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Dietary mercury exposure in a population with a wide range of fish consumption — Self-capture of fish and regional differences are important determinants of mercury in blood

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HIGHLIGHTS

- Dietary exposure to total Hg was calculated among fish-consuming Norwegians.
- Total Hg in blood (BTHg) and urine was measured in the same participants.
- BTHg ranged from 0.6 to 30 μg/L (n = 184).
- Up to 65% of the variance in BTHg were explained by linear regression.
- Fish consumption, coastal residence and self-catching were significant factors.

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ABSTRACT

Human, low level, chronic exposure to mercury (Hg) from fish is of concern because of potential neurodevelopmental and cardiovascular toxicity. The purpose of the study was to 1) measure total mercury (THg) in blood and estimate dietary exposure in a population group with a wide range of seafood consumption, 2) assess the intake and blood concentration in relation to tolerable intake values, 3) characterise dietary sources, and 4) to investigate the relationship between dietary THg with THg in blood (BTHg), including factors that can explain the variance in BTHg concentrations.

The participants (n = 184) filled in an extensive food frequency questionnaire which was combined with a database on THg concentrations in Norwegian food, and donated blood and urine. Median consumption of seafood was 65 g/day (range 4 to 341 g/day). The calculated mean dietary THg exposure was 0.35 (median 0.30) μg/kg body weight/week. Seafood contributed on average 95% to the exposure. The JECFA Provisional Tolerable Weekly Intake (PTWI) of 1.6 μg MeHg/kg bw/week was not exceeded by any of the participants.

BTHg ranged from 0.6 to 30 μg/L, with a mean of 5.3 (median 4.0 μg/L). There was a strong relationship between total seafood consumption and BTHg concentrations (r = 0.58 95%CI: 0.48, 0.67) and between estimated THg dietary exposure and BTHg (r = 0.46 95%CI: 0.35, 0.57). Fish consumption, sex, catching >50% of their seafood themselves, and living in coastal municipalities were significant factors in linear regression models with lnBTHg. Including urinary Hg in the regression model increased the explained variance from 54% to 65%. In a toxicokinetic model, the calculated dietary intake appeared to moderately

Abbreviations: BTHg, Blood total Hg; BMDL, Bench mark dose, lower bound; FFQ, Food frequency questionnaire; IHg, Inorganic Hg compounds; LB, Lower Bound values<LOQ are set to 0; LOQ, Level of quantification; MeHg, Methyl Hg; NOEL, No observable effect level; NFG study, The Norwegian Fish and Game Study; PTWI, Provisional Tolerable Weekly Intake; THg, Total Hg; UB, Upper Bound values>LOQ are set to LOQ; UHg, Urinary Hg.

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underestimate the measured BTHg among the participants with the highest BTHg. Only two of the participants had BTHg slightly above a value equivalent to the JECFA PTWI, but none of them were women in fertile age.

1. Introduction

Mercury (Hg) is a naturally occurring element which may exist as elemental, inorganic or organic Hg. Human activities have increased the pool of Hg that cycles between atmosphere, land and water, and it is regarded as a global pollutant of concern even in the most remote areas (AMAP, 2005). Inorganic mercury compounds (IHg) can be bioconverted to the much more toxic and bioavailable organic form methyl mercury (MeHg), mainly by microorganisms in water, soil and sediment (WHO, 1990). Aquatic species are especially vulnerable because of MeHg's bioaccumulative properties, making top level predators, including humans, susceptible to high MeHg exposure (Mergler et al., 2007).

More than 90% of total Hg in the fish muscle is present as MeHg (Bloom, 1992), and it is well established that the majority of human MeHg exposure originates from eating fish and other types of seafood (Mahaffey et al., 2004; Mergler et al., 2007). MeHg concentrations within and amongst different edible species may vary more than 100 fold (Mergler et al., 2007), with the highest concentrations often found in large predatory fish such as swordfish and northern pike (Barregård, 2005).

The main concern regarding chronic, low level MeHg exposure is the risk of subtle negative neurodevelopmental effects in infants of mothers with high consumption of fish and other seafood with high Hg content during pregnancy (JECFA, 2003, 2006; U.S.EPA, 2001). Recently, the concern associated with chronic low level exposure to MeHg in the adult population has increased, as some studies indicate negative effects on the cardiovascular system (Choi et al., 2009; Virtanen et al., 2005). Results from different studies are, however, not consistent, as other studies show no association with cardiovascular risk (Mozaffarian et al., 2011; Wennberg et al., 2011). Many also argue that the benefits of fish consumption generally outweigh the negative effects posed by MeHg exposure, especially with a restricted consumption of top predator species with relatively high Hg concentration (Mozaffarian and Rimm, 2006; Mozaffarian et al., 2011). An updated review on possible health effects from low level MeHg exposure is found in (Karagas et al., 2012).

Tolerable intake levels of MeHg have been established to protect even the most vulnerable part of the population, i.e. the developing foetus. Exposures lower than tolerable intakes are considered to be safe for the entire population throughout a lifetime. The Joint FAO/WHO Expert Committee on Food Additives (JECFA) established a Provisional Tolerable Weekly Intake (PTWI) of 1.6 μg/kg bw/week. This PTWI is used in EU and in Norway. The US Environmental Protection Agency (US EPA) established a reference dose (RfD) of 0.1 μg/kg bw/day. The evaluations were based on the same epidemiological cohort studies describing foetal Hg exposure (JECFA, 2003, 2006; U.S.EPA, 2001).

In general, health authorities encourage fish and other seafood as a part of a balanced diet. To prevent elevated exposure to MeHg and other contaminants, avoidance or restricted consumption of certain fish species is recommended. In Norway, pregnant women are encouraged to eat 2–3 fish dinners a week, with half being from oily fish, in addition to fish spread, but to avoid certain predatory fish species (from salt water: fresh tuna, swordfish, skates and sharks, from fresh water: pike, and large species of perch, brown trout and arctic char). The general population should restrict their consumption of these special species to not more than an average of once a month (Norwegian Food Safety Authority, 2009).

Little data are available on dietary Hg exposure in the general population of Norway, even though Norwegians have one of Europe's highest average consumption rates of fish and other seafood (VKM, 2007). Consumption was estimated to be 65–70 g/person/day in independent dietary surveys of the general adult population (Bergsten, 2004; Johansson and Solvoll, 1999; Meltzer et al., 2002). Norway is known for its ample availability of fish and other seafood, due to a long coastline, many lakes and long traditions in using what nature has to offer. Fishing licences for freshwater lakes and rivers are usually reasonably priced and often for free for younger and older age groups. A fishing licence is not necessary for recreational fishing in marine waters, making it an available recreational activity for large parts of the population. Norway has one of the World's most productive marine fisheries, and export of wild and farmed fish is an important part of Norwegian economy. Thus, better knowledge about the range of exposure to Hg in Norway, based on data both providing dietary intakes and biomarkers of exposure, is of interest also beyond the national border.

The Norwegian Fish and Game (NFG) Study was initiated to obtain detailed information on consumption of fish and game, which may contain elevated concentrations of environmental contaminants and to characterise exposure to these in the Norwegian population. The sub-study presented here includes a detailed assessment of the total habitual diet the previous year, demographic data and relevant lifestyle factors as well as analyses of total Hg in blood (BTHg), and urine (UHg). BTHg is predominantly MeHg in non-occupationally exposed population groups whereas UHg is a measure of IHg exposure.

The objective of this study was to1) measure total mercury (THg) in blood and estimate dietary exposure in a group of Norwegians with a wide range of seafood consumption, 2) assess the intake and blood concentration in relation to tolerable intake values, 3) characterise dietary sources, and 4) compare estimated dietary exposure of THg with BTHg and characterise demographic variables and other factors which might explain the variance in BTHg concentrations.

This study is the first to investigate both estimated dietary THg exposure and measured BTHg and UHg concentrations in an adult Norwegian population. The inclusion criteria of the study assured that the participants had a wide range of fish and other seafood consumption.

2. Materials and methods

2.1. Study subjects and data collection

The study subjects were participants in the NFG Study Part C, a cross sectional study administered by the Norwegian Institute of Public Health (NIPH) and initiated to estimate contaminant exposure from fish and game consumption in Norway. The participants were recruited from the NFG Study Part B as described elsewhere (Knutsen et al., 2008; Kvalem et al., 2009). Briefly, in the NFG Study part B, 5502 individuals living in 27 coastal and inland municipalities across Norway without known point source of persistent organic pollutants and toxic elements, answered a semi-quantitative food frequency questionnaire (FFQ) focusing especially on the consumption of fish, shellfish and game (Bergsten, 2004). Based on answers in Part B, and rough estimates of concentrations of selected environmental contaminants or toxic elements (Hg, cadmium, dioxins and polychlorinated biphenyls) in different food items, 420 participants assumed of high exposure were invited to participate in Part C of the study. Additionally, 282 individuals were drawn randomly from the remainder of the Part B participants in order to constitute a reference group, portraying general population exposure. Data collection for Part C was conducted in 2003. Participants

delivered blood and urine samples and answered an extensive FFQ and a one page query regarding height, weight and demographic information, as described previously (Kvalem et al., 2009).

Of the 702 invited subjects, 193 (28%) gave informed consent and completed data collection. Four participants with unlikely energy intakes (less than 1077 kcal (4.5 MJ) or more than 4784 kcal (20 MJ)) were excluded from the analyses (Meltzer et al., 2008) and another 5 participants were not included because of lacking BTHg measurements. This resulted in 184 participants, of which 109 were from the high consumer group and 75 were from the reference group. They were from inland (n = 98) and coastal (n = 86) municipalities. The sample comprised 84 men and 100 women, with a mean age of 54 years (range 21–80 years) and a mean body mass index (BMI) of 25.2. The mean body weight was 74.4 kg (sd 13.6). Information regarding the share of fish consumption reported to be self-captured (both recreational angling and subsistence fishing) was imported from part B of the study. The reference group did not have significantly different means from all participants in the NFG part B with respect to place of living (coast/inland), age, sex, body weight, body mass index, smoking, and total fish consumption. The reference group can be considered representative for the NFG part B, which again was country representative (Knutsen et al., 2008; Kvalem et al., 2009).

The project protocol was approved by the Regional Committee for Medical Research Ethics and the Norwegian Data Protection Agency (Id: 02138).

2.2. Dietary information

Dietary information for the preceding 12 months was obtained using a detailed semi-quantitative FFQ designed and validated for the Norwegian Mother and Child Cohort study (Brantsæter et al., 2008; Meltzer et al., 2008). The FFQ was adapted to Norwegian food traditions and contained 340 questions, covering 255 different food items, of which 26 questions specifically regarded fish and shellfish consumption. For each food item, the participants were asked to report consumption frequency by selecting one out of 8 to 10 alternatives on a scale ranging from never eaten to several times a month, week or day. Seafood eaten as bread spread, dinner fillets or in a mixed dish was grouped into four categories. The first category comprised oily and semi-oily fish (denoted oily fish) and included e.g. salmon, trout, mackerel, halibut, herring, monkfish and catfish (>2% lipid). The second category denoted lean fish included e.g. cod, saithe, haddock, pollock, plaice, sole, tuna, perch and pike (<2% lipid). The third category comprised shellfish and included shrimp, crab and mussels, and the fourth category comprised fish liver and fish roe. The same FFQ was used by the Norwegian Birth Cohort (MoBa), except that questions 31–38 in the MoBa FFQ addressing pregnancy-related changes in the diet were replaced by questions about major changes in diet during life, and that the recall period covered the preceding 12 months. An English translation of the MoBa FFQ is available at http://www.fhi.no/dokumenter/253304bd64.pdf.

The questionnaires were read optically and food frequencies were converted to consumption (food eaten in grams per day) by multiplying with standard gender specific portion sizes (Kvalem et al., 2009). Total energy intake was calculated using FoodCalc Software (Lauritsen, 2010) and the Norwegian Food Composition Table (Rimestad et al., 2006).

2.3. Dietary exposure calculations

A database on THg concentrations in Norwegian fish, seafood and other food items was established. THg concentrations in marine and freshwater fish and shellfish were compiled mainly from Norwegian samples. Samples expected to be impacted by local pollution sources in addition to the general background (i.e. from areas with known point sources) were excluded. For each species of fish or other seafood, the mean THg concentration was estimated by averaging the means. When the mean value was not available, the reported median concentrations were used (Table 1).

The dietary exposure to THg was estimated by combining data on reported consumption (grams per day) and THg concentration in specific food items. No monitoring of Hg has been conducted in other foods than seafood in Norway, thus literature from other countries, mostly Nordic were used (Supplementary Table 1). For food items with no available data, comparable food items were used to estimate the concentration if applicable (example: THg concentration in poultry used as the THg concentration in goose). For a number of general food items, the majority of available data where below the respective levels of quantification (LOQ). To account for the uncertainty associated with values below LOQ, two different exposure estimates were conducted; the lower bound (LB) approach (values<LOQ set to zero), and the upper bound (UB) approach (values<LOQ set to LOQ). For non-seafood items (with most of the concentrations<LOQ) the LB approach will underestimate exposure while the UB approach will overestimate this part of the exposure, especially if the LOQ is high. In addition, two different values for THg in cod were used to account for potential difference between commercial Atlantic cod and coastal cod (Table 1). The 'commercial cod' THg concentration is assumed to reflect cod sold in most grocery stores, while the 'coastal cod' THg concentration is assumed to better reflect the THg concentrations of locally captured cod (Table 1). The mean THg concentration in the 'coastal cod' is approximately two times higher than in commercial cod. Commercial cod values are used in the exposure estimates if not otherwise stated.

2.4. Analytical methods

Blood and urine concentrations of Hg were determined by inductively coupled plasma-sector field mass spectrometry using an Element 2 mass spectrometer (Thermo Electron, Bremen, Germany) following a previously described method (Ellingsen et al., 2006). Shortly, 1.5 mL of 65% ultrapure nitric acid was added to 1 mL of whole blood in a polypropylene digestion tube and the tube was heated to 95 °C for 1 h. After cooling, the digested blood sample was added an internal standard solution containing ^{205}Tl for ^{200,201,202}Hg before dilution to volume (10 mL). To prevent any risk of laboratory acquired infections and to dissolve urine precipitates, all urine samples were, after thawing heated for one hour at 95 °C prior to analysis. To 1 mL of urine was added an internal standard solution containing ^{205}Tl for ^{200,201,202}Hg and diluted to volume (5 mL). The instrument was calibrated with whole blood and urine matrix matched standard solutions and the accuracy was determined by use of Seronorm Trace Elements human whole blood and urine quality control samples (Sero Ltd., Asker, Norway). The measured values were on average within ±5% of the recommended values supported by the producer. The within assay precision was typically 3–10%. The detection limits (three times standard deviations of all blank samples) in whole blood and urine was 2 and 0.35 nmol/L of Hg, respectively. None of the samples had values below the detection limits. Creatinine in urine spot samples was measured by Fürst Medical Laboratory (Oslo) and Hg in urine was normalized for creatinine concentration.

2.5. Toxicokinetic model

We used a one-compartment toxicokinetic model (U.S.EPA, 2001) in order to calculate expected Hg concentrations blood based on the estimated dietary intake of THg, using the following formula:

$$c = (d * A * f * bw)/(b * V)\mu g/L$$

where

c = concentration of MeHg in blood (μg/L)
d = daily dietary MeHg intake (μg/kg/day)

Table 1
Concentration of total Hg (μg/g) in different species of fish, fish products and shellfish used in the exposure calculations.

Fish, fish products and shellfish	Latin name	Samples	Year	Mean	Min	Max	References
Arctic char	*Salvelinus alpinus*	>900	1995–2008	0.077	0.01	1.04	1,2,3,4[a,c]
Atlantic cod (including unpolluted coastal cod)	*Gadus morhua*	>3200	1972–2007	0.063	0.01	0.45	5,6,7,8[b,c]
Atlantic cod 'commercial'(North-East Arctic cod)	*G. morhua*	494	1995–2007	0.034	0.01	0.45	5
Atlantic cod liver	*G. morhua*	>159	1994–2007	0.017	0.01	0.09	5,6,8[b]
Atlantic cod roe	*G. morhua*	85	1972–2006	0.013	<0.01	0.12	8,9,10[b]
Atlantic halibut	*Hippoglossus hippoglossus*	>60	1975–2007	0.350	0.07	1.2	5,8,10,11
Atlantic salmon, farmed	*Salmo Salar*	479	1994–2008	0.029	0.01	0.06	5,8,12
Blue mussel	*Mytilus edulis*	>600	1983–2007	0.014	0.004	0.068	6,7,13[b]
Brown trout	*Salmo trutta*	>2500	1965–2008	0.120	0.01	3.14	1,3,4,14–18[d]
Canned tuna	*Thunnus thynnus*	48	2001–2006	0.091	0.03	0.16	10,12,19
Crab brown meat	Brachyura	12	2002–2007	0.065	0.033	0.13	5,12
Crab claws	Brachyura	>14	1999–2007	0.127	0.08	0.17	5,12,13,
Haddock	*Melanogrammus aeglefinus*	>160	1975–2003	0.023	0.01	0.08	5,8[b]
Herring	*Clupea harengus*	>1250	1975–2007	0.027	0.006	0.4	5,8,12[b]
Mackerel	*Scomber scombrus*	>275	1994–2006	0.030	0.01	0.1	5,8,20[b]
Mackerel (cold smoked)	*S. scombrus*	>7	2007	0.040	0.03	0.08	21[b]
Mackerel (warm smoked)	*S. scombrus*	>10	2007	0.043	0.03	0.05	21[b]
Mackerel in tomato sause	*S. scombrus*	>12	2007	0.021	0.01	0.03	21[b]
Perch	*Perca fluviatilis*	>5000	1965–2008	0.328	0.01	4.16	1,4,14,15,22[d,a]
Northern pike	*Esox lucius*	24520	1965–2008	0.570	0.01	6.02	1,4,15[d,a]
Plaice	*Pleuronectes platessa*	>130	1990–2007	0.040	0.0155	0.115	6,7,8
Pollock/Coalfish/Saithe	*Pollachius virans*	225	1994–2004	0.033	0.01	0.19	5, 7[b]
Sardines in oil	*Sardina pilchardus*		1980	0.019	0.004	0.035	23
Shrimp	Caridea		1984–2007	0.024	0.01	0.21	5,8,10[b]
Wolffish/Catfish/rock turbot/Seawolf	Anarhichadidae	>50	1975–2005	0.067	0.02	0.11	5,8

[a] Mean Hg is missing from the studies, median values are used.
[b] Some data are from composite samples.
[c] Arlantic cod (including coastal) are Hg concentrations used for the 'coastal' calculations.
[d] Majority of data are from the Nordic studies. References: [1]Munthe et al., 2007, [2]AMAP, 2005, [3]Skjelkvåle et al., 2009, [4]Rognerud et al., 1996, [5]NIFES, 2009, [6]Fromberg et al., 2005, [7]Green et al., 2008, [8]SNT, 1997, [9]SNT, 1999, [10]Julshamn and Måge, 2007, [11]Julshamn et al., 2008, [12]Peterson-Grawè et al., 2007, [13]NIFES, 2007, [14]Christensen et al., 2008, [15]Fjeld and Rognerud, 2004, [16]Løvik et al., 2009, [17]Rognerud et al., 2003, [18]Jenssen et al., 2010, [19]SCOOP, 2004, [20]Julshamn and Frantzen, 2009, [21]Julshamn and Måge, 2007, [22]Fjeld and Rognerud, 2009, [23]Koivistoinen, 1980.

A = absorption factor (0.95)
f = the absorbed fraction distributed to the blood (0.059)
bw = body weight (kg)
A = elimination constant (0.014 day^{-1})
V = volume of blood in the body (5 L).

By use of this formula it is suggested that estimated dietary THg exposure is equal to MeHg exposure.

2.6. Statistical methods

We first explored the data using scatter plots and descriptive statistics to investigate departures from normal distribution. The BTHg concentrations as well as variables describing fish consumption were not normally distributed and nonparametric tests were used for unadjusted analyses. The BTHg concentration distribution was skewed with a long tail to the right and hence transformed to the natural log for use in the adjusted analyses.

To test for differences in blood and urine concentrations as well as differences in estimated intakes between independent groups we used Mann–Whitney *U* test (dichotomous groups) or Kruskall–Wallis test. We used Spearman correlation coefficients to calculate the association between BTHg, seafood consumption and estimated THg exposure.

The relationships between fish consumption, estimated THg exposure and BTHg concentrations were explored using multivariable linear regression. In all models, the natural log of BTHg was used as the dependent variable. The study population comprised two groups (one group with high fish and game consumption and one randomly selected group), all participants were combined in the regression analysis as recruitment group did not influence the association between exposure and BTHg as outcome. Three different models were constructed. In the first model we included estimated dietary THg as the main exposure variable, while in the second and third models, total seafood consumption was included as the main exposure variable. The dietary Hg intake (model 1), or consumption of food from different food groups (models 2 and 3), living in coastal or inland municipalities, amount of self-capture, total energy intake (kcal/day), BMI, age, age squared, parity, breastfeeding, education level, sex and smoking status were included as covariates. The third model differed from the second by including UHg to control for the fraction of IHg in blood. The models were built individually by stepwise exclusion of non-statistically significant covariates ($p > 0.05$). Further, the models were checked for interactions, and interaction terms were included if statistically significant ($p \leq 0.05$).

Residual plots for each model were examined to ensure that standard assumptions of linearity, normality and homogeneity of variance were met. Several influential points were identified, examined and found to be error free. Also, we compared results from original regression with robust regression, which reduces the effect of extreme values by weighting points in proportion to their leverage. Our results did not change from the original regressions, thus the results presented come from the original regressions.

All statistical analyses were performed using Stata version 11 (Stata Corporation, College Station, Texas). The level of statistical significance was set to 0.05.

3. Results

3.1. Participant characteristics and Hg concentrations in blood and urine

BTHg ranged from 0.6 to 30 μg/L for all the included participants (n = 184), with a mean and median of 5.3 and 4.0 μg/L respectively. For the reference group (n = 75) the average was 4.4 μg/L, the median 3.8 μg/L and the concentrations ranged from 0.6 to 30 μg/L (Table 2). This was significantly lower than in the high consumer group (mean 6.0, median 4.7, range 1.2 to 28 μg/L). The BTHg was significantly higher in men (n = 84) than in women (n = 100) (median 5.1 vs. 3.4 μg/L, p<0,001), and positively associated with total fish and other

seafood consumption (total seafood consumption, $p<0.001$). BTHg also increased with increasing age group, decreasing education level, living in a coastal municipality and with being more than 50% self-sufficient with fish and other seafood. The BTHg concentrations were not associated with BMI and energy intake. More details on participant characteristics and the BTHg distribution in different categories can be found in Table 2, together with the UHg concentrations, which ranged from 0.1 to 5.1 μg/g creatinine.

3.2. Estimated Hg exposure, fish consumption and sources of Hg exposure

The calculated dietary exposure to Hg (LB) among all participants was 0.35 (mean) and 0.30 (median) μg/kg body weight/week (Table 2). Differences between groups were similar to the BTHg results, with the exposure estimates being positively associated with e.g. increasing total seafood consumption, sex and age group.

The LB estimated THg exposures did not exceed the JECFA's PTWI of 1.6 μg MeHg/kg bw/week, whereas for the UB calculation one participant slightly exceeded. When deriving the PTWI, JECFA used the maternal THg in hair, representing the average between the $BMDL_{10}$ for neurodevelopmental effects in children from the Faroe Island cohort and the no observable effect level (NOEL) from children in the Seychelles cohort as a starting point (JECFA, 2003). The hair value was converted into a corresponding BTHg using a conversion factor of 250 and an uncertainty factor of 2 was included. The BTHg obtained was 28 μg/L. Two of the participants in the present study exceeded this value, but none of them were women in fertile age. JECFA later pointed out that a value twice their PTWI (equivalent to a BTHg of 56 μg/L) would not pose any risk of neurotoxicity in adults (JECFA, 2006). None of our participants exceeded this value.

In total 7.1% of the participants (1.3% in the reference group and 11% in the high consumers group) had estimated exposure above the US EPA RfD of 0.1 μg/kg bw/day. For the UB estimates 21.2% of the participants exceeded the US EPA RfD. The blood concentration of 5.8 μg/L, which corresponds to the US EPA RfD of 0.1 μg/kg bw/day (Mahaffey et al., 2009; National Research Council, 2000) was exceeded by 23% from the reference group and 42% from the high consumer group.

Estimated total seafood consumption ranged from 4 to 341 g/day for all participants, with a mean and median consumption of 74 and 65 g/day respectively (Table 3). Even though seafood on average only comprised 2.1% of the total grams consumed per day of food and beverages, seafood contributed to an average 95% (3.5 of 3.7 μg Hg /day) of calculated dietary THg exposure using LB THg values (Table 3). When using the higher concentration in cod ('coastal cod', see 2.3), seafood contributed with approximately the same (95%) proportion (data not shown). Lean (35%) and oily fish (49%) were the dominant dietary sources of LB THg for both the reference and the high consumer group (Fig. 1, Table 3). For the UB THg estimates, fish and other seafood consumption only made up for 60% of dietary THg exposure (Table 3), reflecting the high uncertainty of the available THg concentration in non-fish food items.

The highest concentrations of THg in Norwegian seafood were found in pike, perch and Atlantic halibut, with mean concentrations of 0.57, 0.33 and 0.35 mg/kg, respectively (Table 1). High maximum concentrations were also observed in e.g. brown trout, which reflect large variation in Hg concentration, depending on location, age and size. The minimum and maximum THg concentrations in seafood

Table 2
Blood total Hg concentrations (BTTHg) (μg/L), dietary exposure estimates (μg Hg/kg bw/week) and μg urine Hg concentration (UHg) stratified by demographic variables and tertiles of fish consumption for the participants in the NFG Study Part C.

				BTHg							Dietary exposure estimates (LB)						UHg			
				(μg/L)							(μg Hg/kg bw/week)						(μg/g creatinine, N = 178)			
	Group	N	%	Mean	Min	Max	Median	p90	p95	P-value	Mean	Median	p75	p90	p95	P-value	Mean	Min	Max	P-value
All participants		184	100	5.3	0.6	30	4.0	9.7	12		0.3	0.3	0.5	0.6	0.8		1.3	0.1	5.1	
Selection group[a]	Randomly selected	75	41	4.4	0.6	30	3.8	8.0	10		0.3	0.3	0.3	0.5	0.5		1.3	0.1	4.2	
	High consumers	109	59	6.0	1.2	28	4.7	12	14	**0.005**	0.4	0.4	0.5	0.7	0.8	**<0.001**	1.3	0.1	5.1	0.471
Sex[a]	Male	84	46	6.6	0.7	30	5.1	11	16		0.4	0.3	0.5	0.8	0.8		1.4	0.1	5.1	
	Female	100	54	4.3	0.6	16	3.4	8.2	11	**<0.001**	0.3	0.3	0.4	0.5	0.6	**0.036**	1.1	0.1	3.9	0.172
Age group[b]	<40	34	18	2.3	0.6	6.5	1.6	4.3	5.4		0.2	0.2	0.3	0.4	0.4		0.9	0.1	4.0	
	40-60	75	41	5.7	1.3	30	4.2	11	16		0.3	0.3	0.5	0.6	0.7		1.7	0.3	5.1	
	>60	75	41	6.3	1.0	28	5.9	10	13	**<0.001**	0.4	0.4	0.5	0.8	0.8	**<0.001**	1.1	0.1	2.8	**<0.001**
Municipality[a]	Coastal	86	47	7.2	0.8	30	6.3	12	16		0.4	0.4	0.5	0.6	0.7		1.4	0.1	5.1	
	Inland	98	53	3.7	0.6	25	3.1	6.8	8.0	**<0.001**	0.3	0.3	0.4	0.6	0.8	**0.001**	1.1	0.1	4.2	**<0.001**
Self capture[a]	<50%	69	38	3.5	0.6	9.6	3.2	6.6	7.1		0.3	0.3	0.4	0.5	0.6		1.1	0.1	4.2	
	>50%	115	63	6.4	0.8	30	5.4	12	16	**<0.001**	0.4	0.3	0.5	0.7	0.8	**0.004**	1.4	0.1	5.1	**0.036**
Education[b]	Basic	52	28	6.8	1.2	28	5.3	12	14		0.4	0.4	0.6	0.8	0.8		1.3	0.1	5.1	
	High school	62	34	5.7	0.6	30	3.9	11	16		0.4	0.3	0.5	0.6	0.7		1.3	0.1	4.2	
	University, college	66	36	3.8	1.0	10	3.4	7.0	7.7	**0.002**	0.3	0.3	0.3	0.4	0.5	**<0.001**	1.2	0.2	4.1	0.274
	Missing	4	2	5.3	2.7	8.9	4.9	8.9	8.9		0.3	0.3	0.5	0.6	0.6		1.0	0.6	1.5	
BMI[b]	<25	103	56	5.0	0.6	30	3.6	9.7	12		0.4	0.3	0.5	0.6	0.8		1.3	0.1	5.1	
	25-30	62	34	5.2	0.7	13	4.8	9.5	12		0.4	0.3	0.5	0.6	0.7		1.2	0.2	2.8	
	>30	19	10	7.6	1.0	28	6.3	20	28	0.079	0.3	0.2	0.4	0.5	0.6	0.266	1.2	0.2	2.8	0.956
Seafood[b]	T1(4–50 g/day)	61	33	2.9	0.6	7.7	2.4	5.4	6.8		0.2	0.2	0.2	0.3	0.3		1.1	0.1	4.2	
	T2 (51–81 g/day)	61	33	5.7	1.2	30	4.4	9.5	13		0.3	0.3	0.4	0.5	0.5		1.3	0.2	4.1	
	T3(82–341 g/day)	62	34	7.4	1.0	28	6.5	12	14	**<0.001**	0.5	0.5	0.6	0.8	0.8	**<0.001**	1.4	0.2	5.1	0.054
Kcal[b]	T1(1094–1949 Kcal/day)	61	33	5.3	0.8	28	4.2	9.7	13		0.3	0.3	0.4	0.6	0.6		1.3	0.1	5.1	
	T2(1950–2527 Kcal/day)	61	33	4.9	0.6	14	3.7	9.0	10		0.3	0.3	0.4	0.5	0.7		1.2	0.1	3.5	
	T3(2528–4761 Kcal/day)	62	34	5.8	0.8	30	4.4	12	16	0.909	0.4	0.3	0.5	0.7	0.8	0.083	1.3	0.1	4.2	0.895

Significant P values in bold, significant if $P<0.05$.
T1–T3 indicates 1st to 3rd tertile. One extreme UHg outlier was set to missing. Missing Urine Creatinine values interpolated as median creatinine value.
[a] Mann Whitney *U* Test/Wilcoxon rank-sum tests for differences between groups,
[b] Kruskal Wallis Test.

Table 3
Estimated food consumption and estimated THg exposure using lower bound (LB) and upper bound (UB) approaches.

	Food consumption (grams/day)						LB THg (μg/day)						UB THg (μg/day)					
Food group	Mean	Median	25 P	75P	95 P	%	Mean	Median	25 P	75 P	95 P	%	Mean	Median	25 P	75 P	95 P	%
Total seafood consumption[a]	74	65	42	98	160	2.1	3.5	3.0	2.0	4.6	7.6	95	3.5	3.0	2.0	4.6	7.6	60
Oily fish	33	24	15	41	93	0.9	1.8	1.5	0.9	2.4	3.9	49	1.8	1.5	0.9	2.4	3.9	31
Lean fish	29	22	12	40	69	0.8	1.3	0.9	0.5	1.7	3.2	35	1.3	0.9	0.5	1.7	3.2	22
Shellfish	7.8	5.7	2.2	9.9	20	0.2	0.4	0.2	0.1	0.4	1.1	9.7	0.4	0.2	0.1	0.4	1.1	6.2
Fish liver and roe	4.9	2.8	0.5	6.6	20	0.1	0.1	0.0	0.0	0.1	0.3	1.9	0.1	0.0	0.0	0.1	0.3	1.2
Eggs(including gull eggs[b])	23	16	12	29	58	0.6	0.1	0.0	0.0	0.1	0.3	2.2	0.1	0.0	0.0	0.1	0.3	1.4
Vegetables and cereals	950	910	720	1120	1550	27	0.1	0.1	0.0	0.1	0.2	2.0	1.5	1.4	1.1	1.8	2.4	26
Meat products	79	74	56	100	130	2.2	0.0	0.0	0.0	0.0	0.1	0.6	0.1	0.1	0.1	0.2	0.3	2.6
Other[c]	1900	1800	1410	2310	3380	53	0.0	0.0	0.0	0.0	0.0	0.3	0.5	0.5	0.3	0.6	0.9	8.2
Dairy products	530	430	264	645	1300	15	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.0	0.1	0.2	1.3
Total	3600	3400	2900	4000	5600	100	3.7	3.2	2.2	4.8	7.8	100	5.7	5.3	4.0	6.8	10	100

[a] Total seafood consumption is the sum of oily fish, lean fish, shellfish and fish liver and roe.
[c] The "Other" group; includes beverages, sweets and different dry goods foods.
[b] On average, gull eggs compromise 0.006% of the total food consumption and 0.53% of estimated LB THg exposure.

illustrate the relatively large variation possible both between and within species (Table 1). The intra-species variation is not reflected in the exposure calculations, as only an estimated average concentration was used for the intake estimates.

The most important contributors to the LB estimated dietary exposure other than seafood were eggs and the combined group 'vegetables, cereals, fruits and oils', contributing with an average of 2.2 and 2.0% of the LB estimated THg exposure (μg/day), respectively (Fig. 1, Table 3). For the UB estimates, the most noteworthy factors (other than seafood) were the group 'vegetables, cereals, fruits and oils' with 26% contribution and the group 'other' which includes 'beverages, sweets and different dry goods' with 8.2% contribution.

3.3. Association between BTHg, estimated dietary Hg exposure and seafood consumption

There was a strong relationship between total seafood consumption (g/day) and BTHg concentrations, the crude correlation was 0.58 (CI: 0.48, 0.67). The same applied for estimated LB THg exposure and BTHg, with a crude correlation of 0.46 (CI: 0.35, 0.57). Fig. 2 illustrates that BTHg on average was lower for participants living in inland municipalities compared to participants residing in coastal municipalities.

We examined BTHg in three separate multivarible linear regression models (Table 4). First Ln BTHg was modelled with estimated dietary Hg exposure (Model 1), then with total seafood consumption (Model 2) and lastly, with UHg included in addition to total seafood consumption to control for the IHg in whole blood (Model 3).

There was a strong association between measured BTHg concentrations and estimated dietary LB THg exposure (Table 4, Model 1), with an adjusted explained variance (Adj.R^2) of 50.2%.

For a 0.1 μg/kg bw/week increase in estimated dietary THg exposure, an increase in BTHg of about 5.9% is thus expected ($\exp(0.57*0.1) = 1.059$). Similar models using UB exposure (Adj.R^2 49.6%) and LB 'coastal cod'(Adj.R^2 51.01%) gave similar explained variance (data not shown).

The explained variance was 54.4% in Model 2 using total seafood consumption, hence a 4.2% point better fit than for the dietary Hg exposure estimates. Increasing fish and other seafood consumption with 25 g/day (150 g/week), all else equal, will increase the BTHg concentration by 7.8%, whereas high meat consumption will lead to reduced BTHg concentrations, since concomitant fish consumption is low. Inclusion of all food groups in Table 3, thus differentiating between lean and oily fish as well as shellfish, gave a similar R^2. Lean fish consumption appeared to have the highest influence on BTHg in this study group (data not shown).

In general, the same demographic characteristics were significant in all three models. In Model 2, male BTHg concentrations were on average 37% higher than female BTHg concentrations. In addition, participants who attained more than 50% of their seafood by self-capture had on average 26% higher BTHg concentrations than their counterparts. Among participants with the highest level of BTHg, all reported a high percentage of recreationally obtained seafood (Table 2).

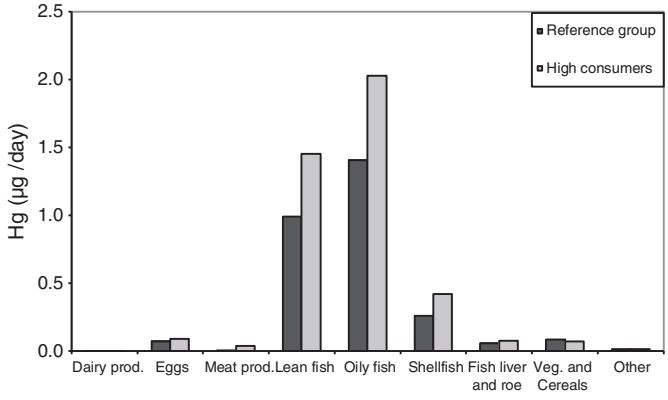

Fig. 1. Contribution from different food groups to mean lower bound THg exposure (μg/day). Data are mean contribution from all participants in the NFG Study Part C (n = 184). Dairy products: milk, cheese, yoghurts and butter. Eggs: Hen and gull eggs, Meat: all meat products, including game and offal. Lean fish: lipid content <2%. Oily fish (includes semi oily fish including salmon and trout): lipid content >2%. Shellfish: crab, shrimp and mussel. Vegetable and cereals: vegetables, cereals and fruits. Other: beverages, sweets and different dry goods.

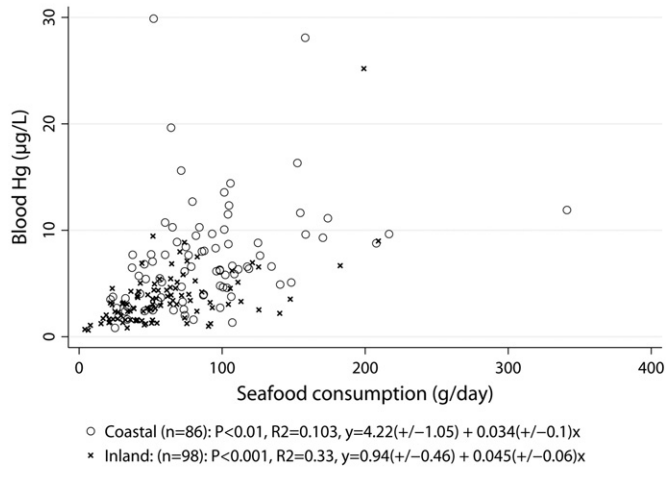

Fig. 2. Correlation between blood total Hg concentration (Blood Hg) and seafood consumption, showing the differences in BTHg between people that live in coastal and inland municipalities.

Table 4
Linear regressions for associations between the natural log of blood total mercury concentrations (BTHg) and estimated dietary total Hg exposure (Model 1), total seafood consumption (Model 2) and total seafood consumption adjusted for UHg (Model 3) (only significant variables included in the models).

Log transformed BTHg		Coefficient	SE	P		95% CI	
Model 1	Dietary THg (μg/kg bw/week)	0.57	0.22	0.009	0.14	–	0.99
(N = 184)	Age, years	0.09	0.018	0.000	0.054	–	0.13
	Age squared	−0.001	0.000	0.000	−0.001	–	0.000
	Sex (1 male, 0 female)	0.25	0.079	0.002	0.096	–	0.41
	Municipality (1 inland, 0 coastal)	−0.470	0.082	0.000	−0.63	–	−0.31
	Self capture (1 > 50%, 0 < 50%)	0.28	0.084	0.001	0.11	–	0.44
	Constant	−1.51	0.47	0.002	−2.44	–	−0.58
	R sqared					**0.52**	
	Adjusted R squared					**0.50**	
Model 2	Total seafood consumption (g/day)	0.003	0.001	0.004	0.001	–	0.005
(N = 184)	Meat consumption (g/day)	−0.004	0.002	0.005	−0.007	–	−0.001
	Age (years)	0.08	0.018	0.000	0.049	–	0.12
	Age squared	−0.001	0.000	0.000	−0.001	–	0.000
	Sex (1 male, 0 female)	0.32	0.094	0.001	0.13	–	0.50
	Municipality (1 inland, 0 coastal)	−0.38	0.081	0.000	−0.54	–	−0.22
	Self capture (1 > 50%, 0 < 50%)	0.23	0.081	0.005	0.071	–	0.39
	Constant	−0.97	0.47	0.040	−1.90	–	−0.047
	R sqared					**0.56**	
	Adj. R squared					**0.54**	
Model 3	Total seafood consumption (g/day)	0.004	0.001	0.000	0.002	–	0.005
(N = 178)	Eggs[a] (g/day)	0.007	0.002	0.002	0.003	–	0.012
	Age (years)	0.015	0.003	0.000	0.010	–	0.021
	Sex (1 male, 0 female)	0.35	0.082	0.000	0.19	–	0.51
	Energy intake (Kcal)	0.000	0.000	0.002	0.000	–	0.000
	Municipality (1 inland, 0 coastal)	−0.19	0.109	0.084	−0.40	–	0.026
	Self capture (1 > 50%, 0 < 50%)	0.24	0.074	0.001	0.093	–	0.38
	UHg (μg/g creatinine)	0.31	0.038	0.000	0.23	–	0.38
	Eggs * municipality	−0.007	0.004	0.043	−0.014	–	0.000
	Constant	0.03	0.221	0.890	−0.41	–	0.47
	R sqared					**0.67**	
	Adjusted R squared					**0.65**	

[a] Inclluded gull eggs.

Participants living in inland municipalities had on average 31% lower BTHg than coastal participants (Fig. 2). Participants from coastal and inland municipalities had a mean fish consumption of 88 and 61 g/day respectively. When controlling for UHg, the explained variance increased from 54% (Model 2, Table 4) to 65% (Model 3, Table 4), an increase in fit of 11% points. There was no correlation between total fish consumption and UHg. Adjusting for UHg in Model 1 resulted in a comparable increase (from 50% to 58%, 8% points, data not shown).

We applied a one-compartment toxicokinetic model (U.S.EPA, 2001) to predict the blood concentrations based on estimated dietary exposure, and plotted the predicted values against the measured BTHg (Fig. 3). The results indicated that our dietary exposure estimates fitted relatively well for participants who mainly eat commercial available fish and other seafood. However, for the participants who report to have 50% or more self-captured seafood in their diet, a large proportion departs from the 1:1 line, and thus had an underestimated dietary THg exposure.

4. Discussion

As anticipated, participants in the group selected according to expected high dietary exposure to different contaminants had higher measured BTHg concentrations and estimated dietary THg exposures than those in the reference group. The coastal participants had significantly higher average BTHg concentrations than participants from inland municipalities. This independent effect of place of residence comes in addition to the higher fish consumption among the coastal participants (88 g/day vs. 61 g/day), and might be related to e.g. where the fish has been caught, type of species consumed and fish size. Some freshwater species such as pike, perch and large brown trout are of special concern by the Norwegian Food Safety Authority because they can contain more Hg than commonly consumed marine species. It could perhaps have been expected that people in inland municipalities would have higher BTHg concentrations than the coastal population because of easier access to these freshwater species. However, since only six participants consumed pike or perch once a month or more often, and because other freshwater species were covered in aggregated questions, the impact of freshwater fish consumption could not be addressed in further detail.

Proximity to coastal areas, as well as more prominent seafood consumption traditions, fits with the finding that the amount of self-capture of fish was also an important determinant of THg exposure in the present study. THg concentrations in species caught by local fishermen or recreational anglers may differ substantially from those found in the same species sold commercially, which most likely

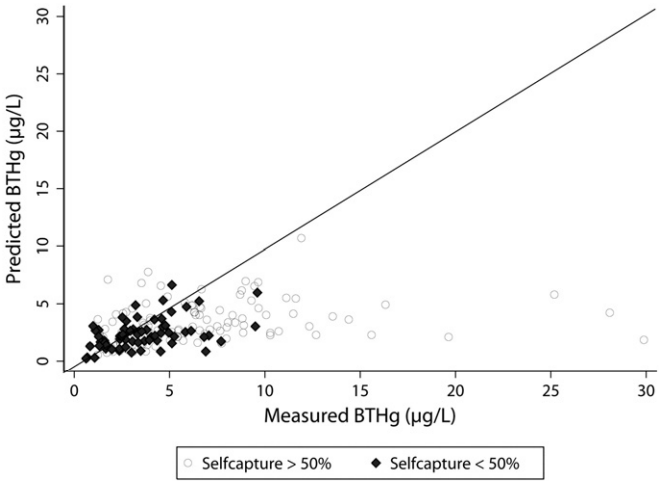

Fig. 3. Predicted BTHg (based on estimated THg exposure) vs. measured BTHg (μg/L) in relation to the 1:1 line and differentiated by participants with more and less than 50% self-captured fish in their diet.

is captured in open water. THg concentrations in marine fish are often higher near harbour areas which are also more easily accessible recreationally (Måge and Frantzen, 2009). In contrast to (e.g. Lincoln et al., 2011; Sirot et al., 2008) who reported estimated THg exposures higher than expected based on the measured BTHg concentrations, toxico-kinetic modelling of our data shows an underestimated dietary THg exposure for several of the participants who had consumed a high proportion of self-captured fish. This might mainly be explained by the limitations in our Hg database, not being able to distinguish between regional differences, and possible higher Hg concentrations in fish closer to the shore. Hence, our findings confirm those of other studies; that the BTHg variability is strongly related to differences in THg concentration in fish both geographically and within and between species, as well as the amount of seafood consumed (Kosatsky et al., 2000; Lincoln et al., 2011; Mahaffey et al., 2009). The apparent underestimation of dietary exposure among those with the highest BTHg values also explains why none (JECFA's PTWI) or few (EPA's RfD) exceeded what is considered a safe exposure, whereas a higher proportion exceeded the corresponding guidance values for BTHg. The limitations in our food mercury database could however partly be compensated by including other factors in the regression models which explained the observed variance in BTHg.

The BTHg in the reference group (4.4 μg/L (0.6–30 μg/L) was higher than in several other population studies (Becker et al., 2002; Gundacker et al., 2006; Mahaffey et al., 2004; Wennberg et al., 2006). This is most likely due to the high average seafood consumption in our reference group and in agreement with the 1997 Norwegian general adult population dietary survey reporting an average fish consumption of 65 g/day (Johansson and Solvoll, 1999). The BTHg concentrations are comparable with those reported previously from Norway (Meltzer et al., 1994) and in studies among high seafood consumers in e.g. Sweden (Johnsson et al., 2004; Måge and Frantzen, 2009), Finland (Airaksinen et al., 2010), France (Sirot et al., 2008) and North America (Kosatsky et al., 2000; Lincoln et al., 2011). The mean BTHg concentration among women <45 years (2.6 ± 1.7 μg/L (0.6–7.7 μg/L)) in this study was slightly higher than, but still in the same range as in pregnant Norwegian women in two recent studies (Brantsæter et al., 2010; Hansen et al., 2011). On the other hand, BTHg concentrations in the present study were generally lower than in high fish consumers in the Mediterranean area (Barregård, 2005; Buzina et al., 1995; Gibicar et al., 2006) who consume Mediterranean fish species with higher Hg contents. The fish species most commonly eaten among the NFG study part B participants and among Norwegian women participating in the EPIC study were cod, saithe and farmed salmon, which are species generally low in Hg content (Bergsten, 2004; Welch et al., 2002). The observation that lean fish consumption appeared to have the highest influence on BTHg in regression analyses might be connected to the fact that some commonly eaten lean marine species (e.g. coastal cod) are often self-captured, and might be more affected by the local environment than self-captured migrating oily species such as herring and mackerel. In addition, fresh water species less commonly eaten, but with generally high and varying mercury content (such as pike and perch), also belong to the group of lean fish.

The increase of BTHg concentrations with age can be explained by higher fish consumption with increasing age. People with shorter educational attainment apparently had higher exposure to Hg (Table 2). However, the influence of education on BTHg was not significant in the adjusted models. This is most likely because higher education is more common in younger age groups. Both positive and negative correlations between education and Hg concentration have been shown in other studies, possibly reflecting different dietary habits related to e.g. socio-economic factors and rurality (Gundacker et al., 2006; Hightower and Moore, 2003; Kosatsky et al., 2000). Men had on average higher BTHg concentrations than women, which also is a common trait in other studies (Airaksinen et al., 2010). Different food consumption patterns might be one of the main factors related to higher BTHg among men than women. However, inter- and intra-individual variation in metabolism, e.g. rate of absorption, excretion and organ distribution, might also explain the often observed sex-related differences (Canuel et al., 2006; Clarkson, 1997; Mergler et al., 2007). Other dietary factors, such as selenium (Mergler et al., 2007), fruit consumption (Passos et al., 2003), alcohol- (Gundacker et al., 2006) and fibre intake (Airaksinen et al., 2010) as well as genetic polymorphisms (Gundacker et al., 2010) may also influence Hg absorption or metabolism.

It has been hypothesised that a main contributor to dietary MeHg exposure in addition to seafood could be fishmeal when used as a source of protein for poultry, swine and cattle (Dorea, 2006; Lindberg et al., 2004). Other foods are believed to mostly contain IHg, which would most likely contribute little to BTHg, especially in frequent fish consumers not exposed occupationally to inorganic or elemental Hg (Lindberg et al., 2004; Mahaffey et al., 2004). We believe that the lack of precise data on THg concentration in other foods is a minor problem in our study. Most of the participants were frequent seafood consumers, and other foods contributed little to the total dietary exposure and were not significant in the models. With the exception of egg consumption, which was positively associated with BTHg in Model 3, we did not find any other positive associations between BTHg and consumption of other foods than fish. The reason for the interaction between municipality and egg consumption could be regional differences in egg consumption, which was a little higher among coastal participants than among inland participants. The egg variable also includes eggs from gulls, which contain more total Hg than hen's eggs (Supplemental Table 1). Consumption of such eggs is rare, but is associated with a traditional Northern coastal dietary pattern (Kvalem et al., 2009). Because other food groups are frequently consumed, and have low concentrations of Hg in comparison with seafood, they would not be expected to turn up as significant predictors of BTHg.

The BTHg concentration is considered a good biomarker of dietary exposure to MeHg in populations with stable dietary patterns and low IHg exposure (Sirot et al., 2008). As MeHg is mainly excreted through the faeces, UHg concentration is considered to reflect the exposure to IHg. The part of IHg originating from diet is uncertain, and amalgam fillings are known to be the main contributor to IHg in non-occupationally exposed individuals (Barregård, 2005; Lindberg et al., 2004; Mergler et al., 2007). Since no measure of dental amalgam restoration was included in the study, we attempted to control for the inorganic fraction of BTHg by using UHg in Model 3. Interestingly, this resulted in an additional 11% points higher explained variance of BTHg. The UHg was in the same range as previously found among non-occupationally exposed Norwegians at comparable age (Ellingsen et al., 2001). In this population, the mean IHg concentration in blood constituted approximately 30% of TBHg.

Limitations and strengths of the present study should be noted. The main strengths are the detailed information about dietary sources and the detailed information about demographic factors in addition to Hg measurements in blood and urine. Limitations in the database used for estimating dietary THg include that intra-species variation were not taken into consideration, as discussed above. However, although the FFQ has been shown to be valid for accurately ranking participants according to energy, nutrient and food intakes (Brantsæter et al., 2008), food frequency questionnaires are rather imprecise instruments for estimating intakes (Meltzer et al., 2008). In addition to the limitations pertaining to details and accuracy of the FFQ, the association between BTHg as estimated dietary THg exposure will also be influenced by the participants' ability to recall and average their habitual diet correctly. Increased focus on some food items can lead to over-reporting of the particular food item, while underreporting often is found when several questions are grouped into one category (Björnberg et al., 2005; Lincoln et al., 2011; Mina et al., 2008). The FFQ used in the present study was designed to capture consumption of a range of seafood items, but other species than those included may still represent sources of THg that has not been properly accounted for and consequently contributed to the perceived underestimation e.g., whale and seal meat as

well as exotic fish and more rarely consumed Norwegian fish species. Overall, underestimation of the higher dietary exposures appeared to be more the case than overestimation in the present study.

As estimates are based on 12 months dietary recall, the use of hair THg could have been a more appropriate biomarker than BTHg, which better reflects exposure over the last few months, because the average half-life of MeHg in blood is about 50–70 days (JECFA, 2003, 2006; Mergler et al., 2007). Seasonal differences in consumption patterns and species choice may translate to variation in BTHg through the year. For example, shellfish consumption is often associated with the summer months in Norway, and cod is often eaten excessively during certain parts of the year. In addition, non-fasting BTHg concentrations can possibly be more influenced by recent fish consumption.

The average BTHg concentrations and dietary exposure estimates for the 184 participants are not directly representative for the Norwegian population as a whole. However, the reference group can be considered representative for the NFG Study, which again was country representative. Possibly, an overrepresented proportion of participants with an interest in fishing and hunting chose to participate in the NFG Study. This could explain the high proportion of participants reporting a high amount of self-captured fish in their diet. Anyway, we believe the participants cover a possible range of background exposure in Norway, which was the purpose of the study design.

Since concentrations of Hg in commonly consumed seafood in Norway are usually low to moderate, most of the species of concern are found in inland waters; however a low proportion of the population eat these species regularly. Even though we found higher dietary exposure and blood concentrations among participants living close to the coast in the present study, it is important that women of fertile age follow the official Norwegian dietary recommendations regarding certain fresh- and saltwater species in order to avoid high Hg exposure.

In conclusion, seafood was the main contributor to dietary THg exposure in this group of Norwegians, but the JECFA PTWI for MeHg was not exceeded by the participants. Seafood consumption together with demographic and other variables explained up to 65% of the observed variation in BTHg. Two of the participants had BTHg slightly above a value equivalent to the JECFA PTWI, but none of them were women in fertile age. There is a broad consensus that the benefit of fish consumption in general outweighs the risk posed by environmental toxic compounds like Hg. However, in a public health perspective, the beneficial effects of fish consumption might have been larger if Hg did not interfere, stressing the need for further emission reductions.

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Appendix A. Supplementary data

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