some areas, PCBs have also been in building materials and is a continuous source of release, for example during precipitation (e.g. Jartun and Pettersen (2010)). This implies that efforts should be done to reduce further spread for example during precipitation events. The run-off water, which contain various suspended material and water, may spread the contaminants to the surrounding area and finally to the marine environment. In populated areas, particulate material in the run-off water will be deposited in sand traps and in the end reach a wastewater treatment plant. The accumulation of these substances in animals in remote areas, have also shown that these can be subjected to long range transport through atmosphere and sea currents (e.g., MacDonald et al. (2000)).

5.3.1 Reduction in PCDD/Fs and DL-PCBs release

The release of PCDD/Fs and DL-PCBs has been reduced by improved sorting of waste, technical improvements of the waste incineration process and better cleaning systems for the smoke. In modern waste treatment plants, the PCDD/Fs are removed from the smoke with a filter and a scrubber, which implies that the smoke goes through a filter to collect particles (fly ash) followed by washing procedure (scrubbing) with water added various additives, such as active coal. Organic substances, such as PCDD/Fs and PCBs, are readily adsorbed to sorbents such as active coal. The fly ash and sludge from the water treatment is then deposited at facilities for hazardous waste.

Important measures to reduce exposure to PCDD/Fs, PCBs and other persistent chemicals have been improved waste treatment control and clean-up measures at contaminated sites. In addition, there have been substantial effort to identify sources of release to be able to take appropriate measures and reduce the spread. For example, much effort has been done to collect old luminaires and transformers containing PCB for hazardous waste disposal (Lovdata, 2020). The current regulations on contaminated land is described in The Pollution Control Regulations, which gives instructions on measures to limit pollution of various contaminants (Lovdata, 2004).

5.3.2 Reduction of spread of already released PCDD/Fs and DL-PCBs

An approach that has been used to reduce the spread of PCDD/Fs and DL-PCBs is to mix or cap the contaminated topsoil with a sorbent to stabilize the contaminant and reduce spread (Zhang et al., 2016). This approach may be particularly relevant at waste disposal sites in which soil and sediments are deposed. Relevant adsorbents may be various types of charcoal and natural clay, which have high affinity to organic compounds (Chai et al., 2012). At land the clean topsoil or excavated area can be re-vegetated to avoid further erosion and stabilize the treated area. Another approach is natural recovery, which rely on natural properties to decrease contaminants by natural sedimentation of clean sediments (Zhang et al., 2016). At land, this can be facilitated by speeding up the re-vegetation to avoid extensive erosion, which is an important cause of contaminant spread. Regardless of remediation measures, it is however important to have control on the sources of the contaminants to avoid re-contamination of the site.

In contaminated harbors or sites at land, often in combination with dredging, contaminated sediment have been covered with a capping, which may be clean sand, soil, or sediment and/or a sealing with a cloth. Capping with clean sediment is an approach to increase the rate of natural sedimentation and reduce the contaminant flux to the overlying water and regarded as a cost-effective approach. By adding active adsorbents, it is possible to increase the efficiency and reduce the thickness of the capping (Zhang et al., 2016). Sediments in the Grenland fjords, Norway contaminated with PCDD/Fs, PCBs were capped with active carbon and clay in 2009 (Schaanning et al., 2021). Nine years after, it was shown that capping with active carbon significantly reduced the PCDD/Fs and PCB levels in sediment dwelling organisms. Capping with clay had no effect. The capping with active carbon, however, affected the benthic community, and nine years after capping, the communities had still not recovered (Trannum et al., 2021).

5.3.3 Removal of sources of PCDD/Fs and DL-PCBs

A common approach is to remove the sediment or soil by dredging/excavation followed by deposition at facilities for hazardous waste or landfills.

Despite extensive remediation efforts to remove sources of PCDD/Fs, PCBs and other persistent chemicals at polluted sites, there are relatively few studies showing the effects of

dredging in a long-term perspective. Even though the main sources of contamination have been removed, effects of the concentration levels in biota have not been conclusive (National Research Council, 2001). In a recent report by Magnussen et al. (2019) remediation projects at contaminated sites in Norway was evaluated and it was concluded that the contaminant levels were substantially decreased, but the effects of the measures on biota was poorly documented. During dredging it is often observed a temporarily increase in the contaminant levels in the water column and in organisms due to release from resuspended sediments (Bremle and Larsson, 1998; National Research Council, 2001; Nelson and Bergen, 2012; Rice et al., 1987). In 1993, a PCB-contaminated lake in Sweden was remediated by dredging showing a considerable decrease in the concentration of PCBs in the lake sediment (Bremle and Larsson, 1998). The concentration of PCB in fish collected three years after the measures showed an approximate 50% reduction that was attributed to the measures. The decrease was, however, only marginally better than the decrease in fish from the background sites. In a study by Jude et al. (2010) a 65% decrease in PCB concentration in Walleyes from the Saginaw River (Lake Huron, US) was observed between 1990 and 2007, which partly was attributed to dredging of PCB hotspots in 2001. In 1998, a PCB contaminated area at the Haakonsvern naval base harbor in Bergen was dredged without any observable decrease in the PCB levels in caged blue mussel four years after the measures compared to pre-dredging levels (Johnsen et al., 2003).

6 Uncertainties

An overview of the main factors contributing to uncertainty in the conclusions in this risk assessment is given below. The uncertainties in setting the TWI was assessed by EFSA and applies also for the present risk assessment. From EFSA (2018a): "*The CONTAM Panel considered that the impact of the uncertainties on the risk assessment of PCDD/Fs in food is moderate. For the sum of PCDD/F and DL-PCBs, due to the uncertainty in therelative potency of PCB-126 in humans, the impact of the uncertainties on the risk assessment is high. Overall, the assessment is likely to be conservative.*" The uncertainty assessment in this report address the uncertainties in the occurrence data, the consumption data and the estimated overall exposure to PCDD/Fs and DL-PCBs, including the exposure scenarios for selected food items.

Occurrence of PCDD/Fs and DL-PCBs in food

The uncertainty introduced by low availability of occurrence data for a food group increases with the decreasing number of data. For foods frequently consumed in large quantities even a small change in the occurrence data could have a significant effect on the exposure estimate. With regard to the VKM dataset, considered to be the most appropriate regarding occurrence of PCDD/Fs and DL-PCBs in food consumed by the Norwegian population, the occurrence data for meat, grain, and fruits, vegetables and potatoes were scarce. The effect on the estimated exposure may be an over- or an underestimation.

No Norwegian occurrence data were available for fruits, vegetables and potatoes for the exposure estimations, and only a limited number were available from EFSA (2018a). EFSA indicated that the uncertainty related to reported occurrence levels in samples of plant origin was high, due to the low number of samples in most categories. In addition, occurrence of these substances in fruits, vegetables and potatoes was unexpected, as PCDD/Fs and DL-PCBs are lipid soluble and accumulate in the food chain, whereas fruits, vegetables and potatoes generally have low fat content (0.1-0.4%) and are low in the food chain. To account for these uncertainties, the exposure was estimated both with and without fruits, vegetables and potatoes. The estimates including fruits, vegetables and potatoes most likely represents an overestimation, whereas the estimates without fruits, vegetables and potatoes most likely represents an underestimation. However, VKM considered the estimates without fruits, vegetables and potatoes to be the most appropriate since the presence of these substances in foods with low fat content and low in the food chain are hard to explain. Analytical results from recent (2022) samples of apples, banana, carrots, cauliflower, broccoli, cabbage, and potatoes on the Norwegian market became available after the exposure was calculated by VKM confirm that the concentrations are low and that fruit, vegetables and potatoes are not major contributors to exposure in Norway (NFSA 2022, results made available to VKM).

It is not known to which extent the samples with analyzed concentrations of food produced in Norway or food reported to EFSA is representative of the food marketed or consumed in

Norway. For instance, the mean concentration in different fish species was used for exposure assessment, but any geographical differences in concentrations was not taken into consideration. The market share of fish from different catching areas is not known. This can lead to both under-and over-estimates of exposure. For fish it is known that fish from areas with local pollution (e.g. harbours and fjords) may contain higher contamination levels and for people consuming fish from self-catch the exposure may be underestimated.

Consumption data

Several parameters introduced uncertainty in the consumption data (see below).

- There is a tendency for FFQ surveys, used for the 1- and 2-year-olds, to overestimate food consumption. E.g. for 1-year-olds and 2-year-old girls in Småbarnskost 3, the reported energy was 1.4-times and 1.3-times the estimated energy requirement in the Nordic Nutrition Recommendations (NNR, 2012), respectively. This leads to an overestimation of the exposure.
- There is a tendency for recording methods such as 24-hour recalls and food diaries, used for 4, 9-, and 13-year-olds and adults, to underestimate energy intake. The effect of this recall bias is different for the different age groups. For Ungkost 3, the energy requirement for 4-year-olds was in line with the estimated daily energy requirements (NNR, 2012) and the uncertainty in food consumption is thus considered small in this age group. The underreporting of energy intake in adults and 13-year-olds were 16 and 33%, respectively. The exposure, in particular in 13-year-olds, is considered an underestimate.
- Two 24-hour recalls introduce uncertainty in the intake estimates of food eaten seldom or not frequently because the number of survey participants and/or survey days per participand is too low to capture seldomly consumed food. This is partly compensated by using MMs. The results from the propensity questionnaire in Norkost 3, which could have reduced the uncertainty in lack of registration of food not reported by participants over the survey days, were not taken into consideration in the modelling. For exposure to PCDD/Fs and DL-PCBs this leads to an underestimate of the contribution from seldomly consumed food that can contain relatively high levels of PCDD/Fs and DL-PCBs. The exposure from seldomly consumed foods was assessed by scenarios.
- Regarding reported intake of marine oil supplements, it is likely that marine oils and cod liver oils may be mixed. This can lead to both over- and underestimation of exposure. The concentration in both cod liver oils and other marine oils were varying and information on the market share of different brands is not available to VKM and could not be accounted for.
- Regarding reported portion sizes, these may be too small or two large and can lead to both over- and underestimation of exposure.
- Regularly updated dietary surveys are needed to capture changes in the general population's diet. The data in Norkost 3 (adults 18-70 years) were collected in 2010-2011, and the data in Ungkost 3 (4-, 9-, and 13-year-olds) were collected in 2015-2016. This increases the uncertainty in the estimates, particularly for adults.

Approaches for exposure estimation

While correcting for population representativity, survey respondents were not given different weights for all relevant characteristics, as either the data were not available (e.g., household income) or original survey sampling did not allow it (e.g., country of birth was restricted in the way that was incompatible with population representativity). Household income and education level are correlated. Thus, weighting for the latter partially makes up for lack of weighting for the former

It is known that OIMs, and particularly W-OIMs, do a reasonably good job in estimating the mean of the exposure distribution for the population, while overestimating the standard deviation, with too low levels for low percentiles and too high levels for high percentiles (Boon and van der Voet, 2015). It is known that MMs improve on W-OIM-based habitual-exposure distributions by estimating the day-to-day (within person) variation (Boon and van der Voet, 2015). The survey data for 4-, 9-, and 13-year-olds are for 3-4 consecutive days. The day-to-day correlation of food intake and exposure introduced by this study design can be negative, if participants follow the day(s) of eating in excess by eating "too little" the following day(s), or positive, if participants eat leftovers on the following day(s). Attributing too much or too little of the overall variability to day-to-day variation will affect the habitual-exposure estimates at the individual level. 13-year-olds exhibit highest (also higher than adults) variability in habitual exposure at the individual level (even when averaging the daily exposure at the 1-year horizon). This could be attributable to both the misattribution of day-to-day variation and generally poorer reporting quality in this age group, as also evident from the above-mentioned widespread underreporting of energy.

Scenarios

Several parameters introduced uncertainty in the scenarios (see below), and the effect on the estimated exposure may be an over- or an underestimation.

- Occurrence data for liver from livestock animals, white crab meat, and reindeer fat and liver, were not available. The concentrations used were estimated values based on few samples and have high uncertainty in both directions
- The availability to occurrence data for cod roe-liver pâté (n=2) and liver pâté (n=3) was low, and the year of sampling was before 2010. To ensure a better representativity of the occurrence data, it was stated in the protocol that data sampled before 2010 should not be included. However, as these data were the only available samples, they were included and are associated with high uncertainty in both directions, but due to decreasing time trend it most likely represents an overestimate.
- The occurrence data for reindeer were limited to reindeer from Finnmark. The representativity for reindeer from other areas is not known.

The portion sizes were estimated based on previous reported portion sizes and expert judgement. The uncertainty can go in both directions.

The exposure estimates based on the EFSA dataset is considered an overestimate of the true exposure in Norway. The exposure estimate based on the VKM dataset is considered closer to the true exposure in Norway and should be considered a best estimate. The overall uncertainties in the exposure estimates are smaller for fish, eggs and dairy products than for meat and other foods with low number of samples.

TEF-values

There is high uncertainty in the WHO_{2005} TEF-values (Van den Berg et al., 2006, EFSA, 2018a) and these TEFs are under revision. A reduction of one or more TEF-values will reduce the total estimated exposure, whereas an increase will result in an increased total exposure. There are indications that the relative potency of the most contributing DL-PCB (PCB-126) may be much lower in humans than suggested by its current TEF of 0.1. The contribution of the 12 DL-PCBs to the LB total estimated exposure to PCDD/Fs and DL-PCBs ranged from 55 to 62% for the different age groups, whereas the contribution to the UB estimated exposure ranged from 33 to 38%. According to EFSA (2018a), 63.2% of the LB mean exposure comes from DL-PCBs, and the contribution from PCB-126 alone amounts to about 55% of the total LB estimated exposure. Thus, a reduction of the TEF-value for PCB-126 will result in a significant reduction of the total estimated PCDD/F and DL-PCB exposure. To get an impression of the impact of a substantially lower TEF for PCB-126, the exposure to PCDD/Fs alone using the WHO_{2005} TEF-values in comparison to the TWI was also addressed. This is indicative of a possible exposure after revision of the TEFs, but should be considered an underestimate, as some contribution of DL-PCBs will still be present.

7 Conclusions and answers to the terms of reference

Terms of reference 1: *Perform exposure assessments of dioxins and dl-PCBs for the total Norwegian diet and assess if the Norwegian population or sub-groups of the population have different eating patterns leading to different dietary dioxin and dl-PCB exposures compared to what EFSA reported for the European population. NFSA asks VKM to assess if separate calculations are needed for sub-groups of the population or for certain food categories (beyond those already mentioned in 2. and 3. below). If yes, NFSA asks VKM to perform the necessary assessments and calculations.*

VKM decided to use the exposure estimates based on the VKM dataset (Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods) without fruit, vegetables and potatoes as basis for the risk characterisation because these are considered most likely to be representative of the true exposure in Norway. This was concluded by VKM after considering the uncertainties connected to contribution of fruits, vegetables and potatoes to the total exposure, comparison of concentrations in food produced domestically to the concentrations in food submitted to ESFA, and the degree of self-sufficiency of different types of food. Analytical results from recent (2022) samples of apples, banana, carrots, cauliflower, broccoli, cabbage, and potatoes on the Norwegian market became available after the exposure was calculated by VKM. These results confirm that the concentrations are low and that fruit, vegetables and potatoes are not major contributors to exposure in Norway. New analytical results on meat (cattle, pig, chicken and liver pâté, not included in the exposure assessment) also confirmed low concentrations (NFSA 2022, results made available to VKM).

The available occurrence data indicate that concentrations of PCDD/Fs and DL-PCBs are lower in fish species commonly consumed in Norway (e.g. farmed salmon, mackerel, herring) and in eggs, milk and meat sampled in Norway compared with the concentrations in similar products submitted by European countries. For some foods (e.g. meat) this conclusion is based on only a few samples and has high degree of uncertainty, whereas for fish, milk and eggs a larger number of samples form basis for the conclusion.

A comparison of the exposures in adults reported by EFSA and the exposures estimated by VKM based on consumption data in Norkost 3 indicate that the exposure estimates reported by EFSA and by VKM are quite similar, indicating that the exposure in Norway is in similar range as in the rest of Europe.

The critical effect for PCDD/Fs and DL-PCBs is reduced sperm concentration in boys following pre- and postnatal exposure and there may be a postnatal period of sensitivity that might expand into puberty. The exposure for women from birth, during childhood and up to childbearing age (18-45) is of particular relevance since PCDD/Fs accumulate in the body

and are transferred to the foetus and child via breastfeeding. VKM therefore assessed exposure in women aged 18-45 years separately from adults, adolescents and children, all age groups.

The dietary exposures were compared to the TWI of 2 pg TEQ/kg bw per week for PCDD/Fs and DL-PCBs as given by EFSA (2018a). The WHO_{2005} TEF-values are under revision, and there are indications that the relative potency of the most contributing DL-PCB (PCB-126) may be much lower in humans than suggested by its current TEF of 0.1. Because such a decrease in the TEF factor for PCB-126, which with the present TEF-factors contributes 87% of the mean exposure to DL-PCBs TEQ, will reduce the contribution from DL-PCBs substantially, VKM find it relevant to also compare the exposure to PCDD/Fs alone using the WHO_{2005} TEF-values to the TWI, in addition to the exposure from the sum of PCDD/Fs and DL-PCBs.

The mean and 95-percentile estimated weekly exposure to the sum of PCDD/Fs and DL-PCBs (pg WHO_{2005}-TEQ/kg bw/week) for the available dietary surveys in Norway and for women aged 18-45 years is shown in Table 7.7-1.

Table 7.1-1. Estimated exposure to PCDD/Fs and the sum of PCDD/Fs and DL-PCBs (pg WHO_{2005}-TEQ/kg bw/week), without fruits, vegetables and potatoes, obtained using the VKM dataset.

Age group	Sum of PCDD/Fs and DL-PCBs				PCDD/Fs			
	Mean		95-percentile		Mean		95-percentile	
	LB	UB	LB	UB	LB	UB	LB	UB
Adults (18-70 years)[a]	2.8	4.6	5.2	7.9	1.0	2.6	1.7	4.3
18-45 years (women)[a]	2.5	4.4	4.8	7.4	0.97	2.6	1.7	4.2
13-year-olds[a]	2.3	4.7	4.3	8.5	1.0	3.0	1.8	5.3
9-year-olds[a]	3.2	6.6	5.6	10.8	1.5	4.2	2.4	6.7
4-year-olds[a]	5.9	10.9	9.9	16.3	2.3	6.7	3.6	9.5
2-year-olds[b]	7.1	12.3	14.5	22.4	2.6	7.2	4.7	12.5
1-year-olds[b]	7.3	12.2	15.8	24.2	2.6	7.1	5.1	13.2

[a]mixed model approach; [b]adjusted OIM

The mean LB exposure in adults 18-70 years and women 18-45 years exceed the TWI for the sum of PCDD/Fs and DL-PCBs. At LB estimate, 71% of adults and 63% of women of childbearing age had an exposure above the TWI, whereas almost all (98% and 97%, respectively) had an exposure above the TWI at the UB estimate.

In adults (18-70 years), the mean LB and UB values were 1.4 and 2.3-times the TWI and the 95th-percentile LB and UB values were 2.6 and 3.9-times the TWI, respectively.

In other age groups (except 13-year-olds) the proportion of the population exceeding the TWI was higher than for adults and was 100% for 2-, 4- and 9-year-olds at the UB estimate. The degree of exceedance was approximately two-times higher in children at 4 years of age than in adults, although higher in 2-year-olds and lower in 9-year-olds. VKM considers that the degree of exceedance is of similar concern in adults and children, since a 2-times higher

dietary exposure in children than in adults was already accounted for in the modelling done by EFSA.

Considering the exposure to PCDD/Fs alone, 2% of the adults exceeded the TWI at the LB estimate, whereas 74% of adults (18-70 years) and 71% of women of fertile age exceeded the TWI at the UB estimate. The degree of exceedance at UB 95th-percentile exposure was in adults 2.1-times.

The major food groups contributing to exposure was (fatty) fish, meat and dairy products in all age groups.

VKM identified a need for separate scenario exposure calculations for sub-groups of the population in order to cover exposure from particular foods items. In addition to the foods addressed under terms of reference 2 (marine oils) and 3 (reindeer meat), whole crabs, fish liver, liver from livestock and reindeer and fat from reindeer can contain relatively high levels of PCDD/Fs and DL-PCBs that are not captured by the dietary surveys because the foods are consumed by few people and/or not on a daily basis. The scenarios on reindeer fat and liver are presented together with reindeer meat under terms of reference 3.

Crabs

Crabs contain brown crab meat, which has considerably higher concentrations of PCDD/Fs and DL-PCBs than the white meat. The scenario calculations indicate that one portion of whole crab meat leads to an exposure 2.8-times the TWI in 13-years-olds and 2.5-times the TWI in adults. Eating filled crab shells that are commercially available leads to exposure that is 2.1-times the TWI in 13-years-olds and 1.4-times the TWI in adults. Crabs are not a major part of the diet and the exposure from crab meat consumption therefore comes in addition to that from the regular diet

Fish liver

Consumption of a small to large portion of cod liver leads to an exposure 4.8- to 14.4-times the TWI in 13-years-olds and 3.1- to 9.3-times the TWI in adults. Cod roe-liver pâté used as bread-spread on one slice of bread weekly contributes with approximately 50% of the TWI.

VKM noted that the fish liver and cod roe-liver pâté consumption is low in the dietary surveys. In adults, less than 3% report fish liver consumption monthly or more often and less than 10% report consumption of roe-liver pâté monthly or more often. In children, less than 1% at 2 and 4 years of age reported consumption. Still, this low consumption constitutes from 8-16% (LB) of the estimated mean exposure, including both consumers and non-consumers. Analysis of exposure in the 10% in the dietary surveys with the highest estimated total exposure in the different age groups confirmed that fish liver is a major contributor in those with top 10% exposure.

Liver from livestock animals

The scenario calculations indicate that liver from pork or beef or use of liver pâté as bread spread are no major contributors to the total exposure to PCDD/Fs and DL-PCBs. The uncertainties in the estimates are high, due to estimated and not analysed concentrations in Norwegian beef and pork liver.

Terms of reference 2: *Perform a risk assessment of dioxins and dl-PCBs in marine oils taken as food supplements.*

In adults, a daily intake of cod liver oil contributes with 0.50 pg TEQ (UB) of PCDD/Fs and DL-PCBs, which is 25% of the TWI. If the cod liver oil consumed contains PCDD/Fs and DL-PCBs equal to the ML applicable in Norway (4.0 pg TEQ/kg bw per week), the weekly exposure will be 1.8 pg TEQ/kg bw, which is 90% of the TWI.

In 2-year-olds, exposure from daily consumption of 5ml cod liver oil is 3.0 pg TEQ/kg bw per week, and if the marine oil consumed in this age group has concentrations equal to the ML of 4 pg TEQ/g fat the exposure will be 11 pg TEQ/kg bw per week, which is similar to the estimated mean UB exposure (12 pg TEQ/kg bw/week) from the total diet for that age group.

According to the dietary survey Ungkost 3, 28% of 4-year-olds consumed cod liver oil and 8% consumed an omega 3 supplement at least one of the 3-4 food registration days. Although a high proportion are consumers, the mean exposure is influenced by the mixing with non-consumers and thus, the actual exposure of the consumers is higher as shown by the scenario. The scenario calculations indicate that whereas marine oil supplement intake accounts for only 1.9 to 7.0% of the mean weekly (UB) exposure for different age groups, marine oil supplements can contribute a much larger proportion among daily consumers of such supplements, depending on type and brand.

Terms of reference 3: *Calculate how much reindeer meat (with the reported dioxin and dl-PCB values) that can be consumed before the TWI of dioxins and dl-PCBs will be exceeded. Alternatively, what is the additional contribution of dioxins and dl-PCBs from reindeer meat compared to an average diet?*

VKM identified a need to calculate scenarios on reindeer fat and liver as indicated above and present it together with the reindeer meat.

According to scenarios, one weekly meal of reindeer meat contributes approximately 23% (UB) of the TWI for adults, whereas a weekly portion of reindeer fat contributes approximately 34% of the TWI in adults. The contribution is higher for adolescents and children due to their lower body weight, despite the lower portion size in children. However, the contribution of a weekly meal of reindeer meat or reindeer fat compared to the mean estimated dietary exposure in the respective age group is similar (for reindeer meat 5.6% in adults and 5.7% in 2-year-olds, for reindeer fat 8.5% in adults and 9.3% in 2-years-olds). An adult with body weight of 77.7 kg (mean in Norkost) will have exposure equal to the TWI of 2 pg TEQ/kg bw per week by consuming 777 g reindeer meat weekly containing 0.2 pg

TEQ/g meat (whole weight). For adolescents and children, the amount of meat would be smaller, because of their lower bw. However, exposure from other food will come in addition. Regular consumption of reindeer meat (with or without reindeer fat) is expected to replace other food and is not considered to be of particular concern by VKM.

Consumption of one weekly portion of reindeer liver results in exposure which is more than 12 times the TWI for adults, and the exceedance of the TWI by one portion of reindeer liver is substantial also in adolescents and children. Compared to the estimated UB mean exposure in adults, a weekly meal of reindeer liver would contribute 3-times more. This indicates that reindeer liver consumption is a potential high contributor of PCDD/Fs and DL-PCBs.

The uncertainty in the concentrations in reindeer liver in Norway is high, since it is estimated based on meat and kidney tallow concentration using the ratio between liver and fat in reindeer samples from Finland.

Terms of reference 4: *Assess health consequences of exceeding the TWI, both related to duration and degree of TWI exceedances.*

The critical effect for PCDD/Fs and DL-PCBs is reduced sperm concentration in boys following pre- and postnatal exposure. EFSA (2018a) stated that the available evidence indicates that there may be a postnatal period of sensitivity that might expand into puberty. The exposure for women from birth, during childhood and up to childbearing age (18-45) as well as exposure during childhood for boys, is therefore of particular relevance since the exposure in utero, via breastmilk and via food in childhood into puberty affect the blood concentration in young boys.

The dietary exposure to PCDD/Fs and DL-PCBs generally exceed the TWI and this is of concern as it might reduce semen quality. The main contributing food groups are fish, dairy products and meat. These are foods that are central in the diet also to fulfil nutritional needs. It should be noted that this risk assessment addresses only the risk of dietary exposure of PCDD/Fs and DL-PCBs and does not take into consideration any beneficial health effects of foods or nutrients in food in a wider context.

The probability of a decrease in sperm concentration increase by higher exceedance of the TWI. In the critical study identified by EFSA (Minguez-Alarcon et al., 2017) there was a non-linear dose-response association for PCDD/Fs. A decrease in sperm concentration of about 40% was observed already in the second exposure quartile and the sperm concentration did not decrease further at higher exposure. A similar maximal effect size was observed in a cohort from Seveso and also in animal studies (EFSA, 2018a). Based on the current knowledge the sperm concentration is not expected to decrease more than approximately 40% even at high exceedance of the TWI.

VKM notes that there are many environmental and genetic factors that can lead to decreased semen quality and exposure to PCDD/Fs and DL-PCBs above the TWI of 2 pg TEQ/kg bw per

week is regarded as a contributing factor but not sufficient by itself to result in male infertility.

The tolerable intake of PCDD/Fs and DL-PCBs was set on a weekly (and not daily or monthly) basis by EFSA. This was done to account for the fact that higher intake on a single day may not have high impact on the concentration of these substances in the blood provided that the exposure over a week is not exceeding the TWI. If the TWI would have been set on a monthly basis, the blood concentration of PCDD/Fs and DL-PCBs could have been increased substantially by occasional consumption of food with high concentrations. This could lead to elevated exposure of the foetus or sensitive tissues during a critical developmental stage. VKM has therefore not averaged exposure from seldomly consumed foods over a longer time period than one week.

Regarding other possible effects from exposure to PCDD/Fs and DL-PCBs, only an effect on development of teeth (enamel hypomineralization) after exposure via breastmilk is considered relevant at current exposure levels. It was estimated by EFSA that enamel hypomineralization might be associated with a concentration in breast milk of around 9.2 pg PCDD/F-TEQ/g fat (DL-PCBs were not considered in the epidemiological studies addressing these effects). Data on breastmilk from first time mothers in Uppsala, Sweden (Gyllenhammar et al., 2021, see Chapter 1.4) indicate that total TEQ concentrations in this range may be present in some women in Sweden in 2017, although the majority had much lower concentrations. The situation is expected to be similar in Norway, based on geographical and cultural similarities, and that concentration in breastmilk in Norway in 2006 was in similar range as it was in Uppsala (VKM 2014).

Terms of reference 5: *Identify risk-reducing factors, which could reduce dioxin and dl-PCB exposure in the population. If possible, present the risk reducing effects quantitatively.*

VKM has not been able to identify single factors that alone can reduce the exposure to a level where all parts of the population will have exposure below the TWI for PCDD/Fs and DL-PCB. Instead, several factors may together contribute to a continued declining trend in exposure.

One way to reduce human exposure to PCDD/Fs and DL-PCBs is to reduce the levels of PCDD/Fs and DL-PCBs in products of farmed animals. Farmed animals, including fish, are mainly exposed to PCDD/Fs and DL-PBCs though their feed, but exposure from contaminated abiotic environment (e.g. soil) is also possible.

The inclusion of fish oil in Atlantic salmon feed has declined from 31% in year 2000 but has been stable around 10-11% the later years. The inclusion of fish meal in Atlantic salmon feed has also declined and is currently around 14.5%. Both fish oil and fish meal can be decontaminated. By scenario calculations VKM assessed the impact of decontamination of fish oil, or decontamination of both fish oil and fish meal, in Atlantic salmon feed on the UB exposure in the different age groups.

The results indicated that cleaning of fish oil and fish feed has the potential to reduce the total mean dietary exposure to PCDD/Fs and DL-PCBs by 2.7-6.5% in different age groups. If only the fish oil was decontaminated the reduction in exposure was 1.8-4.3%. The relative reductions for the 17 PCDD/F congeners were lower (2.3-4.9% reduction with refining of both fish oil and fish meal and 1.5-3% reductions with cleaning of only fish oil). This is because the concentrations of PCDD/Fs in decontaminated fish oil and meal are approaching the detection limits and no further decrease is quantifiable. The relative reduction to the 95th percentile was somewhat greater (maximally 7.7% reduction) than the reduction to the mean exposure for all groups except for children of 1, 2, and 4 years of age.

To reduce concentration in eggs, it is important to control the environment for free-range hens with outdoor access, i.e., not keeping hens on contaminated soil/land and removing remains of old building material.

Reduction in levels in wild fish and animals can only be achieved by reducing releases and clean-up of specific contaminated sites. A general decrease in contamination level of PCDD/Fs and DL-PCBs in the environment will over time lead to lower concentration in food, and consequently in people. The most important measures to reduce exposure to PCDD/Fs, PCBs and other persistent chemicals in the past have been improved waste treatment control and clean-up measures at contaminated sites.

8 Data gaps

Biomonitoring data
Biomonitoring data of PCDD/Fs and DL-PCBs in children and adults, and in particular women of child bearing age in Norway is needed. This would decrease the uncertainty by obtaining knowledge whether Norwegians have higher exposure due to the high fish consumption relative to most European countries, as the exposure obtained by the use of EFSA dataset indicates. Biomonitoring data is also necessary to update time trends in exposure, as the most recent biomonitoring samples from Norway are from 2006.

Occurrence data
More occurrence data in food consumed in Norway is needed in order to reduce uncertainty in exposure assessments. This is in particular applicable to vegetables, potatoes and fruit, which are consumed in large quantities and that can be important contributors even if concentrations are low. Such information should be obtained from samples that are representative of what is consumed in Norway. More occurrence data in meat from livestock is needed. Since the self-sufficiency is high, it should be obtained from food produced in Norway. More and updated concentrations in cod roe-liver pâté and the bread spread "Kaviar" is needed to reduce the uncertainties in exposure estimates, in particular for children.

Consumption data
Consumption data need to be updated on a regular basis in order to capture changes in food consumption and the impact on exposure. If dietary changes in the direction that larger shares of the population eat more plant-based food and meat imitates occurrence data in such food is also needed.

There is a need for updated information on exposure of people with high consumption of self-captured fish and other seafood.

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10 Appendix I: Technical details for the exposure assessments

10.1 Data imputation

10.1.1 Adults

The following variables had missing values and were imputed: Sex, Age, Weight, Height, Education, Region, HH.size, and HH.children. HH.size and HH.children stand for household size and the number of children in household, respectively. While these two variables and height are not directly used in the present assessment, their inclusion may improve imputation accuracy for the variables of interest. There were 1, 1, 32, 2, 6, 1, 11, and 4 missing observations, respectively.

Imputations were implemented in R using mice package v.3.13.0, setting the seed for imputation to 123 (reported for complete replicability). The number of iterations was set to 150.

In imputation, the variables were used as predictors for each other (without intercepts). The models used for imputation were *logreg, pmm, pmm, pmm, polr, polyreg, polr,* and *polr*, respectively. "logreg" stands for Logistic regression, "pmm"—for Predictive mean matching, "polr"—for Proportional odds model, and "polyreg"—for Polytomous logistic regression.

10.1.2 1-, 2-, 4-, 9-, and 13-year-olds

The following variables had missing values and were imputed: Weight, Education, and Region. There were 607, 25, and 2 missing observations, respectively. Of missing weight observations, 1-, 2-, 4-, 9-, and 13-year-olds contribute 155, 380, 17, 29, and 26, respectively. The same parameters and approach were used as for adults. The models used for imputation were *pmm, polr,* and *polyreg*, respectively.

10.2 Adoption of nutritional databases

There are three available food-composition databases in KBS, from newest to oldest: AE-18, AE-14, and N3. Each subsequent database includes updates to product formulations. Using the most recent database is thus desirable. However, more recent databases do not cover all foods included in earlier surveys. A hybrid food-composition database is therefore used for the exposure estimations. It is a superset of all three food-composition databases, where the most recent available data is selected for each food code. This ensures the broadest coverage of reported food intake along with the most recent food-composition data available.

Exposure to PCDD/Fs and DL-PCBs at the individual level is based on survey responses and information on PCDD/F and DL-PCB concentrations in the coded foods and drinks that have been consumed. In some cases, a food code refers to an elementary ingredient, e.g., cod fillet. In other cases, a code refers to processed food with multiple ingredients (composite products), e.g., fish balls.

For those composite products that had a recipe in the food composition database, ingredient amounts were calculated based on the list of ingredients in the recipe and the nutrient content of the product and its ingredients. If the recipe ingredient list did not allow to find product weights that would give a good correspondence between the composite product and the ingredients, as measured by the nutritional content, the recipe was rejected, and the product remained as a composite product. Otherwise, the optimal ingredient weights, the weights with the best correspondence between the composite product and the ingredients, were used to translate the composite product into its ingredients. The exception was made for fish composite products. Even when the recipe was otherwise deemed to be of insufficient quality, the fish fillet was extracted from the dish using the best fitting value match between the nutrient values of the product and the nutrient values of the fish. Splitting composite dishes into ingredients, whenever possible, allows a tighter match between dietary intake and occurrence data for dioxins, as well as better attribution of sources of dioxins grouped by food categories.

10.3 Specification of the mixed model applied

10.3.1 Fixed effects

The model used to estimate exposure to PCDD/Fs and DL-PCBs for adults is presented below:

$$f(Compound) \sim Const + Sex + Age + Education.High + Region + Weekday + Month$$

- $f()$ is the Box-Cox transformation $(Compound_i^{\lambda} - 1)/\lambda$
- $Const$ is the constant term (or intercept)
- Sex, $Education.High$, $Region$, $Weekday$, and $Month$ are treated as factor (or dummy) variables
- Age is treated as a continuous variable, measured in years

For 4-, 9-, and 13-year-olds, the variables $Month$ and Age are not included in the model, as the dietary survey for these age groups, Ungkost 3, cover only a short period within a calendar year, and each age cohort is estimated in a separate regression.

The key underlying assumption that needs to be made when applying Bayesian MMs relates to the distribution of the residuals, and while several choices are feasible, in this case, the choice is limited to normal (or Gaussian) distribution. In this risk assessment, the Box-Cox

transformation was applied to achieve a distribution where the fitted residuals are not too divergent from normality (see 10.4.1).

10.3.2 Random effects

MMs are particularly useful when there is more than than one source of uncertainty in the data. There is flexibility when it comes to specifying the structure of uncertainty (the structure of the error term of the regression). Two components are included as random effects. First, the random component allows to model variability by group, implemented in the present assessment to model day-to-day variability by the respondent. Second, the residual component captures remaining variability of each observation for the respondents (see Chapter 10.4 for further technical details). In the adopted models, both components are clustered by sex.

10.3.3 Estimation of chronic-exposure distributions

The objective of utilizing the MM approach is to simulate outcomes of the fitted model(s), to arrive at distribution estimates for chronic exposures.

The steps to estimate the distribution of chronic exposure are as follows:

- The model is fit: the coefficients for the fixed effects, and the elements of the variance-covariance matrices for the random effects are estimated.
- For each survey respondent, $365 * 100$ daily observations are simulated (equivalent to 100 years of daily data). The transformation of the modelled compound is reversed.
- Averages of each of the 365 daily observations are taken. For each survey respondent, there are now 100 simulated results.
- For the 100 simulated results, 100 weighted distributions are computed.
- Based on these 100 distribution results, both the average distribution and its confidence intervals are computed. The average distributions form the background data for the MM results presented in the present assessment.

10.4 Technical details for the mixed model applied

There are some further technical ingredients in MMs beyond those that are introduced in Chapter 10.3. In implementing the MM approach for the current assessment, the MCMCglmm v.2.32 library in R v.4.1.0 was used, a package for fitting generalised linear mixed models using Markov chain Monte Carlo techniques. While not strictly necessary, it is recommended

to specify priors. Priors set original values used in the variance-covariance matrices of the random effects and residuals.

Four alternative priors were considered. The code used to specify the priors is as follows:

```
priors<-list(
 list(R = list(R1 = list(V = diag(2)/10, nu=3)),
  G = list(G1 = list(V = diag(2)/10, nu=3))),
 list(R = list(R1 = list(V = diag(2)/1, nu=1)),
  G = list(G1 = list(V = diag(2)/1, nu=1))),
 list(R = list(R1 = list(V = diag(2), nu=1.002)),
  G = list(G1 = list(V = diag(2), nu=2,alpha.mu=c(0,0),
   alpha.V=diag(2)*a))),
 list(R = list(R1 = list(V = diag(2)*1e-6, nu=3)),
  G = list(G1 = list(V = diag(2), nu=2,alpha.mu=c(0,0),
   alpha.V=diag(2)*a))))
```

Model estimates showed little dependence on the priors. The first prior gave marginally better fit (as measured by the DIC measure), so it is the model results under the first prior that are reported in the MM tables, in all cases.

The model was estimated after setting the seed to 1234 (set and reported for reproducibility). For adults the model was run as follows:

```
MCMCglmm(
 as.formula(paste0(transformed.nutr.cols.MM[i],
  "~Sex + Age + Education.High+Landsdel+Weekday+Month")),
 prior = p,
 random = ~idh(Sex):BB.ID, rcov = ~idh(Sex):units,
 data = df[grepl("^N",BB.ID) & Month!=0,],
 nitt=35000, thin=10, burnin=5000)
```

where $BB.ID$ is the respondent identifier, random specifies the random component, and rcov is the residual component introduced in Chapter 10.3.2.

For minors (4-, 9-, and 13-year-olds), the model was run as follows:

```
MCMCglmm(
 as.formula(paste0(transformed.nutr.cols.MM[i],
  "~Sex + Education.High+Landsdel+Weekday")),
 prior = p,
 random = ~idh(Sex):BB.ID, rcov = ~idh(Sex):units,
 data = df[Age==a,], nitt=35000, thin=10, burnin=5000)
```

The next step is to generate a table comprising fixed-effect structures for each survey participant for one year:

```
simulation.data<-copy(df)[,.(BB.ID,Sex,Age,Weight,Education.High,Landsdel)]
simulation.data[,Sex:=as.factor(Sex)][,Landsdel:=as.factor(Landsdel)][,
```

```
 Education.High:=as.factor(Education.High)]
simulation.data<-simulation.data[rep(1:.N,each=365),][,
 Date:=seq(as.Date("2021-01-01"),by=1,length.out=365),by="BB.ID"]
simulation.data[,Weekday:=relevel(as.factor(format(Date,"%u")),ref = 1)][,
 Month:=as.factor(as.integer(format(Date,"%m")))]
simulation.data[,Date:=NULL]
```

Note, in the simulations, the days are set from Monday to Sunday, from January 1 through December 31.

This table along with the previously estimated model results (y below) are combined to generate simulated results:

```
simulate(object=y, nsim = 100, seed = 1234L,
 newdata=(simulation.data[grep("^N",BB.ID)][,
  c("BB.ID",strsplit(as.character(y$Fixed$formula)[3]," + ",fixed = TRUE)[[1]]),with=FALSE][
,
   (gsub("log\\(([^)]*)\\).*$","\\1",y$Fixed$formula[2])):=0]),
 type = "response", it=NULL, posterior = "all", verbose=TRUE)
```

Note, 100 vectors of the left-hand-side variable (LHS) are simulated, 100 years' worth of data for each survey participant.

To get back to the original scale of the LHS, the transformation is reversed. The result is merged with the respondent IDs, for example, for the log transformation:

```
data.table(data.frame(
 BB.ID=simulation.data[grep(paste0("^N"),BB.ID)][,BB.ID],
 Prior=p,V1=exp(sim.try.A[[c]][[p]])
```

Each year of data is then converted to daily averages for that year:

```
sim.A.yr<-lapply(1:length(sim.try.A),
 function(c) lapply(1:length(priors),
  function(p) unique(copy(sim.try.A[[c]][[p]])[,
   (setdiff(names(sim.try.A[[c]][[p]]),c("BB.ID","Prior"))):=lapply(.SD,function(x) sum(x)/36
5),
 by=c("BB.ID","Prior"),.SDcols=setdiff(names(sim.try.A[[c]][[p]]),c("BB.ID","Prior"))])))
```

Thus, there are 100 annual averages for each respondent.

As an indication of estimate reliability at the level of each participant, the mean and the standard deviation of the annual averages was computed. It was done for each person, each compound, and each prior. The average mean and average standard deviation across all participants, for the chosen prior, is then considered. For compounds where this average standard deviation is high (relative to the average mean), it can be concluded that the

probabilistic nature of the model structure adds more uncertainty to the estimate, by forcing an ill-suited distributional assumption, than it takes away, by modelling the within-person day-to-day variation (something that cancels out in longer-term averages reducing long-term estimate variability). In the present assessment, after adopting the Box-Cox transformation (see 10.3.1), all estimated distributions exhibited improved statistical properties compared to the OIM-based distributions: in particular, the MM-based distributions had thinner tails.

Having 100 observations per participant is equivalent to having 100 cross-sections. The starting point of the analysis is one observed cross-section, the survey. The result is 100 simulated cross-sections based on the original data and the model structure. From each simulated cross-section arises one distribution and corresponding percentiles of interest. The distribution is weighted using demographic weights. Calendar weights are unnecessary, as the simulated data are for annual averages. All percentiles of interest are averaged and reported in the tables. Furthermore, for each percentile, the 5th lowest and the 5th highest values are taken. As there are 100 values altogether, the two values correspond to the 5th and 95th percentile, or 5% confidence interval for the considered percentile.

10.4.1 Transformations and their reversals

The assumption of residual normality is a strong assumption that is ill-suited for many empirically observed exposures. The dependent variable (the modelled compound) is transformed to make the fitted residuals not too divergent from normality. There are two commonly used transformation functions, the log transformation and the Box-Cox transformation. The former is easier to work with, but inflexible. The latter has a λ parameter that allows to adapt the transformation to the data. Note, that the Box-Cox transformation with $\lambda = 0$ is equivalent to the log transformation. Due to its flexibility, the Box-Cox transformation can produce a significantly better model fit. The Box-Cox transformation has been used in other studies of exposure. For example, SPADE: Statistical Program to Assess habitual Dietary Exposure developed by the RIVM, while adopting a different MM approach to their estimations, incorporates the Box-Cox transformation as the first step of the modelling procedure (Dekkers et al., 2014).

Some distributions cannot be transformed in such a way that residuals become approximately normal. For example, distributions with a sizeable probability mass on zero will have a strong deviation from normality even after transformation. For them, the chosen model approach cannot be used. This is the reason why MMs were not used to assess mean contribution from e.g. food groups or food items. In contrast, all considered measures for total exposures to PCDD/Fs and DL-PCBs were well-suited for Box-Cox transformation. The effect of transforming the modelled exposure is illustrated using the example of Q-Q plots for residuals of PCDD/Fs and DL-PCBs exposure (without fruits, vegetables, and potatoes), under LB for the VKM dataset for adults. Q-Q plots present quantiles of residuals versus quantiles of a normal distribution. If residuals are normal, all residuals will be on the diagonal line, where quantiles for residuals are equal to quantiles of the normal distribution. In Figure 10.4.1-1 below, the dashed lines represent the best fitting lines to the observed

quantile combinations. The line for untransformed exposure significantly diverges from the diagonal, while the residual values themselves exhibit significant deviation from the straight line. The residuals for the transformed exposure clearly have much better alignment with the diagonal, and, thus, greatly demished divergence from normality.

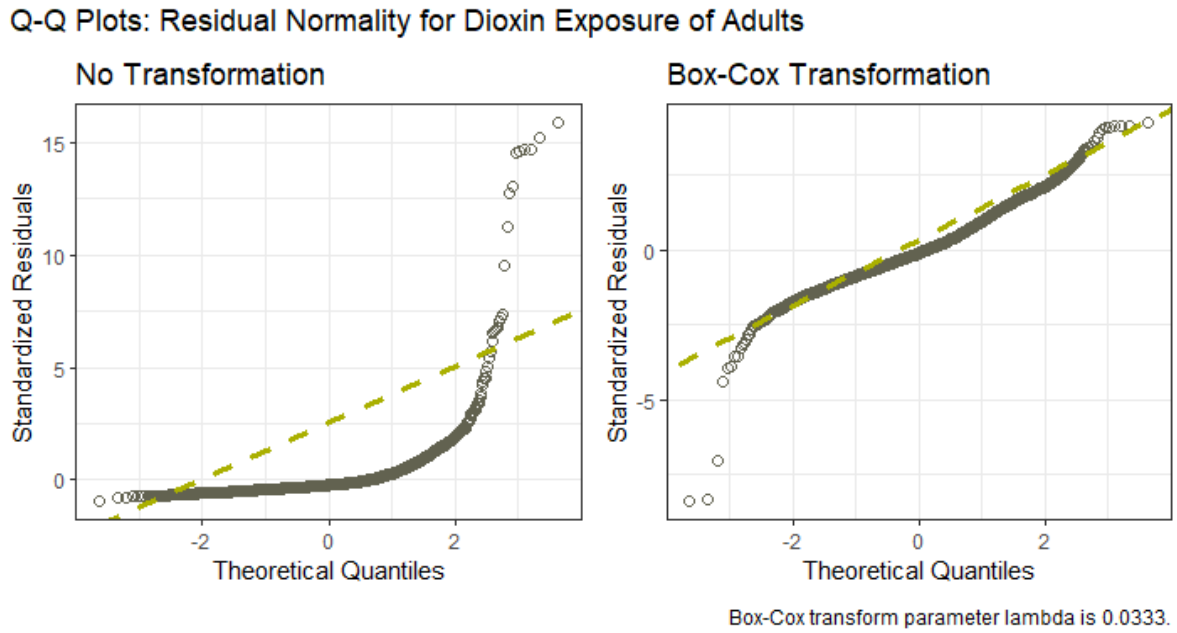

Figure 10.4.1-1. Q-Q plots for total PCDD/Fs and DL-PCBs exposure residuals (excluding fruits, vegetables, and potatoes), LB VKM dataset, for Norkost 3. The left subplot is for raw, untransformed exposure. The right subplot is for Box-Cox-transformed exposure using the optimal lambda found as per procedure in Chapter 10.4.2. In both cases, the dashed lines represent the best fitting lines to the residuals.

The Box-Cox transformation, $z = (y^{\lambda} - 1)/\lambda$, can be performed for all non-negative values. All exposure values are non-negative, and, thus, the transform is defined for all values in the database. After the transformed values are modeled and simulated, the transformation has to be reversed before the estimated distribution can be reported. The reversal is performed as follows: $y = (z\lambda + 1)^{1/\lambda}$. The reverse transformation is only defined for such z that $z\lambda + 1 \geq 0$ (assuming $1/\lambda$ is non-integer). For $\lambda > 0$, when simulated zs are lower, the function is undefined. Such values of the simulated daily exposure are mapped back to zero. In the present assessment, the optimal choice of λ was positive for all measures of exposures to PCDD/Fs and DL-PCBs.

10.4.2 Finding the optimal λ

The standard Box-Cox transformation is of the form $(y^{\lambda} - 1)/\lambda$. The regression, for example, of the form

$$\frac{Compound_i^{\lambda} - 1}{\lambda} = \alpha + \beta_1 Sex + \beta_2 Age + \beta_3 Education.High + \beta_4 Landsdel + \beta_5 Weekday + \beta_6 Month + \epsilon_i$$

is repeatedly run using the maximum likelihood estimation, for different values of λ. The process is implemented using the boxcox function of MASS package, v.7.3.54, in R. λ associated with the highest log-likelihood is selected and utilized in the corresponding MM.

11 Appendix II: Concentration data used for the scenarios

11.1 Crab

The ratio of total TEQ between brown crab meat (6.6 pg WHO_{1998}-TEQ/g ww) and white crab meat (1.31 pg WHO_{1998}-TEQ/g ww) was calculated based on mean concentration in 7 crabs sampled in 2004 (NIFES, 2004). The ratio was 11.6, which was rounded to 12. The mean concentration in brown crab meat from Norway is LB/UB 3.47/3.62 pg WHO_{2005}-TEQ/g ww (Table 3.1-2). The concentration in white crab meat was calculated to be 0.29/0.30 pg WHO_{2005}-TEQ/g ww, by dividing the concentration in brown meat by the ratio between brown and white meat. Then the concentration in whole crabs and filled crab shells was calculated based on the relative content of brown and white crab meat as described in 3.5.1.

11.2 Liver from livestock animals

VKM used the liver:meat concentration ratio for the sum of PCDD/Fs and DL-PCBS based on EFSA's concentration data (EFSA, 2018a) and the concentration in kidney tallow from Norwegian beef and pork (Table 11.2-1) to estimate PCDD/Fs and DL-PCB concentrations for Norwegian beef and pork liver. Both the LB and UB concentrations in beef and pork liver and meat was used to calculate the liver/meat ratio based on concentrations given in EFSA 2018 (Annex table 2A). For pork, the LB ratio between pork liver and pork meat was 0.86. The LB concentration in Norwegian pork kidney fat was 0.024 pg WHO_{2005}-TEQ/g lipid. This value was multiplied by the ratio of 0.86 to obtain the concentration of 0.021 pg WHO_{2005}-TEQ /g whole weight in Norwegian pork liver. Similar calculations were done for LB in pork and for UB and LB in cattle, as shown in Table 11.2-1.

Table 11.2-1. Concentrations of PCDD/Fs and DL-PCBs in beef and pork meat and liver (from EFSA, 2018a), and in kidney tallow from beef and pork from Norway, used as basis for calculation of concentration in liver from Norwegian beef and pork.

Food	n	Sum of PCDD/Fs and DL-PCBs		Data origin
		Mean LB	Mean UB	
Pork liver (pg WHO_{2005}-TEQ/g ww)	55	0.12	0.13	EFSA, 2018a
Pork meat (pg WHO_{2005}-TEQ/g fat)	459	0.139	0.236	EFSA, 2018a
Ratio between pork liver and meat, EFSA		0.86	0.55	
Norwegian pork kidney fat (pg WHO_{2005}-TEQ/g fat)	7	0.024	0.173	This report, table 3.1-2

Food	n	Sum of PCDD/Fs and DL-PCBs		Data origin
		Mean LB	Mean UB	
Calculated concentration in Norwegian pork liver (Norwegian pork meat x ratio) (pg WHO_{2005}-TEQ/g ww)		*0.024 x 0.86 = 0.021*	*0.173 x 0.55 = 0.095*	
Beef liver (pg WHO_{2005}-TEQ/g ww)	183	0.15	0.15	EFSA, 2018a
Cattle meat (pg WHO_{2005}-TEQ/g fat)	869	2.14	2.23	EFSA, 2018a
Ratio between beef liver and meat, EFSA		0.07	0.07	EFSA, 2018a
Norwegian beef kidney fat (pg WHO_{2005}-TEQ/g lipid)	19	0.306	0.387	This report, table 3.1-2
Calculated concentration in Norwegian beef liver (Norwegian pork meat x ratio) (pg WHO_{2005}-TEQ/g ww)		*0.021*	*0.027*	

11.3 Reindeer

In this report we did not have data on the PCDD/Fs and DL-PCBs in Norwegian reindeer liver. It is known that PCDD/Fs and DL-PCBs accumulates in the liver and is higher than in other animal tissues, even on a lipid-based concentration (EFSA, 2011). The concentration in the liver of Norwegian reindeer was estimated based on the measured ratio between liver and muscle from a Finnish study (Holma-Suutari et al., 2014). A study of reindeer from Russia was also considered (Makarov et al., 2021), but this study had only PCDD/F data and not DL-PCD data. In addition, the study had only approximate data on the lipid concentrations in the muscle and liver tissues, which may have considerable influence on the lipid-based concentrations of the PCDD/Fs. In the study by Holma-Suutari et al. (2014), muscle and liver samples from five reindeer hinds and two calves were analysed for PCDD/Fs and DL-PCBs. Mean and median ratio between liver and muscle of sum PCDD/Fs and DL-PCBs from the seven animals, based on lipid concentration, were 35 and 39 respectively, with a maximum and minimum ratio of 14 and 68, respectively (Table 10.3-1). The median ratio of 35 was selected to reduce the contribution of the highest and lowest values. The ratio between liver and muscle was estimated to be higher for PCDD/F compared to DL-PCBs, but did not reach the level of significance (paired two-sided Student t-test; $p = 0.07$), the median ratio of 35 which was calculated for the sum PCDD/F + DL-PCBs was therefore selected.

Table 10.3-1. Estimated ratio of PCDD/Fs and DL-PCBs between liver and muscle from reindeer, based on lipid concentration (from Holma-Suutari et al., 2014).

	Ratio PCDD/F	Ratio DL-PCB	Ratio PCDD/F + DL-PCB
Reindeer hind	61.8	60.5	61.1

	Ratio PCDD/F	Ratio DL-PCB	Ratio PCDD/F + DL-PCB
Reindeer hind	13.3	14.1	13.7
Reindeer hind	43.3	29	34.1
Reindeer hind	39.8	33.6	36.4
Reindeer hind	99.8	39.5	67.6
Reindeer calf	69.3	26.4	34.7
Reindeer calf	32.6	21.1	24.1
Mean	51	32	39
Median	43	29	35
Min	13	14	14
Max	100	61	68

12 Appendix III: Estimated exposure

Exposure estimates including potatoes, fruits and vegetables

An overview of the estimated exposure, including fruits, vegetables and potatoes, is shown in Tables 12-1 and 12-2.

Table 12-1. Estimated exposure to PCDD/Fs and the sum of PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week), with fruits, vegetables and potatoes, obtained using the VKM dataset (Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods), applying the mixed model approach for all population groups except for 1-and 2-year-olds, for which adjusted OIMs are shown.

Age group	Sum of PCDD/Fs and DL-PCBs				PCDD/Fs			
	Mean		95-percentile		Mean		95-percentile	
	LB	UB	LB	UB	LB	UB	LB	UB
Adults (18-70 years)	3.25	6.18	5.78	10.43	1.23	3.82	2.02	6.26
18-45 years (women)	2.97	6.04	5.15	10.02	1.16	3.86	1.92	6.25
13-year-olds	2.64	5.70	4.76	10.19	1.18	3.79	2.00	6.70
9-year-olds	3.69	8.32	6.15	13.43	1.67	5.62	2.66	9.09
4-year-olds	6.58	14.19	11.06	21.01	2.66	9.40	4.03	13.60
2-year-olds	8.79	16.76	17.66	31.77	3.34	10.47	6.37	19.46
1-year-olds	10.63	21.29	22.49	44.25	4.05	13.69	8.30	28.19

Table 12-2. Estimated exposure to PCDD/Fs and the sum of PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week) obtained using the EFSA dataset, applying the mixed model approach for all population groups except for 1-and 2-year-olds, for which adjusted OIMs are shown.

Age group	Sum of PCDD/Fs and DL-PCBs				PCDD/Fs			
	Mean		95-percentile		Mean		95-percentile	
	LB	UB	LB	UB	LB	UB	LB	UB
Adults (18-70 years)	6.94	9.43	12.04	15.66	2.49	4.55	4.17	7.35
18-45 years (women)	6.18	8.73	10.70	14.45	2.24	4.41	3.75	7.13
13-year-olds	6.87	9.53	12.05	16.45	2.43	4.61	4.18	7.91
9-year-olds	9.56	13.58	15.45	21.39	3.43	6.79	5.46	10.72
4-year-olds	15.72	22.41	24.85	33.58	5.46	11.18	8.12	16.17
2-year-olds	20.83	27.14	42.30	51.75	7.10	12.47	13.74	21.88
1-year-olds	24.06	32.16	54.73	67.16	7.98	15.00	16.80	29.76

An overview of the percent in the population with exposure above the TWI with potatoes, fruits, vegetables and potatoes included in the exposure assessment is shown in Tables 12-3 (PCDD/Fs and DL-PCBs) and 12-4 (PCDD/Fs).

Table 12-3. Comparison of the estimated exposure to the sum of PCDD/Fs and DL-PCBs, with fruits, vegetables and potatoes, obtained using the VKM dataset (Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods).

Age (years)	2	4	9	13	18-45 (women)	18-70
Participants above the TWI (LB)	100%	100%	93%	69%	80%	84%
Times above the TWI (mean LB)	4.4	3.3	1.9	1.3	1.5	1.6
Times above the TWI (P95 LB)	8.8	5.5	3.1	2.4	2.6	2.9
Participants above the TWI (UB)	100%	100%	100%	99%	100%	100%
Times above the TWI (mean UB)	8.4	7.1	4.2	2.9	3.0	3.1
Times above the TWI (P95 UB)	15.9	10.5	6.7	5.1	5.0	5.2

Table 12-4. Comparison of the estimated exposure to PCDD/Fs, including fruits, vegetables and potatoes, obtained using the VKM dataset (Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods)

Age (years)	2	4	9	13	18-45 (women)	18-70
Participants above the TWI (LB)	82%	81%	24%	5%	4%	5%
Times above the TWI (mean LB)	1.7	1.3	0.9	0.6	0.6	0.6
Times above the TWI (P95 LB)	3.2	2.0	1.3	1.0	1.0	1.0
Participants above the TWI (UB)	100%	100%	99%	91%	95%	95%
Times above the TWI (mean UB)	5.2	4.7	2.8	1.9	1.9	1.9
Times above the TWI (P95 UB)	9.7	6.8	4.6	3.4	3.1	3.1

12.1 OIMs

12.1.1 VKM dataset including fruits, vegetables and potatoes

12.1.1.1 PCDD/Fs and DL-PCBs (29 congeners)

The estimated exposure to PCDD/Fs and DL-PCBs is shown in Tables 12.1.1.1-1 (LB) and 12.1.1.1-2 (UB).

Table 12.1.1.1-1. The lower bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	10.54	7.50	3.33	5.98	8.98	12.99	22.32
2		1,413	8.73	5.25	3.40	5.36	7.62	10.64	17.83
4		399	6.88	4.60	2.65	4.17	5.74	8.34	14.01
9		636	3.88	2.45	1.35	2.33	3.26	4.65	8.34
13		687	2.79	2.08	0.84	1.58	2.24	3.30	6.49
18-45	F	466	2.98	3.46	0.72	1.31	2.10	3.48	7.59
18+		1,787	3.44	4.06	0.79	1.49	2.35	3.94	8.91

Table 12.1.1.1-2. The upper bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	21.01	13.27	7.26	12.49	18.08	25.93	43.96
2		1,413	16.62	8.23	7.53	11.21	14.97	19.93	31.62
4		399	14.60	6.40	7.49	10.50	13.51	17.31	25.78
9		636	8.58	4.06	3.37	5.83	7.89	10.28	15.86
13		687	5.91	3.36	2.05	3.56	5.15	7.40	12.22
18-45	F	466	5.91	4.40	1.88	3.47	4.88	7.09	12.47
18+		1,787	6.35	4.90	1.99	3.65	5.23	7.60	13.76

12.1.1.2 PCDD/Fs (17 congeners)

The estimated exposure to PCDD/Fs is shown in Tables 12.1.1.2-1 (LB) and 12.1.1.2-2 (UB**).**

Table 12.1.1.2-1. The lower bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	4.00	2.39	1.47	2.46	3.47	4.90	8.09
2		1,413	3.28	1.62	1.47	2.20	2.96	3.92	6.30
4		399	2.70	1.37	1.27	1.95	2.42	3.22	4.65
9		636	1.71	0.85	0.67	1.16	1.57	2.14	3.34
13		687	1.22	0.76	0.42	0.77	1.09	1.50	2.32
18-45	F	466	1.09	0.79	0.34	0.59	0.90	1.36	2.31
18+		1,787	1.27	1.49	0.37	0.66	0.98	1.47	2.78

Table 12.1.1.2-2. The upper bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	13.47	8.31	4.84	8.13	11.59	16.52	27.91
2		1,413	10.33	4.74	4.96	7.17	9.31	12.20	19.26
4		399	9.59	3.48	4.97	7.38	8.93	11.19	15.29
9		636	5.79	2.52	2.40	4.01	5.40	6.98	10.46
13		687	3.90	2.02	1.39	2.43	3.52	4.93	7.68

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
18-45	F	466	3.71	2.00	1.32	2.28	3.39	4.62	7.53
18+		1,787	3.89	2.39	1.33	2.39	3.46	4.82	7.54

12.1.2 VKM dataset without fruits, vegetables and potatoes

12.1.2.1 PCDD/Fs and DL-PCBs (29 congeners)

The estimated exposure to PCDD/Fs and DL-PCBs is shown in Tables 12.1.2.1-1 (LB) and 12.1.2.1-2 (UB)**.**

Table 12.1.2.1-1. The lower bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	7.23	6.03	2.22	4.03	5.99	8.83	15.90
2		1,413	7.12	4.71	2.62	4.25	6.13	8.65	14.70
4		399	6.17	4.42	2.26	3.62	5.08	7.62	13.01
9		636	3.44	2.36	1.17	1.97	2.79	4.12	8.02
13		687	2.48	2.04	0.73	1.33	1.95	2.89	6.06
18-45	F	466	2.54	3.38	0.51	0.96	1.61	2.91	7.06
18+		1,787	2.98	3.99	0.59	1.14	1.82	3.30	8.30

Table 12.1.2.1-2. The upper bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	12.16	7.71	4.30	7.61	10.69	14.80	24.23
2		1,413	12.32	6.33	5.33	8.19	11.25	14.75	22.63
4		399	11.18	5.59	5.27	7.70	10.01	13.17	21.26
9		636	6.83	3.52	2.57	4.51	6.07	8.34	13.32
13		687	4.86	3.06	1.61	2.88	4.15	5.91	10.69
18-45	F	466	4.26	4.04	1.24	2.22	3.30	4.87	9.99
18+		1,787	4.76	4.62	1.32	2.39	3.54	5.44	11.50

12.1.2.2 PCDD/Fs (17 congeners)

The estimated exposure to PCDD/Fs is shown in Tables 12.1.2.2-1 (LB) and 12.1.2.2-2 (UB**).**

Table 12.1.2.2-1. The lower bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	2.54	1.52	0.96	1.61	2.23	3.05	5.13
2		1,413	2.57	1.30	1.12	1.72	2.29	3.11	4.79
4		399	2.37	1.29	1.09	1.64	2.14	2.82	4.11
9		636	1.50	0.80	0.56	0.97	1.37	1.84	2.91
13		687	1.07	0.73	0.36	0.65	0.94	1.32	2.13
18-45	F	466	0.90	0.74	0.24	0.44	0.71	1.11	2.05
18+		1,787	1.06	1.46	0.27	0.50	0.77	1.21	2.50

Table 12.1.2.2-2. The upper bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	6.98	3.43	2.79	4.66	6.36	8.59	13.07
2		1,413	7.18	3.06	3.44	5.08	6.70	8.62	12.48
4		399	6.72	2.56	3.43	5.02	6.34	7.91	11.42
9		636	4.36	1.95	1.77	3.04	3.99	5.32	7.82
13		687	3.07	1.68	1.09	1.87	2.77	3.80	5.99
18-45	F	466	2.38	1.48	0.78	1.43	2.07	2.94	5.03
18+		1,787	2.62	2.04	0.86	1.52	2.20	3.09	5.61

12.1.3 EFSA dataset including fruits, vegetables and potatoes

12.1.3.1 PCDD/Fs and DL-PCBs (29 congeners)

The estimated exposure to PCDD/Fs and DL-PCBs is shown in Tables 12.1.3.1-1 (LB) and 12.1.3.1-2 (UB).

Table 12.1.3.1-1. The lower bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	23.82	18.25	6.17	12.05	19.52	29.81	54.04
2		1,413	20.74	11.40	8.13	13.01	18.55	25.35	41.43
4		399	16.13	7.96	6.80	10.62	14.32	20.23	30.27
9		636	9.93	5.58	3.71	6.30	8.76	12.17	19.89
13		687	7.16	4.51	2.24	4.27	6.10	8.57	15.56
18-45	F	466	6.12	5.68	1.72	2.94	4.70	7.19	15.18
18+		1,787	7.04	5.88	1.90	3.51	5.45	8.71	17.78

Table 12.1.3.1-2. The upper bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	31.79	20.92	10.36	18.24	27.19	39.58	66.37

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
2		1,413	26.97	13.11	12.12	18.30	24.53	32.83	51.13
4		399	22.93	9.58	11.27	16.27	20.75	27.54	39.33
9		636	14.04	6.61	5.51	9.74	13.04	16.94	25.79
13		687	9.85	5.36	3.39	6.14	8.86	12.15	19.36
18-45	F	466	8.66	6.39	2.98	5.15	7.16	10.46	18.56
18+		1,787	9.59	6.55	3.17	5.54	8.11	11.59	21.57

12.1.3.2 PCDD/Fs (17 congeners)

The estimated exposure to PCDD/Fs is shown in Tables 12.1.3.2-1 (LB) and 12.1.3.2-2 (UB**).**

Table 12.1.3.2-1. The lower bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	7.89	5.35	2.52	4.47	6.66	9.78	16.63
2		1,413	7.04	3.42	3.20	4.74	6.37	8.56	13.35
4		399	5.60	2.37	2.70	3.93	5.07	6.88	10.05
9		636	3.54	1.78	1.35	2.35	3.19	4.30	6.60
13		687	2.51	1.45	0.85	1.55	2.22	3.04	5.13
18-45	F	466	2.16	1.57	0.67	1.17	1.74	2.62	4.82
18+		1,787	2.50	1.86	0.77	1.35	1.98	3.02	5.97

Table 12.1.3.2-2. The upper bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	14.81	8.16	5.72	9.45	13.16	18.03	29.44
2		1,413	12.33	5.14	6.13	8.90	11.35	14.70	21.88
4		399	11.39	4.04	6.21	8.56	10.77	13.23	18.45
9		636	6.98	2.94	3.05	4.93	6.58	8.42	12.36
13		687	4.75	2.35	1.80	3.01	4.38	5.91	8.99
18-45	F	466	4.32	2.36	1.59	2.71	3.87	5.35	8.33
18+		1,787	4.63	2.55	1.67	2.87	4.11	5.69	9.43

12.1.4 EFSA dataset without fruits, vegetables and potatoes

12.1.4.1 PCDD/Fs and DL-PCBs (29 congeners)

The estimated exposure to PCDD/Fs and DL-PCBs is shown in Tables 12.1.4.1-1 (LB) and 12.1.4.1-2 (UB).

Table 12.1.4.1-1. The lower bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	15.97	11.06	4.87	8.93	13.83	20.26	34.31
2		1,413	17.85	9.36	7.27	11.47	16.20	21.94	34.02
4		399	15.42	7.76	6.53	10.07	13.77	19.43	29.72
9		636	9.50	5.51	3.56	5.91	8.24	11.59	19.64
13		687	6.85	4.46	2.07	3.97	5.79	8.28	15.24
18-45	F	466	5.68	5.62	1.47	2.60	4.19	6.61	15.09
18+		1,787	6.58	5.81	1.66	3.12	5.01	8.04	17.31

Table 12.1.4.1-2. The upper bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	20.19	12.24	6.78	12.40	17.85	25.11	40.92
2		1,413	21.89	10.47	9.75	14.83	20.16	26.49	39.99
4		399	19.51	8.67	9.38	13.50	17.85	24.56	34.95
9		636	12.29	6.16	4.72	8.32	11.18	15.00	23.34
13		687	8.81	5.08	2.97	5.37	7.82	10.89	18.02
18-45	F	466	7.01	6.11	2.14	3.56	5.58	8.28	16.47
18+		1,787	8.00	6.28	2.32	4.14	6.34	9.67	19.49

12.1.4.2 PCDD/Fs (17 congeners)

The estimated exposure to PCDD/Fs is shown in Tables 12.1.4.2-1 (LB) and 12.1.4.2-2 (UB).

Table 12.1.4.2-1. The lower bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	5.26	3.01	1.89	3.32	4.69	6.51	10.55
2		1,413	6.00	2.73	2.73	4.14	5.55	7.30	10.71
4		399	5.27	2.28	2.60	3.65	4.77	6.54	9.56
9		636	3.33	1.74	1.23	2.16	2.93	4.03	6.47
13		687	2.37	1.42	0.80	1.43	2.07	2.86	4.90
18-45	F	466	1.97	1.54	0.56	1.01	1.58	2.36	4.68
18+		1,787	2.29	1.82	0.64	1.16	1.78	2.75	5.59

Table 12.1.4.2-2. The upper bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	8.70	4.19	3.47	5.91	7.98	10.70	16.17
2		1,413	9.29	3.82	4.68	6.74	8.70	11.07	15.99
4		399	8.52	3.09	4.46	6.39	7.95	10.13	14.02

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
9		636	5.54	2.42	2.33	3.94	5.12	6.68	9.91
13		687	3.91	2.02	1.38	2.50	3.60	4.85	7.41
18-45	F	466	2.99	1.93	1.01	1.76	2.54	3.63	6.23
18+		1,787	3.36	2.19	1.13	1.94	2.86	4.09	7.51

12.2 W-OIMs

12.2.1 VKM dataset including fruits, vegetables and potatoes

12.2.1.1 PCDD/Fs and DL-PCBs (29 congeners)

The estimated exposure to PCDD/Fs and DL-PCBs is shown in Tables 12.2.1.1-1 (LB) and 12.2.1.1-2 (UB)**.**

Table 12.2.1.1-1. The lower bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	10.63	7.27	3.33	6.14	9.12	13.13	22.49
2		1,413	8.79	5.08	3.43	5.54	7.71	10.77	17.66

Table 12.2.1.1-2. The upper bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	21.29	13.04	7.41	12.88	18.45	26.30	44.25
2		1,413	16.76	8.10	7.64	11.40	15.12	20.00	31.77

12.2.1.2 PCDD/Fs (17 congeners)

The estimated exposure to PCDD/Fs is shown in Tables 12.2.1.2-1 (LB) and 12.2.1.2-2 (UB).

Table 12.2.1.2-1. The lower bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	4.05	2.35	1.50	2.50	3.53	5.02	8.30
2		1,413	3.34	1.61	1.49	2.28	3.06	3.99	6.37

Table 12.2.1.2-2. The upper bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	13.69	8.23	4.91	8.30	11.88	16.73	28.19

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
2		1,413	10.47	4.75	5.01	7.27	9.39	12.37	19.46

12.2.2 VKM dataset without fruits, vegetables and potatoes

12.2.2.1 PCDD/Fs and DL-PCBs (29 congeners)

The estimated exposure to PCDD/Fs and DL-PCBs is shown in Tables 12.2.2.1-1 (LB) and 12.2.2.1-2 (UB).

Table 12.2.2.1-1. The lower bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	7.25	5.76	2.25	4.08	6.01	8.89	15.75
2		1,413	7.11	4.53	2.65	4.30	6.19	8.61	14.54

Table 12.2.2.1-2. The upper bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	12.24	7.45	4.34	7.76	10.86	14.94	24.23
2		1,413	12.34	6.23	5.38	8.25	11.28	14.83	22.43

12.2.2.2 PCDD/Fs (17 congeners)

The estimated exposure to PCDD/Fs is shown in Tables 12.2.2.2-1 (LB) and 12.2.2.2-2 (UB)**.**

Table 12.2.2.2-1. The lower bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	2.56	1.47	0.97	1.64	2.27	3.10	5.13
2		1,413	2.60	1.29	1.15	1.78	2.34	3.12	4.76

Table 12.2.2.2-2. The upper bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	7.06	3.38	2.82	4.73	6.47	8.66	13.19
2		1,413	7.24	3.12	3.49	5.13	6.75	8.66	12.48

12.2.3 EFSA dataset including fruits, vegetables and potatoes

12.2.3.1 PCDD/Fs and DL-PCBs (29 congeners)

The estimated exposure to PCDD/Fs and DL-PCBs is shown in Tables 12.2.3.1-1 (LB) and 12.2.3.1-2 (UB).

Table 12.2.3.1-1. The lower bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	0.05	0.25	0.5	0.75	0.95
1		1,957	24.06	17.97	6.11	12.18	20.07	30.00	54.73
2		1,413	20.83	11.21	8.09	13.31	18.67	25.43	42.30

Table 12.2.3.1-2. The upper bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	0.05	0.25	0.5	0.75	0.95
1		1,957	32.16	20.63	10.42	18.61	27.63	39.75	67.16
2		1,413	27.14	12.98	12.12	18.61	24.84	33.07	51.75

12.2.3.2 PCDD/Fs (17 congeners)

The estimated exposure to PCDD/Fs is shown in Tables 12.2.3.2-1 (LB) and 12.2.3.2-2 (UB).

Table 12.2.3.2-1. The lower bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	0.05	0.25	0.5	0.75	0.95
1		1,957	7.98	5.29	2.52	4.55	6.76	9.88	16.80
2		1,413	7.10	3.41	3.19	4.88	6.43	8.67	13.74

Table 12.2.3.2-2. The upper bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	0.05	0.25	0.5	0.75	0.95
1		1,957	15.00	8.08	5.82	9.62	13.47	18.21	29.76
2		1,413	12.47	5.19	6.22	9.07	11.40	14.91	21.88

12.2.4 EFSA dataset without fruits, vegetables and potatoes

12.2.4.1 PCDD/Fs and DL-PCBs (29 congeners)

The estimated exposure to PCDD/Fs and DL-PCBs is shown in Tables 12.2.4.1-1 (LB) and 12.2.4.1-2 (UB).

Table 12.2.4.1-1. The lower bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	15.95	10.73	4.89	9.04	13.90	20.36	33.54
2		1,413	17.82	9.26	7.25	11.72	16.15	21.83	34.02

Table 12.2.4.1-2. The upper bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P5	P25	P50	P75	P95
1		1,957	20.25	11.91	6.87	12.54	18.02	25.35	40.67
2		1,413	21.91	10.44	9.74	15.12	20.10	26.39	39.99

12.2.4.2 PCDD/Fs (17 congeners)

The estimated exposure to PCDD/Fs is shown in Tables 12.2.4.2-1 (LB) and 12.2.4.2-2 (UB)**.**

Table 12.2.4.2-1. The lower bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	0.05	0.25	0.5	0.75	0.95
1		1,957	5.27	2.93	1.91	3.36	4.70	6.60	10.43
2		1,413	6.03	2.76	2.73	4.24	5.54	7.30	10.77

Table 12.2.4.2-2. The upper bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	0.05	0.25	0.5	0.75	0.95
1		1,957	8.77	4.12	3.52	6.00	8.14	10.82	16.18
2		1,413	9.36	3.91	4.68	6.87	8.71	11.10	16.08

12.3 Mixed model

12.3.1 VKM dataset including fruits, vegetables and potatoes

12.3.1.1 PCDD/Fs and DL-PCBs (29 congeners)

The estimated exposure to PCDD/Fs and DL-PCBs is shown in Tables 12.3.1.1-1 (LB) and 12.3.1.1-2 (UB).

Table 12.3.1.1-1. The lower bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
4		399	6.58	2.40	3.49	4.87	6.21	7.86	11.06

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
9		636	3.69	1.34	1.90	2.74	3.49	4.44	6.15
13		687	2.64	1.12	1.23	1.85	2.45	3.22	4.76
18-45	F	466	2.97	1.16	1.47	2.14	2.79	3.58	5.15
18+		1,785	3.25	1.34	1.54	2.30	3.02	3.95	5.78

Table 12.3.1.1-2. The upper bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
4		399	14.19	3.81	8.79	11.48	13.78	16.47	21.01
9		636	8.32	2.83	4.37	6.29	7.98	9.98	13.43
13		687	5.70	2.39	2.64	4.00	5.30	6.96	10.19
18-45	F	466	6.04	2.15	3.15	4.50	5.76	7.25	10.02
18+		1,785	6.18	2.29	3.11	4.54	5.84	7.45	10.43

12.3.1.2 PCDD/Fs (17 congeners)

The estimated exposure to PCDD/Fs is shown in Tables 12.3.1.2-1 (LB) and 12.3.1.2-2 (UB)**.**

Table 12.3.1.2-1. The lower bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
4		399	2.66	0.76	1.61	2.12	2.58	3.11	4.03
9		636	1.67	0.55	0.90	1.28	1.60	1.99	2.66
13		687	1.18	0.44	0.58	0.87	1.12	1.43	2.00
18-45	F	466	1.16	0.41	0.62	0.87	1.11	1.39	1.92
18+		1,785	1.23	0.43	0.65	0.93	1.17	1.47	2.02

Table 12.3.1.2-2. The upper bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
4		399	9.40	2.36	5.98	7.73	9.18	10.85	13.60
9		636	5.62	1.92	2.91	4.25	5.41	6.77	9.09
13		687	3.79	1.56	1.75	2.68	3.54	4.63	6.70
18-45	F	466	3.86	1.30	2.07	2.92	3.70	4.61	6.25
18+		1,785	3.82	1.33	1.99	2.87	3.64	4.58	6.26

12.3.2 VKM dataset without fruits, vegetables and potatoes

12.3.2.1 PCDD/Fs and DL-PCBs (29 congeners)

The estimated exposure to PCDD/Fs and DL-PCBs is shown in Tables 12.3.2.1-1 (LB) and 12.3.2.1-2 (UB)**.**

Table 12.3.2.1-1. The lower bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
4		399	5.90	2.14	3.07	4.37	5.58	7.08	9.88
9		636	3.24	1.27	1.58	2.34	3.03	3.92	5.58
13		687	2.32	1.04	1.03	1.58	2.12	2.84	4.28
18-45	F	466	2.54	1.16	1.12	1.71	2.32	3.11	4.75
18+		1,785	2.78	1.27	1.22	1.89	2.54	3.41	5.17

Table 12.3.2.1-2. The upper bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
4		399	10.91	3.02	6.67	8.78	10.59	12.71	16.33
9		636	6.61	2.28	3.49	5.00	6.30	7.92	10.78
13		687	4.67	2.06	2.11	3.21	4.28	5.70	8.52
18-45	F	466	4.40	1.64	2.25	3.22	4.16	5.30	7.47
18+		1,785	4.62	1.74	2.32	3.38	4.35	5.56	7.88

12.3.2.2 PCDD/Fs (17 congeners)

The estimated exposure to PCDD/Fs is shown in Tables 12.3.2.2-1 (LB) and 12.3.2.2-2 (UB)**.**

Table 12.3.2.2-1. The lower bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
4		399	2.33	0.67	1.40	1.85	2.26	2.72	3.55
9		636	1.46	0.51	0.76	1.10	1.39	1.75	2.39
13		687	1.04	0.41	0.50	0.75	0.98	1.26	1.79
18-45	F	466	0.97	0.38	0.48	0.70	0.91	1.18	1.69
18+		1,785	1.02	0.38	0.52	0.75	0.96	1.22	1.71

Table 12.3.2.2-2. The upper bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
4		399	6.67	1.62	4.32	5.53	6.54	7.68	9.53
9		636	4.24	1.38	2.30	3.25	4.08	5.05	6.74
13		687	2.98	1.24	1.38	2.10	2.78	3.63	5.28

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
18-45	F	466	2.55	0.87	1.38	1.93	2.44	3.05	4.15
18+		1,785	2.61	0.89	1.39	1.97	2.49	3.11	4.26

12.3.3 EFSA dataset including fruits, vegetables and potatoes

12.3.3.1 PCDD/Fs and DL-PCBs (29 congeners)

The estimated exposure to PCDD/Fs and DL-PCBs is shown in Tables 12.3.3.1-1 (LB) and 12.3.3.1-2 (UB)**.**

Table 12.3.3.1-1. The lower bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
4		399	15.72	5.06	8.79	12.11	15.08	18.60	24.85
9		636	9.56	3.23	5.06	7.26	9.17	11.45	15.45
13		687	6.87	2.78	3.22	4.89	6.45	8.39	12.05
18-45	F	466	6.18	2.43	3.01	4.44	5.82	7.52	10.70
18+		1,785	6.94	2.74	3.32	4.98	6.50	8.42	12.04

Table 12.3.3.1-2. The upper bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
4		399	22.41	6.18	13.72	18.01	21.76	26.09	33.58
9		636	13.58	4.34	7.42	10.47	13.11	16.16	21.39
13		687	9.53	3.71	4.60	6.89	8.98	11.56	16.45
18-45	F	466	8.73	3.09	4.53	6.50	8.32	10.48	14.45
18+		1,785	9.43	3.36	4.86	7.02	8.95	11.30	15.66

12.3.3.2 PCDD/Fs (17 congeners)

The estimated exposure to PCDD/Fs is shown in Tables 12.3.3.2-1 (LB) and 12.3.3.2-2 (UB)**.**

Table 12.3.3.2-1. The lower bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
4		399	5.46	1.48	3.39	4.41	5.30	6.34	8.12
9		636	3.43	1.12	1.89	2.64	3.29	4.08	5.46
13		687	2.43	0.95	1.17	1.75	2.29	2.95	4.18
18-45	F	466	2.24	0.81	1.15	1.66	2.12	2.69	3.75
18+		1,785	2.49	0.91	1.27	1.84	2.36	2.99	4.17

Table 12.3.3.2-2. The upper bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
4		399	11.18	2.80	7.14	9.20	10.95	12.89	16.17
9		636	6.79	2.19	3.66	5.24	6.56	8.10	10.72
13		687	4.61	1.79	2.21	3.33	4.35	5.61	7.91
18-45	F	466	4.41	1.49	2.35	3.34	4.24	5.28	7.13
18+		1,785	4.55	1.53	2.41	3.45	4.36	5.44	7.35

12.3.4 EFSA dataset without fruits, vegetables and potatoes

12.3.4.1 PCDD/Fs and DL-PCBs (29 congeners)

The estimated exposure to PCDD/Fs and DL-PCBs is shown in Tables 12.3.4.1-1 (LB) and 12.3.4.1-2 (UB)**.**

Table 12.3.4.1-1. The lower bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
4		399	15.07	4.82	8.41	11.63	14.46	17.86	23.83
9		636	9.09	3.13	4.76	6.86	8.69	10.91	14.77
13		687	6.59	2.74	3.01	4.64	6.16	8.07	11.67
18-45	F	466	5.77	2.43	2.65	4.03	5.38	7.07	10.34
18+		1,785	6.49	2.67	2.99	4.58	6.06	7.91	11.48

Table 12.3.4.1-2. The upper bound estimated exposure to PCDD/Fs and DL-PCBs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
4		399	19.20	5.39	11.60	15.40	18.61	22.39	28.84
9		636	11.87	3.84	6.51	9.12	11.40	14.13	18.80
13		687	8.51	3.36	4.11	6.11	7.99	10.32	14.75
18-45	F	466	7.19	2.84	3.48	5.15	6.75	8.74	12.49
18+		1,785	7.97	3.08	3.86	5.76	7.50	9.66	13.69

12.3.4.2 PCDD/Fs (17 congeners)

The estimated exposure to PCDD/Fs is shown in Tables 12.3.4.2-1 (LB) and 12.3.4.2-2 (UB)**.**

Table 12.3.4.2-1. The lower bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
4		399	5.15	1.38	3.19	4.18	5.01	5.98	7.58
9		636	3.23	1.07	1.75	2.47	3.09	3.85	5.18
13		687	2.28	0.91	1.07	1.64	2.15	2.79	3.96
18-45	F	466	2.05	0.81	1.00	1.47	1.93	2.49	3.57
18+		1,785	2.28	0.87	1.12	1.66	2.15	2.76	3.89

Table 12.3.4.2-2. The upper bound estimated exposure to PCDD/Fs (pg $TEQ_{WHO-2005}$/kg bw/week).

Age	Sex	N	Mean	SD	P05	P25	P50	P75	P95
4		399	8.39	2.00	5.47	6.98	8.22	9.61	11.88
9		636	5.40	1.70	3.00	4.19	5.20	6.41	8.48
13		687	3.79	1.49	1.81	2.73	3.57	4.60	6.54
18-45	F	466	3.14	1.12	1.63	2.33	2.98	3.77	5.21
18+		1,785	3.37	1.17	1.76	2.53	3.22	4.04	5.53

12.4 Contribution from different food groups to PCDD/F and total PCDD/F and DL-PCB exposure

The contribution from different food groups and marine oil supplements to the total PCDD/Fs and DL-PCBs exposure was calculated. For the food groups' fish and meat, contributions from various sources within the groups have also been calculated. The results for the sum of PCDD/Fs and the sum of PCDD/Fs and DL-PCBs for individual age groups, in pg WHO_{2005}-TEQ/kg bw per week, are shown in 13.4.1 to 13.4.6.

12.4.1 Adults (18-70-year-olds)

The contribution from different food groups and marine oil supplements to the total PCDD/Fs and DL-PCBs for adults (18-70-year-olds) are shown in Table 12.4.1-1 to 12.4.1-3. The results for the sub-group women 18-45-year-olds are shown in Chapter 12.4.1.1.

Table 12.4.1-1. Contribution from different food groups and marine oil supplements to mean total PCDD/F and DL-PCB exposure of adults (pg WHO_{2005}-TEQ/kg bw per week).

Food groups	**Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)**			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Fish	0.33	0.75	1.44	1.86
Shellfish	0.13	0.14	0.25	0.26
Meat	0.18	0.33	0.41	0.57
Milk and other dairy	0.16	0.63	0.40	0.93

Food groups	Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Egg	0.04	0.13	0.07	0.17
Grain and grain products	0.06	0.14	0.07	0.16
Fruit and vegetables	0.21	1.27	0.46	1.60
Other food groups	0.15	0.47	0.19	0.63
Marine oils (supplement)	0.01	0.04	0.15	0.18
Intake based on EFSA dataset[b]				
Fish	0.88	1.12	2.95	3.37
Shellfish	0.07	0.08	0.15	0.16
Meat	0.41	0.55	1.25	1.41
Milk and other dairy	0.50	0.75	1.25	1.50
Egg	0.14	0.17	0.33	0.37
Grain and grain products	0.06	0.14	0.07	0.16
Fruit and vegetables	0.21	1.27	0.46	1.60
Other food groups	0.15	0.47	0.19	0.64
Marine oils (supplement)	0.07	0.08	0.38	0.39

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a).

Table 12.4.1-2. Contribution from fish and seafood to mean total PCDD/F and DL-PCB exposure of adults (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Lean fish	0.04	0.14	0.20	0.30
Fatty fish, sum	0.26	0.56	1.03	1.33
Fatty fish: salmon/trout	*0.09*	*0.28*	*0.44*	*0.63*
Fatty fish: mackerel	*0.09*	*0.16*	*0.35*	*0.42*
Fatty fish: other[c]	*0.08*	*0.12*	*0.24*	*0.28*

Food groups	Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
Roe and liver	0.04	0.05	0.21	0.22
Shellfish	0.13	0.14	0.25	0.26
Intake based on EFSA dataset[b]				
Lean fish	0.13	0.25	0.57	0.84
Fatty fish, sum	0.68	0.78	2.06	2.18
Fatty fish: salmon/trout	*0.31*	*0.37*	*1.00*	*1.07*
Fatty fish: mackerel	*0.16*	*0.18*	*0.57*	*0.61*
Fatty fish: other[c]	*0.21*	*0.23*	*0.49*	*0.51*
Roe and liver	0.07	0.09	0.33	0.35
Shellfish	0.07	0.08	0.15	0.16

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a). [c] Other fatty fish species are halibut, wild and fresh-water trout, summer and winter herring, and char.

Table 12.4.1-3. Contribution from meat to mean total PCDD/F and DL-PCB exposure of adults (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	LB	LB	LB
Intake based on VKM dataset[a]				
Meat, excluding liver pâté	0.18	0.33	0.41	0.57
Liver pâté	0.01	0.01	0.01	0.01
Intake based on EFSA dataset[b]				
Meat, excluding liver pâté	0.41	0.55	1.25	1.41
Liver pâté	0.02	0.02	0.02	0.03

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a).

12.4.1.1 Women (18-45 years)

The contribution from different food groups and marine oil supplements to the total PCDD/Fs and DL-PCBs for women aged 18-45-year-olds are shown in Table 12.4.1.1-1 to 12.4.1.1-3.

Table 12.4.1.1-1. Contribution from different food groups and marine oil supplements to mean total PCDD/F and DL-PCB exposure of women in the age group 18-45 years (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	Observed individual means (pg WHO_{2005}-TEQ /kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Fish	0.27	0.63	1.24	1.60
Shellfish	0.04	0.04	0.08	0.09
Meat	0.17	0.29	0.36	0.50
Milk and other dairy	0.16	0.61	0.39	0.91
Egg	0.04	0.12	0.06	0.15
Grain and grain products	0.06	0.14	0.08	0.16
Fruit and vegetables	0.20	1.33	0.44	1.65
Other food groups	0.16	0.52	0.21	0.71
Marine oils (supplement)	0.01	0.03	0.13	0.15
Intake based on EFSA dataset[b]				
Fish	0.67	0.86	2.42	2.71
Shellfish	0.04	0.04	0.08	0.09
Meat	0.36	0.48	1.11	1.25
Milk and other dairy	0.49	0.74	1.22	1.48
Egg	0.13	0.16	0.30	0.33
Grain and grain products	0.06	0.14	0.08	0.16
Fruit and vegetables	0.20	1.33	0.44	1.65
Other food groups	0.16	0.52	0.21	0.71
Marine oils (supplement)	0.05	0.06	0.28	0.28

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a).

Table 12.4.1.1-2. Contribution from fish and seafood to mean total PCDD/F and DL-PCB exposure of women in the age group 18-45 years (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Lean fish	0.04	0.11	0.16	0.24
Fatty fish, sum	0.20	0.47	0.87	1.15

Food groups	Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Fatty fish: salmon/ trout	*0.08*	*0.25*	*0.40*	*0.58*
Fatty fish: mackerel	*0.09*	*0.16*	*0.35*	*0.42*
Fatty fish: other[c]	*0.03*	*0.06*	*0.12*	*0.15*
Roe and liver	0.03	0.04	0.20	0.22
Shellfish	0.04	0.04	0.08	0.09
Intake based on EFSA dataset[b]				
Lean fish	0.10	0.17	0.41	0.59
Fatty fish, sum	0.52	0.61	1.71	1.82
Fatty fish: salmon/ trout	*0.28*	*0.34*	*0.91*	*0.98*
Fatty fish: mackerel	*0.16*	*0.18*	*0.57*	*0.61*
Fatty fish: other[c]	*0.08*	*0.09*	*0.22*	*0.23*
Roe and liver	0.06	0.07	0.30	0.31
Shellfish	0.04	0.04	0.08	0.09

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a). [c] Other fatty fish species are halibut, wild and fresh-water trout, summer and winter herring, and char.

Table 12.4.1.1-3. Contribution from meat to mean total PCDD/F and DL-PCB exposure of women in the age group 18-45 years (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Meat, excluding liver pâté	0.17	0.29	0.36	0.50
Liver pâté	0.01	0.01	0.01	0.01
Intake based on EFSA dataset[b]				
Meat, excluding liver pâté	0.36	0.48	1.11	1.25
Liver pâté	0.02	0.03	0.02	0.03

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a).

12.4.2 Thirteen-year-olds

The contribution from different food groups and marine oil supplements to the total PCDD/Fs and DL-PCBs for thirteen-year-olds are shown in Table 12.4.2-1 to 12.4.2-3.

Table 12.4.2-1. Contribution from different food groups and marine oil supplements to mean total PCDD/F and DL-PCB exposure of 13-year-olds (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	**Observed individual means** (**pg WHO_{2005}-TEQ**/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Fish	0.14	0.44	0.70	1.00
Shellfish	0.04	0.04	0.07	0.07
Meat	0.28	0.47	0.56	0.78
Milk and other dairy	0.19	0.76	0.48	1.13
Egg	0.03	0.11	0.06	0.14
Grain and grain products	0.13	0.32	0.19	0.38
Fruit and vegetables	0.14	0.83	0.31	1.05
Other food groups	0.25	0.92	0.35	1.28
Marine oils (supplement)	0.00	0.02	0.07	0.09
Intake based on EFSA dataset[b]				
Fish	0.48	0.62	1.64	1.88
Shellfish	0.02	0.02	0.03	0.03
Meat	0.76	0.95	2.62	2.83
Milk and other dairy	0.58	0.90	1.53	1.87
Egg	0.12	0.15	0.28	0.31
Grain and grain products	0.13	0.32	0.19	0.38
Fruit and vegetables	0.14	0.83	0.31	1.05
Other food groups	0.24	0.92	0.34	1.28
Marine oils (supplement)	0.04	0.05	0.23	0.23

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a).

Table 12.4.2-2. Contribution from fish and seafood to mean total PCDD/F and DL-PCB exposure of 13-year-olds (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Lean fish	0.00	0.05	0.04	0.08
Fatty fish, sum	0.14	0.39	0.65	0.90
Fatty fish: salmon/ trout	*0.11*	*0.33*	*0.54*	*0.77*
Fatty fish: mackerel	*0.03*	*0.05*	*0.11*	*0.13*
Fatty fish: other[c]	*0.00*	*0.00*	*0.00*	*0.00*
Roe and liver	0.00	0.00	0.01	0.01
Shellfish	0.04	0.04	0.07	0.07
Intake based on EFSA dataset[b]				
Lean fish	0.05	0.10	0.22	0.35
Fatty fish, sum	0.43	0.51	1.39	1.49
Fatty fish: salmon/ trout	*0.38*	*0.45*	*1.21*	*1.30*
Fatty fish: mackerel	*0.05*	*0.06*	*0.18*	*0.19*
Fatty fish: other[c]	*0.00*	*0.00*	*0.00*	*0.00*
Roe and liver	0.01	0.01	0.02	0.03
Shellfish	0.02	0.02	0.03	0.03

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a). [c] Other fatty fish species are halibut, wild and fresh-water trout, summer and winter herring, and char.

Table 12.4.2-3. Contribution from meat to mean total PCDD/F and DL-PCB exposure of 13-year-olds (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Meat, excluding liver pâté	0.28	0.47	0.56	0.78
Liver pâté	0.01	0.01	0.01	0.01
Intake based on EFSA dataset[b]				
Meat, excluding liver pâté	0.76	0.95	2.62	2.83
Liver pâté	0.03	0.04	0.03	0.04

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a).

12.4.3 Nine-year-olds

The contribution from different food groups and marine oil supplements to the total PCDD/Fs and DL-PCBs for nine-year-olds are shown in Table 12.4.3-1 to 12.4.3-3.

Table 12.4.3-1. Contribution from different food groups and marine oil supplements to mean total PCDD/F and DL-PCB exposure of 9-year-olds (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	**Observed individual means** (**pg WHO_{2005}-TEQ**/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Fish	0.21	0.61	0.98	1.39
Shellfish	0.02	0.02	0.05	0.05
Meat	0.35	0.61	0.70	1.00
Milk and other dairy	0.29	1.11	0.71	1.65
Egg	0.05	0.17	0.09	0.22
Grain and grain products	0.21	0.48	0.29	0.58
Fruit and vegetables	0.21	1.43	0.43	1.75
Other food groups	0.37	1.30	0.49	1.78
Marine oils (supplement)	0.01	0.04	0.14	0.17
Intake based on EFSA dataset[b]				
Fish	0.61	0.8	2.28	2.63
Shellfish	0.06	0.08	0.03	0.04
Meat	0.98	1.24	3.32	3.61
Milk and other dairy	0.85	1.33	2.25	2.75
Egg	0.18	0.22	0.43	0.48
Grain and grain products	0.21	0.48	0.29	0.58
Fruit and vegetables	0.21	1.43	0.43	1.75
Other food groups	0.36	1.30	0.47	1.77
Marine oils (supplement)	0.08	0.09	0.42	0.43

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a).

Table 12.4.3-2. Contribution from fish and seafood to mean total PCDD/F and DL-PCB exposure of 9-year-olds (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	**Observed individual means** (**pg WHO_{2005}-TEQ**/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Lean fish	0.01	0.09	0.08	0.16
Fatty fish, sum	0.19	0.52	0.88	1.21
Fatty fish: salmon/ trout	*0.14*	*0.42*	*0.68*	*0.97*
Fatty fish: mackerel	*0.05*	*0.09*	*0.20*	*0.24*
Fatty fish: other[c]	*0.00*	*0.00*	*0.00*	*0.00*
Roe and liver	0.01	0.01	0.02	0.02
Shellfish	0.02	0.03	0.05	0.05
Intake based on EFSA dataset[b]				
Lean fish	0.08	0.17	0.37	0.60
Fatty fish, sum	0.56	0.67	1.85	1.98
Fatty fish: salmon/ trout	*0.48*	*0.57*	*1.53*	*1.64*
Fatty fish: mackerel	*0.09*	*0.10*	*0.33*	*0.35*
Fatty fish: other[c]	*0.00*	*0.00*	*0.00*	*0.00*
Roe and liver	0.01	0.02	0.05	0.06
Shellfish	0,02	0,02	0,03	0,04

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a). [c] Other fatty fish species are halibut, wild and fresh-water trout, summer and winter herring, and char.

Table 12.4.3-3. Contribution from meat to mean total PCDD/F and DL-PCB exposure of 9-year-olds (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	**Observed individual means** (**pg WHO_{2005}-TEQ**/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Meat, excluding liver pâté	0.35	0.61	0.70	1.00
Liver pâté	0.02	0.02	0.02	0.02
Intake based on EFSA dataset[b]				
Meat, excluding liver pâté	0.98	1.24	3.32	3.61
Liver pâté	0.06	0.08	0.07	0.08

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a).

12.4.4 Four-year-olds

The contribution from different food groups and marine oil supplements to the total PCDD/Fs and DL-PCBs for four-year-olds are shown in Table 12.4.4-1 to 12.4.4-3.

Table 12.4.4-1. Contribution from different food groups and marine oil supplements to mean total PCDD/F and DL-PCB exposure of 4-year-olds (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	**Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)**			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Fish	0.51	1.31	2.3	3.09
Shellfish	0.05	0.05	0.08	0.09
Meat	0.42	0.71	0.90	1.24
Milk and other dairy	0.50	1.97	1.24	2.91
Egg	0.09	0.29	0.16	0.36
Grain and grain products	0.30	0.72	0.42	0.87
Fruit and vegetables	0.33	2.87	0.70	3.32
Other food groups	0.47	1.54	0.60	2.05
Marine oils (supplement)	0.04	0.13	0.47	0.56
Intake based on EFSA dataset[b]				
Fish	1.43	1.92	5.09	5.92
Shellfish	0.02	0.02	0.04	0.05
Meat	1.08	1.38	3.41	3.75
Milk and other dairy	1.47	2.32	4.00	4.88
Egg	0.31	0.38	0.72	0.80
Grain and grain products	0.30	0.72	0.42	0.87
Fruit and vegetables	0.33	2.87	0.70	3.42
Other food groups	0.46	1.53	0.58	2.04
Marine oils (supplement)	0.21	0.24	1.14	1.17

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a).

Table 12.4.4-2. Contribution from fish and seafood to mean total PCDD/F and DL-PCB exposure of 4-year-olds (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	**Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)**			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Lean fish	0.03	0.19	0.17	0.34
Fatty fish, sum	0.44	1.07	1.93	2.57
Fatty fish: salmon/ trout	*0.22*	*0.67*	*1.08*	*1.54*
Fatty fish: mackerel	*0.22*	*0.39*	*0.84*	*1.01*
Fatty fish: other[c]	*0.00*	*0.01*	*0.01*	*0.02*
Roe and liver	0.04	0.04	0.19	0.19
Shellfish	0.05	0.05	0.08	0.09
Intake based on EFSA dataset[b]				
Lean fish	0.18	0.39	0.84	1.36
Fatty fish, sum	1.14	1.35	3.83	4.08
Fatty fish: salmon/ trout	*0.75*	*0.90*	*2.42*	*2.59*
Fatty fish: mackerel	*0.37*	*0.44*	*1.37*	*1.45*
Fatty fish: other[c]	*0.01*	*0.01*	*0.03*	*0.04*
Roe and liver	0.10	0.18	0.41	0.48
Shellfish	0.02	0.02	0.04	0.05

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a). [c] Other fatty fish species are halibut, wild and fresh-water trout, summer and winter herring, and char.

Table 12.4.4-3. Contribution from meat to mean total PCDD/F and DL-PCB exposure of 4-year-olds (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	**Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)**			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Meat, excluding liver pâté	0.42	0.71	0.90	1.24

Food groups	Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Liver pâté	0.03	0.04	0.06	0.06
Intake based on EFSA dataset[b]				
Meat, excluding liver pâté	1.08	1.38	3.41	3.75
Liver pâté	0.14	0.17	0.16	0.19

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a).

12.4.5 Two-year-olds

The contribution from different food groups and marine oil supplements to the total PCDD/Fs and DL-PCBs for two-year-olds are shown in Table 12.4.5-1 to 12.4.5-3.

Table 12.4.5-1. Contribution from different food groups and marine oil supplements to mean total PCDD/F and DL-PCB exposure of 2-year-olds (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups[c]	Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Fish	0.67	1.50	2.85	3.69
Shellfish	0.0	0.0	0.0	0.0
Meat	0.31	0.47	0.57	0.77
Milk and other dairy	0.74	2.94	1.85	4.35
Egg	0.11	0.36	0.19	0.44
Grain and grain products	0.23	0.57	0.34	0.70
Fruit and vegetables	0.71	3.15	1.61	4.31
Other food groups	0.44	1.13	0.54	1.43
Marine oils (supplement)	0.02	0.15	0.52	0.67
Intake based on EFSA dataset[b]				
Fish	1.56	2.14	5.89	6.91
Shellfish	0.0	0.0	0.0	0.0
Meat	0.84	1.06	2.17	2.42
Milk and other dairy	2.19	3.51	6.08	7.44
Egg	0.38	0.46	0.89	0.98
Grain and grain products	0.23	0.57	0.34	0.70

Food groups[c]	Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Fruit and vegetables	1.04	3.04	2.89	5.08
Other food groups	0.44	1.13	0.54	1.43
Marine oils (supplement)	0.34	0.39	1.86	1.91

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a). [c] Milk protein is not included as a source.

Table 12.4.5-2. Contribution from fish and seafood to mean total PCDD/F and DL-PCB exposure of 2-year-olds (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Lean fish	0.02	0.22	0.16	0.38
Fatty fish, sum	0.59	1.20	2.36	2.97
Fatty fish: salmon/ trout	*0.10*	*0.34*	*0.52*	*0.76*
Fatty fish: mackerel	*0.49*	*0.86*	*1.84*	*2.22*
Fatty fish: other[c]	*0.00*	*0.00*	*0.00*	*0.00*
Roe and liver	0.07	0.08	0.33	0.34
Shellfish	0.00	0.00	0.00	0.00
Intake based on EFSA dataset[b]				
Lean fish	0.21	0.48	1.01	1.67
Fatty fish, sum	1.20	1.41	4.23	4.50
Fatty fish: salmon/ trout	*0.38*	*0.46*	*1.23*	*1.32*
Fatty fish: mackerel	*0.81*	*0.96*	*3.00*	*3.18*
Fatty fish: other[c]	*0.00*	*0.00*	*0.00*	*0.00*
Roe and liver	0.16	0.25	0.65	0.74
Shellfish	0.00	0.00	0.00	0.00

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a). [c] Other fatty fish species are halibut, wild and fresh-water trout, summer and winter herring, and char.

Table 12.4.5-3. Contribution from meat to mean total PCDD/F and DL-PCB exposure of 2-year-olds (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	**Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)**			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Meat, excluding liver pâté	0.31	0.47	0.57	0.77
Liver pâté	0.07	0.07	0.11	0.12
Intake based on EFSA dataset[b]				
Meat, excluding liver pâté	0.84	1.06	2.17	2.42
Liver pâté	0,28	0,35	0,31	0,38

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a).

12.4.6 One-year-olds

The contribution from different food groups and marine oil supplements to the total PCDD/Fs and DL-PCBs for one-year-olds are shown in Table 12.4.6-1 to 12.4.6-3.

Table 12.4.6-1. Contribution from different food groups and marine oil supplements to mean total PCDD/F and DL-PCB exposure of 1-year-olds (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	**Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)**			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Fish	0.72	1.65	3.12	4.07
Shellfish	0.0	0.0	0.0	0.0
Meat	0.44	0.63	0.86	1.10
Milk and other dairy	0.49	2.38	1.39	3.54
Egg	0.10	0.34	0.18	0.42
Grain and grain products	0.44	1.06	0.59	1.28
Fruit and vegetables	1.46	6.50	3.31	8.85
Other food groups	0.31	0.70	0.38	0.86
Marine oils (supplement)	0.02	0.19	0.66	0.85
Intake based on EFSA dataset[b]				
Fish	1.72	2.29	6.38	7.38

Food groups	Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Shellfish	0.0	0.0	0.0	0.0
Meat	0.91	1.21	2.09	2.43
Milk and other dairy	1.09	2.51	3.34	4.90
Egg	0.36	0.44	0.84	0.93
Grain and grain products	0.44	1.06	0.59	1.28
Fruit and vegetables	2.62	6.11	7.85	11.60
Other food groups	0.31	0.70	0.38	0.86
Marine oils (supplement)	0.43	0.49	2.33	2.39

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a). [c] Milk protein is not included as a source.

Table 12.4.6-2. Contribution from fish and seafood to mean total PCDD/F and DL-PCB exposure of 1-year-olds (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Lean fish	0.02	0.22	0.16	0.36
Fatty fish, sum	0.64	1.35	2.60	3.32
Fatty fish: salmon/ trout	*0.15*	*0.50*	*0.78*	*1.13*
Fatty fish: mackerel	*0.48*	*0.85*	*1.82*	*2.20*
Fatty fish: other[c]	*0.00*	*0.00*	*0.00*	*0.00*
Roe and liver	0.07	0.08	0.37	0.38
Shellfish	0.00	0.00	0.00	0.00
Intake based on EFSA dataset[b]				
Lean fish	0.20	0.46	0.97	1.61
Fatty fish, sum	1.37	1.62	4.79	5.09
Fatty fish: salmon/ trout	*0.56*	*0.67*	*1.81*	*1.94*
Fatty fish: mackerel	*0.81*	*0.95*	*2.97*	*3.15*
Fatty fish: other[c]	*0.00*	*0.00*	*0.00*	*0.00*

Food groups	Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Roe and liver	0.14	0.21	0.62	0.69
Shellfish	0,00	0,00	0,00	0,00

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a). [c] Other fatty fish species are halibut, wild and fresh-water trout, summer and winter herring, and char.

Table 12.4.6-3. Contribution from meat to mean total PCDD/F and DL-PCB exposure of 1-year-olds (in pg WHO_{2005}-TEQ/kg bw per week).

Food groups	Observed individual means (pg WHO_{2005}-TEQ/kg bw per week)			
	PCDD/Fs		PCDD/Fs + DL-PCB	
	LB	UB	LB	UB
Intake based on VKM dataset[a]				
Meat, excluding liver pâté	0.44	0.63	0.86	1.10
Liver pâté	0.09	0.10	0.15	0.15
Intake based on EFSA dataset[b]				
Meat, excluding liver pâté	0.91	1.21	2.09	2.43
Liver pâté	0.37	0.46	0.41	0.51

[a] Norwegian occurrence data for fish, meat, eggs and dairy products combined with data from EFSA for other foods. [b] Concentration data in food from EFSA (2018a).

12.5 Respondents in the top 10% of exposure

VKM analysed the contribution from food groups to the toal exposure for the respondents with the top 10% of exposure for their age groups: adults, 13-, 9-, 4-, 2- and 1-year-olds. Tables 12.5-1 through 12.5-4 for respondents in the top 10% of exposure correspond to Tables 3.3-1 through 3.3-4, for an average respondent. Those with the highest 10% exposures had higher contribution from fish and shellfish than those with average intake at both LB and UB exposure.

Table 12.5-1. The contribution of food groups, in percent, to the mean total LB PCDD/F and DL-PCB exposure for respondents in the top 10% of exposure

	Adults (18-70 years)	Women (18-45 years)	13-year-olds	9-year-olds	4-year-olds	2-year-olds	1-year-olds
Fish	71	85	63	62	55	63	69
Shellfish	16	3.3	7.4	2.5	4.2	0.0	0.0
Meat	4.1	3.2	8.5	10	13	4.2	6.1
Dairy	4.1	4.0	8.8	9.6	9.6	15	9.4

	Adults (18-70 years)	Women (18-45 years)	13-year-olds	9-year-olds	4-year-olds	2-year-olds	1-year-olds
Egg	0.72	0.46	1.2	1.0	1.1	1.8	1.6
Grain	0.73	0.79	3.7	3.9	3.0	2.5	3.9
Other	1.8	2.1	6.1	8.1	4.5	3.8	2.9
Marine oils (supplement)	1.7	1.4	1.8	3.2	9.8	9.6	7.5

Table 12.5-2. The contribution of food groups, in percent, to the total UB PCDD/F and DL-PCB exposure for respondents in the top 10% of exposure

	Adults (18-70 years)	Women (18-45 years)	13-year-olds	9-year-olds	4-year-olds	2-year-olds	1-year-olds
Fish	67	78	54	53	49	52	59
Shellfish	13	2.6	4.8	1.7	3.0	0.0	0.0
Meat	4.2	3.2	7.2	8.4	11	3.8	5.5
Dairy	7.4	7.0	13	14	15	24	17
Egg	1.3	0.8	1.7	1.4	1.7	2.8	2.7
Grain	1.2	1.3	4.7	4.7	4.0	3.5	5.9
Other	4.5	5.5	14	15	9.7	6.7	4.6
Marine oils (supplement)	1.6	1.3	1.3	2.4	7.3	7.5	6.5

The higher fish exposure comes to a greater extent from fish roe and liver as well as shellfish, particularly for adults (Table 12.5-3 compared to Table 3.3-3).

Table 12.5-3. Contribution in percent to total lower bound PCDD/F and DL-PCB exposure from fish and shellfish for respondents in the top 10% of exposure

	Adults (18-70 years)	Women (18-45 years)	13-year-olds	9-year-olds	4-year-olds	2-year-olds	1-year-olds
Lean fish	11	13	1.6	5.8	4.8	2.0	2.0
Salmon	19	20	74	73	44	7.7	10
Mackerel	17	23	13	17	38	74	67
Other fatty fish	17	12	0.0	0.0	0.0	0.0	0.0
Roe and liver	18	27	0.62	1.0	5.8	16	20
Shellfish	19	3.8	11	4.0	7.2	0.0	0.0

These respondents also had higher contribution from fish liver than those with average exposure. This is the case for all age groups, except for the surveys of 9- and 13-year-olds, none of whom reported eating any fish liver or fish-liver containing products.

Table 12.5-4. Contribution in percent to total lower bound PCDD/F and DL-PCB exposure from fish offal for respondents in the top 10% of exposure.

	Adults (18-70 years)	Women (18-45 years)	13-year-olds	9-year-olds	4-year-olds	2-year-olds	1-year-olds
Fish Liver	94	99	0.0	0.0	40	73	91
Fish Roe	5.6	1.4	100	100	60	27	9.5

13 Appendix IV: Deviations from the protocol

Occurrence data

One criteria for inclusion of occurrence data in the database was that the samples were taken during year 2010 or later. For cod roe, cod roe-liver pâté, brown crab meat and liver pâté, no occurrence data for samples taken during 2010 or later were identified. To include these foods in the exposure estimation, occurrence data from samples taken before 2010 were included in the database.

VKM planned to prioritise data in the dioxin database as follows: 1) Norwegian occurrence data for foods most likely eaten in Norway, 2) Norwegian data and European data from EFSA will be compared if available Norwegian data are scarce, and 3) European data from EFSA will be used when Norwegian data are lacking. The VKM dataset was developed according to this prioritisation. However, a dataset only based on the EFSA occurrence data was also prepared and used in the exposure estimations. This way, is was possible to compare the exposure estimates obtained using the two datasets.