

Risk Assessment of Methylmercury in Fish

by Kierstin Petersson-Grawé, Gabriela Concha and Emma Ankarberg



**LIVSMEDELS
VERKET**

NATIONAL FOOD
ADMINISTRATION, Sweden

Produktion:

Livsmedelsverket, Box 622
SE-751 26 Uppsala, Sweden

Teknisk redaktör:

Merethe Andersen

Tryck:

Kopieringshuset, Uppsala
Uppsala 2008-04-04

Livsmedelsverkets rapportserie är avsedd för publicering av projektrapporter, metodprövningar, utredningar m m. I serien ingår även reserapporter och konferensmaterial. För innehållet svarar författarna själva.

Rapporterna utges i varierande upplagor och tilltrycks i mån av efterfrågan. De kan rekvireras från Livsmedelsverkets kundtjänst (tel 018-17 55 06) till självkostnadspris (kopieringskostnad + expeditonsavgift).

Contents

Preface.....	3
Introduction	4
Concentrations in food	5
Fish.....	5
Other foods.....	8
Cooking.....	8
Exposure.....	9
Fish consumption	9
Intake calculations.....	11
Concentrations in the body.....	15
Effects in humans.....	20
Effects of prenatal exposure on the central nervous system	20
Cardiovascular effects	23
Immune system	25
Risk assessments	27
Risk characterisation	30
Comparisons between intake/exposure and tolerable intake.....	30
Comparison between exposure on the Faroe Islands and in Sweden.....	31
Comparisons between exposure in Sweden and effect levels	32
Conclusions	34
References	40
Appendix	45

Preface

During 2006 and 2007, the National Food Administration has been working with the scientific data for a revision of the dietary advice on fish containing high concentrations of methyl mercury (MeHg) and persistent halogenated and organic environmental pollutants. This report summarises current knowledge on concentrations of MeHg in consumption fish. It also describes the exposure to which the Swedish consumer is subjected and the epidemiological studies that have been carried out on various population groups in the world. The most important risk assessments are also summarised. Finally, the exposure of the Swedish population is compared against the various risk levels that have been produced in the risk assessments.

The National Food Administration's advice on fish consumption has so far been based on separate nutritional and toxicological evaluations. However, in the revision of the advice as regards polluted fish, a health-based risk-benefit analysis of such consumption will be carried out before the revised advice is formulated. Knowledge of the beneficial aspects of fish consumption have been summarised within the project "*Risk-Benefit Analysis of Fish Consumption*".

Many experts, both within the National Food Administration and externally, contributed to the content of this report. We would like to express particular gratitude to the participants in the National Food Administration's expert meeting "*Environmental pollutants in fish - a health risk?*", which was held at the end of March 2006. Particularly warm thanks also go to Professor Staffan Skerfving, University of Lund, who examined the scientific basis of the relationship between exposure to methyl mercury and the risk of cardiovascular disease.

The project group comprised Emma Ankarberg (F/TO), Marie Aune (F/K2), Gabriela Concha (F/TO) Per Ola Darnerud (F/TO), Anders Glynn (project leader, F/TO), Sanna Lignell (F/TO), Kierstin Peterson-Grawé (F/TO) and Anna Törnkvist (F/K2).

Introduction

Mercury is a metal that occurs naturally in the environment, but humans have considerably increased the amount of mercury in circulation. In soil, water and sediment, inorganic mercury is converted to methyl mercury (MeHg). In the middle of the 1960s, high mercury concentrations were found in fish caught in Sweden in areas of water situated downstream from sources of emission of mercury compounds to water. To prevent fish with high concentrations of methyl mercury being sold commercially, blacklisting of certain waters with high concentrations was introduced in 1967. This blacklisting included a ban on the sale or transfer of fish from these waters. In the 1960s, bans on the use of mercury within the paper pulp industry began to be introduced. In 1996, a ban on mercury dipping of seed was also introduced.

It has been estimated that the mercury concentration in a one-kilo pike exceeds 1 mg/kg in approx. 10 000 inland lakes in Sweden, which bears witness to the fact that the decline in mercury concentrations in fish proceeds very slowly. In 1991 blacklisting of lakes and water courses in Sweden ceased, while in the same year the maximum level for mercury in fish was lowered from 1 mg/kg to 0.5 mg/kg, although with certain fish species excepted (for example pike and eel).

A number of metals are mentioned in the national environmental targets, in particular within a *Toxin-free Environment*. The environmental target requires e.g. that the concentration of MeHg in nature be near the background concentrations, which means that the concentration in fish should be 0.2 mg/kg or up to 0.5 mg/kg for certain nutrient-poor forest lakes.

As early as 1967, the authorities introduced dietary advice on consumption of certain fish that could contain high concentrations of mercury. According to these recommendations, fish with mercury concentrations between 0.2-1.0 mg/kg should not be eaten more than once a week. The dietary advice on fish that can contain high concentrations of mercury has since been revised a number of times, and has become increasingly clear in terms of recommendations that women who are pregnant, breastfeeding or planning to have a baby should avoid certain fish that can have high concentrations of methyl mercury (pike, perch, pike-perch, burbot, eel and giant halibut).

Concentrations in food

Fish

The major source of exposure to methyl mercury (MeHg) is fish. The majority of the mercury in fish, 75-100 %, occurs as MeHg and the remainder as inorganic Hg [1-3]. Usually, the concentration of total-mercury (total-Hg) is measured in fish muscle, while in calculations of exposure to Hg a common assumption is that 100 % of total-Hg in fish consists of MeHg. The mercury concentration varies greatly in indigenous fish species that are particularly interesting for calculation of mercury exposure and depends e.g. on catch site [4]. Fish in nutrient-poor forest lakes have higher mercury concentration than fish in nutrient-rich plains lakes. Other factors that affect the mercury concentration include e.g. the amount of mercury in circulation and a range of chemical, physical and biological factors in the water area [4]. In Skåne, for example, it is reported that the mercury concentration in pike varies at least 10-fold between lakes, from <0.1 mg/kg to over 1 mg/kg in one-kilo pike [5]. Predatory fish have higher Hg concentrations than other species, but the concentration also increases with fish size. For Swedish conditions, it is pike, pike-perch, perch, burbot and eel in particular that can accumulate MeHg, but other species can also exhibit elevated Hg concentrations depending on catch site, e.g. salmonids caught in Lakes Vänern and Vättern [6, 7]. The concentrations of Hg in fish in inland waters are generally markedly elevated as a result of emissions of Hg to air and long distance spread. Local emissions of Hg also affect the Hg concentrations in fish, which can be seen e.g. in the north of Lake Vänern [8]. Coastal pike in areas without local influences have relatively low Hg concentrations [9].

As a basis for review of the dietary advice, the National Food Administration in collaboration with the Swedish Board of Fisheries and the Environmental Protection Agency has mapped the Hg concentrations in consumption fish. Sampling within the framework of the National Food Administration's monitoring activities is not carried out at national level, with the exception of certain checks at border stations. A small number of samples were taken in retail outlets on behalf of the National Food Administration in this mapping project. A summary of the mercury concentrations in consumption fish used in intake calculations carried out at the National Food Administration is shown in Table 1.

Table 1. Mercury concentrations in consumption fish. Revised table from [13].

Fish species	Catch site (n)	Hg conc. mg/kg fresh wt. (mean)	Sampling yr	Reference
Perch	Coast-caught (39)	0.24	2001-2002	National Food Administration
	Lake Vättern (5)	0.32		
Pike	Coast-caught, Norrland coast	0.22	1999	[9]
	Lake Vänern	0.46	1996/97	[6]
Pike-perch	Coast-caught (27)	0.11	2001-2002	National Food Administration [14]
	Lake Mälaren	0.23	2001	
Burbot	Coast-caught (30)	0.25	2001-2002	National Food Administration
	Lakes Vättern and Vänern (15)	0.6	2001-2002	
Salmon	Farmed Norwegian, bought in Swedish shops (10)	0.02	2001-2002	National Food Administration
	Norrland rivers (30)	0.09	2001-2002	
	River Mörrumsån (10)	0.06	2001-2002	
	Baltic salmon	0.09	2001-2002	
	Lake Vänern (20)	0.26	2001-2002	
	Lake Vättern (20)	0.29	2001-2002	
Mackerel	West coast (20)	0.03	2001-2002	National Food Administration
Char	Northern Lake Vättern (3)	0.34	2001	National Food Administration
Plaice	Skagerack (20)	0.04	2001	National Food Administration
North sea herring	Southern Norway	0.03	1995-1996	[15]
Baltic herring	Baltic Sea	0.03	2004	Finnish National Food Administration
Tuna	Fresh, bought in Swedish shops (6)	0.46	2001	National Food Administration
	Canned, bought in Swedish shops (14)	0.06	2001	
Cod	Fladen	0.05	1979-2002	[12]
Eel	Median value, inland lakes and coast (178)	0.06 0.005-0.9	1994-2004	National Food Administration
Trout	Norrland rivers (30)	0.15	2000-2001	National Food Administration
	River Mörrumsån (10)	0.07		
	Lake Vänern (20)	0.48		
	Lake Vättern (20)	0.42		
Crab	Ireland	0.14	1999-2002	National Food Administration
Prawns	West coast	0.04		
Crayfish		0.03		
Mussels	West coast	0.02		

The mean concentration for the most common marine fish species consumed (for example cod, plaice, mackerel, farmed salmon) was < 0.05 mg/kg, while the mean concentration for some species that accumulate MeHg was in the range 0.2-0.5 mg/kg. This applies in the first instance to species such as pike, perch, pike-perch and burbot, but also trout, char and salmon from Lakes Vänern and Vättern. Note that eel, which is one of the species mentioned in the consumption-restricting dietary advice, has lower Hg concentrations than other fish species mentioned in the dietary advice. In 178 samples analysed, mainly samples from the year 2000 onwards, of eel from a total of 18 catch sites, the median concentration was 0.06 mg Hg/kg and the mean concentration 0.13 mg Hg/kg (SD 0.18; variation 0.005-0.9 mg Hg/kg). Approx. 5 % of the samples exceeded 0.5 mg Hg/kg and those eels were caught in Lakes Vänern and Bolmen, plus one sample from a large eel caught in Kalmarsund. Other fish species that can contain high concentrations of Hg include certain marine predatory fish, for example halibut, certain species of tuna, swordfish, shark and ray, for which the mean concentrations have been reported to be in the range 0.7-1.8 mg/kg [3, 10, 11]. Canned tuna has lower Hg concentrations than the tuna that can be bought fresh or frozen. In a minor investigation at the National Food Administration, the median concentration was 0.06 mg/kg.

Studies on Hg concentrations in fish are also carried out by other actors (for example local or county authorities, water protection associations), often within the framework of the national and regional environmental monitoring programme. IVL Swedish Environmental Research Institute Ltd. is the database host for reporting on the studies on Hg concentrations in fish that are carried out within the framework of national and regional environmental monitoring. Appendix 1 presents a summary of the mercury concentrations in pike and perch from 16 and 17 counties respectively, mainly during the 1990s and later. The data were obtained from the IVL database (mercury in Biota, [http://www.ivl.se/db/plsql/dvsb_hg\\$.startup](http://www.ivl.se/db/plsql/dvsb_hg$.startup)) and from 11 county authorities. In each municipality, the mercury concentration in pike and perch from 10-20 lakes was analysed. This summary only provides an overview of the mercury situation in each county in total, and not in individual lakes. Information on concentrations in individual lakes can be found in the IVL database and at the respective county/municipality. The summary shows that the concentrations vary greatly. The EU maximum level for Hg is frequently exceeded in the case of pike (1.0 mg/kg fresh weight), and sometimes exceeded in the case of perch (0.5 mg/kg fresh weight). The mean concentration in pike varies from 0.1 to 2.2 mg/kg (median concentration 0.1-1.6) and in perch from 0.1 to 1.1 mg/kg (median concentration 0.0-0.9). Exceedence of maximum levels in pike and perch vary between 2,7 and 100 %. The summary shows that continued mapping of the local mercury situation in the counties is important.

Since pike is one of the species encompassed by the consumption-restricting dietary advice, it is very important that more recent data are included in the supporting data. Pike was therefore sampled on behalf of the National Food Administration during the Swedish Board of Fisheries fishing tests in 2005. The Hg concentration was analysed in 68 samples of pike with mean weight 1.1 ± 0.9 mg/kg from a total of 27 different lakes in 16 counties. These lakes are included in various series of the environmental monitoring programme and include nine limed lakes. The mean concentration was 0.6 ± 0.4 mg Hg/kg, the median 0.5 mg Hg/kg, and the total variation 0.06-2.1 mg Hg/kg. These values were only somewhat lower when the limed lakes were removed from the calculations. The maximum level for commercial pike is 1.0 mg/kg and this was exceeded in 10 samples (15 %), while the Hg concentration in 25 of the samples (37 %) was in the range 0.5-1.0 mg/kg.

No general continuous temporal trend monitoring regarding the mercury concentration in consumption fish has been carried out. In the Environmental Protection Agency's programme for environmental monitoring of freshwater, there are temporal trend data on mercury concentrations in pike from Storvindeln, perch from Skärgölen and Bälgsjön and char from Abiskojaure. The concentration in perch from Skärgölen increased by 5 % annually up to 2002, while no trends were observed in fish from the other lakes [12]. Fish are collected annually from 18 stations around Sweden, and this is going to be expanded to 30 stations.

Other foods

The contribution to MeHg exposure from other foods is estimated to be negligible, although a certain contribution can derive from fish meal in animal feed intended for pigs and poultry (Swedish Board of Agriculture, personal communication).

Cooking

Mercury is concentrated in protein-rich tissues such as muscles, liver and kidneys in fish [16]. During cooking, the Hg concentrations can become higher than in raw fish, since the relative concentration increases in cooked fish due to the loss of water and oils that occurs during cooking [17].

Exposure

Fish consumption

A compilation of existing data on fish consumption has been carried out recently [13] and in the National Food Administration's ongoing project "Risk-Benefit Analysis of Fish Consumption". The following provides a summary of both of these reports.

Adults

According to the latest national dietary investigation Riksmaten 1997-98, consumption of fish and shellfish among adults amounts to an average of 30-35 g/d [18]. However, fish consumption increases with age, from an average of approx. 25 g fish per day in younger people (18-34 years) to approx. 45 g per day in older people (35-74 years). These figures were based on the results of 7-day recording (menu diaries) of food intake. Details of consumption frequency were also collected through surveys. On average, fish and shellfish were consumed 6.5 times per month. Approximately 73 % of individuals in the age range 17-49 years and somewhat more (81 %) in the age range 50-74 years reported that they ate at least one fish meal per week.

Cod and similar fish plus processed fish (fish fingers, fish balls) were consumed most, followed by other sea fish, shellfish and canned fish. Older people reported higher consumption of oily fish such as herring products, salmon and salmonids, than younger people, while younger people preferred lean fish and processed fish products, for example fish fingers and fish balls.

Approximately half of all subjects (51-59 %) reported that they never eat pike, pike-perch, burbot or perch. This group also included approx. 5 % who did not answer the question or who answered that they did not know. Just over one-third, 38-42 %, reported that they consume pike, pike-perch, burbot or perch a few times per year.

Approximately 2-3 % of all subjects reported that they ate pike, pike-perch, burbot or perch 1-3 times per month, while 1 % of older women and men ate eel 1-3 times per month.

While 3.3 % of men and 1.7 % of older women responded that they ate these fish species 1 time/week, 0.7 % and 1.1 % responded that they ate pike, pike-perch,

burbot and perch a few times per week, in other words they deviated from the dietary advice. For women of childbearing age, 1.1 % reported that they consumed these fish species 1 time/week. No women of childbearing age consumed these fish species more often than 1 time/week, which means that the people in this group follow the dietary advice.

Consumption of eel was somewhat less than for pike, pike-perch, burbot and perch. Approx. three-quarters of individuals responded that they never eat eel. Most of those who eat eel do so a few times per year (20-42 %), while approx. 1 % of the men and older women replied that they eat eel 1-3 times per month.

In several Swedish studies, pregnant women reported a lower fish consumption than other adults; < 4-5.8 fish meals per month compared with 6.5 fish meals per month in Riksmaten [19-22].

In Swedish studies aimed at individuals who eat large amounts of fish (for example sport anglers, professional fishermen and their families), 5-12 fish meals per month have been reported [23-28].

Consumption of self-caught fish

The survey *Fishe 2005* [24, 29] indicates that of the approx. 1.8 million Swedes who fish, over 160 000 (9 %) of these individuals eat the fish they have caught at least 1 time/week, of which approx. 10 000 are women of fertile age. In total, this would correspond to approx. 1-2 % of the entire population eating self-caught fish at least 1 time/week. However, these figures are somewhat uncertain due to some skewedness in drop-out.

Among the population of Norrland, both in the interior and along the coast, and on the plains of Götaland and Svealand, it is common for people to eat large amounts of self-caught fish. Most women of fertile age who eat large amounts of this kind of fish can also be found in Norrland. Most catches by recreational anglers are made in small lakes and tarns and not in the great lakes or in sea areas. Perch and pike were the species caught to the absolute greatest extent.

Children and young people

In a nation-wide dietary investigation "Riksmaten – barn 2003" [30, 31] that included 590 four-year-olds, 889 children in school year 2 (8-9 years old) and 1016 children in school year 5 (11-12 years old), data on fish consumption have been collected. Freshwater fish (for example perch, pike, burbot, pike-perch) were seldom consumed. Twenty-six per cent of four-year-olds (148 of 574), 27 % of 8-9-year-olds (232 of 852) and 30 % of 11-12-year-olds (289 of 978) reported that

they consumed freshwater fish 1-4 times/year, while 66 % (4-year-olds), 59 % (8-9-year-olds) and 56 % (11-12-year-olds) never consumed freshwater fish. Fish fingers/fish balls and lean sea fish were the most frequently consumed types, with a mean daily consumption of 10 g (4-year-olds), 12 g (8-9-year-olds) and 14 g (11-12-year-olds). Approx. 20 % and 4.5 % of children reported that they eat fish fingers once and twice a week respectively. Between 11 and 18 % reported that they consume lean sea fish once a week. After fish fingers and lean sea fish, farmed salmon was the next most consumed fish type, with a mean daily consumption of between 2 and 3 g. The National Food Administration dietary recommendation to consume perch, pike, pike-perch and burbot at most once a week is followed by most children (girls and boys). Only 0.2 % of the 4-year-olds (1 of 574), 0.1 % of the 8-9-year-olds (1 of 852) and 0.4 % of the 11-12-year-olds (4 of 978) reported that they eat freshwater fish more than one time per week.

A total of 245 17-year-olds in Uppsala and Trollhättan were involved in a study of the dietary habits of young people that was carried out in 1995/96 [32]. Approximately 95 % of these young people reported that they ate fish and the mean consumption was 4.4 times per month (min-max 0.2-14.2).

Intake calculations

Adults

An intake calculation for MeHg has recently been made based on the National Food Administration's investigation of dietary habits Riksmaten [13]. Several scenarios were included in the calculations with different assumptions about Hg concentrations in fish. The median value for MeHg exposure in women of childbearing age (17-40 years) varied between 0.1-0.2 µg/kg body weight/week, depending on scenario. The highest exposure was observed in the worst-case scenario, where the pike consumed was assumed to have a mercury concentration of 1.0 mg/kg (i.e. corresponding to the maximum level), and the salmon a mercury concentration of 0.33 mg/kg, corresponding to measured concentrations in salmonids from Lake Vänern/Vättern. For women in the age range 17-40 years who ate large amounts of fish (95th percentile), intake varied between 0.3-0.8 µg/kg body weight/week depending on scenario.

Children and young people

Calculations of MeHg intake in children [31] were carried out based on the National Food Administration's investigation on children [30]. Exposure to mercury was assessed with the aid of questionnaires and concentration data from investigations carried out previously at the National Food Administration (Table 1). With the help of the questionnaires, where fish type and the estimated

consumption frequency during the past year were recorded, it was possible to estimate fish consumption. Consumption frequency was reported as one of the following options: Never, 1-4 times/year, 5-8 times/year, 9-12 times/year, 2-3 times/month, 1 time/week, 2 times/week or 3-6 times/week. Portion size was estimated with the help of the Food Formula, a folder with diagrammatic illustrations of foods and photographs of portion sizes. Fish types/dishes included in the intake calculations were lean sea fish (for example cod, coley, haddock), fish fingers/fish balls (50 % fish), farmed salmon, wild salmon from the Baltic Sea, freshwater fish (for example pike, perch, pike-perch, burbot), flat fish (for example plaice, halibut, turbot), canned tuna, North Sea herring/mackerel, Baltic herring, and shellfish.

Since the Hg concentration in pike can vary greatly, the intake calculations were made according to two consumption options. In one of these (Figure 1b), the Hg concentration in pike with moderately elevated Hg concentrations (0.46 mg/kg fresh weight) caught in Lake Vänern was used for calculation of MeHg intake via fish (pike, perch, pike-perch, burbot). In the other option (Figure 1a), the Hg concentration in coastal pike with low Hg concentrations (0.22 mg/kg fresh weight) was used for calculation of MeHg intake via fish.

Median intake of MeHg per kg body weight was lower in the 11-12-year-olds compared with the 4-year-olds and 8-9-year-olds. Median intake among the 4- and 8-9-year-olds was 0.15-0.20 µg/kg body weight and week, while the 95th percentile varied between 0.45 and 0.62 µg/kg body weight and week (Table 2). Among the 11-12-year-olds, the median intake of MeHg was 0.12-0.15 µg/kg body weight and week and the 95th percentile was 0.35-0.53 µg/kg body weight and week. The highest intake among children in all age groups was 0.84-3.1 µg/kg body weight and week (Table 2). No major differences were observed between boys and girls.

Since mean intake of MeHg from the respective fish type was calculated, it was found that the contribution from pike to the total MeHg intake was almost as great as the contribution from lean sea fish (10-36 % vs. 24-42 %; Figure 1 a,b). This was despite the fact that pike was one of the least consumed fish in all age groups (1.8-6.2 g/week). Consumption of lean sea fish, however, was almost as high (32-41 g/week) as consumption of fish fingers (35-56 g/week), but since fish fingers consist of approx. 50 % cod or similar fish the contribution to total MeHg intake was lower than that from lean sea fish (10-14 %). Median intake of mercury in the children was at the same level as in adults according to a similar study [13].

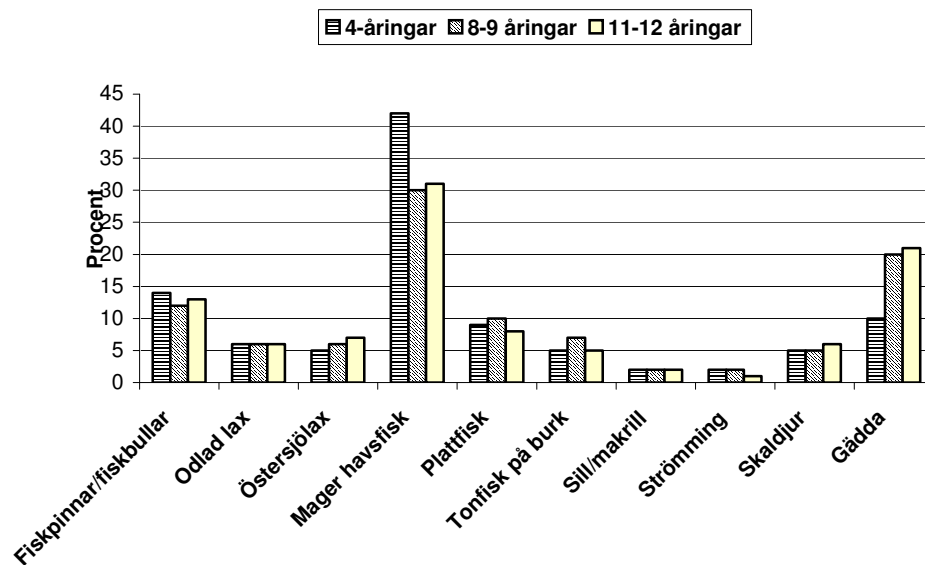


Figure 1a. Percentage contribution to total Hg exposure from different fish types/dishes in the different age groups; 4-year-olds, 8-9-year olds, and 11-12-year-olds (based on mean intake of MeHg from the respective fish type; with consumption of coastal pike with a concentration of 0.22 mg/kg fresh weight).

Note to Figure 1a:

Fiskpinnar/fiskbullar: fish fingers/fish balls

Odlad lax: farmed salmon

Östersjölax: Baltic salmon

Mager havsfisk: lean fish or lean sea fish

Plattfisk: flat fish

Tonfisk på burk: canned tuna

Sill/makrill: herring/mackerel

Strömming: Baltic herring

Skaldjur: seafood

Gädda: pike

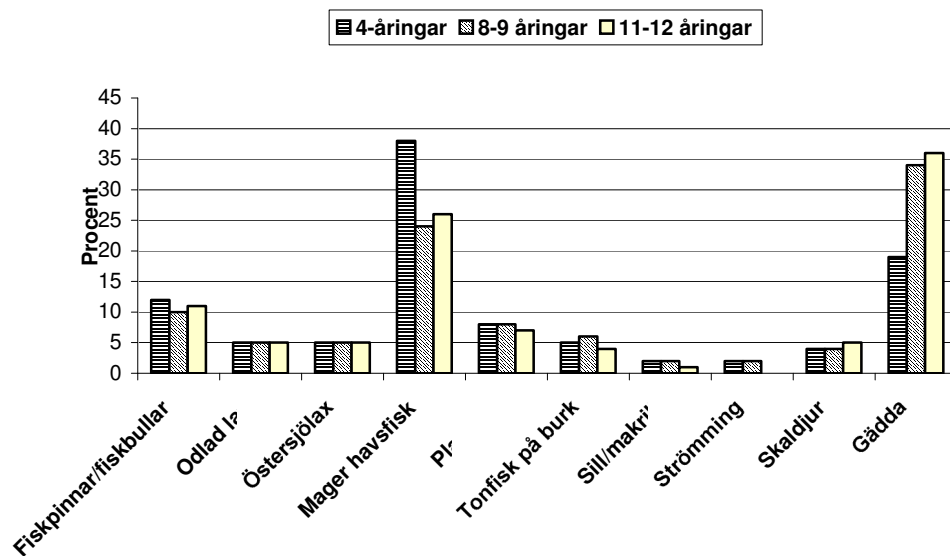


Figure 1b. Percentage contribution to total Hg exposure from different fish types/dishes in the different age groups; 4-year-olds, 8-9-year olds, and 11-12 year-olds (based on mean intake of MeHg from the respective fish type; with consumption of Lake Vänern pike with a concentration of 0.46 mg/kg fresh weight).

Note to Figure 1b:

Fiskpinnar/fiskbullar: fish fingers/fish balls

Odlad lax: farmed salmon

Östersjölax: Baltic salmon

Mager havsfisk: lean fish or lean sea fish

Plattfisk: flat fish

Tonfisk på burk: canned tuna

Sill/makrill: herring/mackerel

Strömming: Baltic herring

Skaldjur: seafood

Gädda: pike

Table 2. Intake of methyl mercury from fish (μg per kg body weight and week).

	4-year-olds ^a		4-year-olds ^b		8-9-year-olds ^a		8-9-year-olds ^b		11-12-year-olds ^a		11-12-year-olds ^b	
	G	B	G	B	G	B	G	B	G	B	G	B
Mean	0.21	0.21	0.24	0.24	0.20	0.19	0.24	0.23	0.14	0.16	0.17	0.20
SD	0.13	0.16	0.17	0.21	0.17	0.16	0.25	0.23	0.13	0.14	0.16	0.22
25th percentile	0.13	0.12	0.13	0.12	0.10	0.10	0.10	0.10	0.06	0.08	0.07	0.09
Median	0.19	0.17	0.20	0.18	0.16	0.15	0.18	0.18	0.12	0.13	0.14	0.15
75th percentile	0.27	0.26	0.31	0.29	0.26	0.23	0.29	0.28	0.19	0.20	0.21	0.24
95th percentile	0.45	0.50	0.51	0.54	0.46	0.45	0.62	0.58	0.35	0.38	0.44	0.53
Min	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Max	0.84	1.07	1.13	1.54	1.72	1.51	3.11	1.75	1.12	1.11	1.12	1.80
N	246	250	246	250	360	374	360	374	434	447	434	447

^aFor consumption of coastal pike with a concentration of 0.22 mg/kg fresh weight.

^bFor consumption of Lake Vänern pike with a concentration of 0.46 mg/kg fresh weight.

G=girl, B=boy.

Concentrations in the body

Established markers for MeHg exposure via fish consumption are Hg in red blood cells and hair, or MeHg in whole blood [33]. MeHg is predominantly bound to the haemoglobin in the red blood cells and the average half-life in humans is approx. 50 days, but half-life periods of up to 100 days have been reported [34]. The MeHg concentration in blood is therefore a measure of recent exposure, while the Hg concentration in hair can also provide information on exposure further back in time since Hg is incorporated into the hair as it grows. The ratio of MeHg-concentration in blood to Hg concentration in hair is generally reported to be 1:250, but the individual variation is very large (1:140-370). In most studies, the frequency of fish meals is positively correlated to the MeHg concentration in blood and hair. It is important to distinguish between different forms of Hg in exposure studies since exposure to inorganic Hg, e.g. via amalgam tooth fillings, results in an increased concentration of inorganic Hg in plasma [33]. Total concentration of Hg in whole blood of individuals who are not occupationally exposed to inorganic Hg therefore provides a measure of MeHg exposure via fish consumption and Hg exposure via dental amalgam.

Adults

The relationship between fish consumption and risk of heart attack was studied in a Swedish case control study that comprised 78 cases and 156 controls [35]. The Hg concentration in red blood cells of individuals who ate fish less than once a week was 3.3 ng Hg/g (80 % variation area 1.8-6.6 ng Hg/g), compared with 5.2 ng Hg/g (80 % variation area 2.1-10 ng Hg/g) in those who ate fish 1 time per

week or more . In individuals who reported that they had not eaten any fish in the past 2 years, the median concentration in blood was 0.10 $\mu\text{g MeHg/L}$ (min-max 0-1.0 $\mu\text{g MeHg/L}$) and T-Hg in hair was 0.06 $\mu\text{g/g}$ (min-max 0.04-0.32 $\mu\text{g/g}$) [36].

Pregnant women

Several studies of exposure to MeHg in pregnant women in Sweden have been carried out during the period 1994-2004. Figure 2 shows a summary of the MeHg concentrations measured in blood. The median concentrations varied between 0.3-0.9 $\mu\text{g/L}$, and the highest MeHg concentrations recorded varied between 1.7-6.8 $\mu\text{g/L}$ in the different studies.

The concentration was higher in umbilical cord blood than in maternal blood [37]. The median concentration of MeHg in umbilical cord blood was 1.4 $\mu\text{g/L}$ (max 4.8 $\mu\text{g/L}$) for women in Solna 1994-96 [37], and 1.3 $\mu\text{g/L}$ (min-max 0.10-5.7 $\mu\text{g/L}$) for women in Uppsala 1996-99 [38].

The Hg concentrations in the hair of pregnant women are shown in Figure 3. The median concentrations varied between 0.19-0.43 $\mu\text{g/g}$ and the highest concentrations in hair varied between 0.83-1.8 $\mu\text{g/g}$ in the different studies. In a study from 1989-1991, the concentration in hair of pregnant women was on average 0.27 $\mu\text{g/g}$ (min-max 0.07-0.96 $\mu\text{g/g}$) [39], i.e. at the same level as in the later studies.

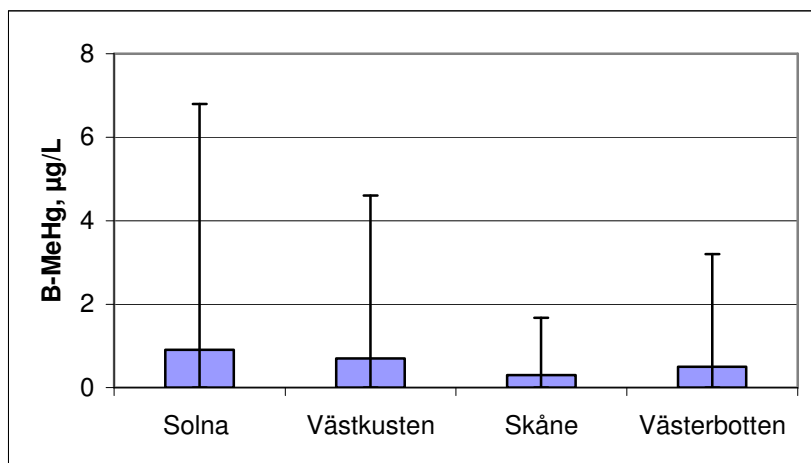


Figure 2. MeHg concentration in the blood of pregnant women from Solna 1994-96 [37], the west coast (Västskusten) 2001-02 [19], Skåne 2002-03 [20] and Västerbotten 2003-04 [21]. The bars show the median value and the lowest and highest concentrations recorded.

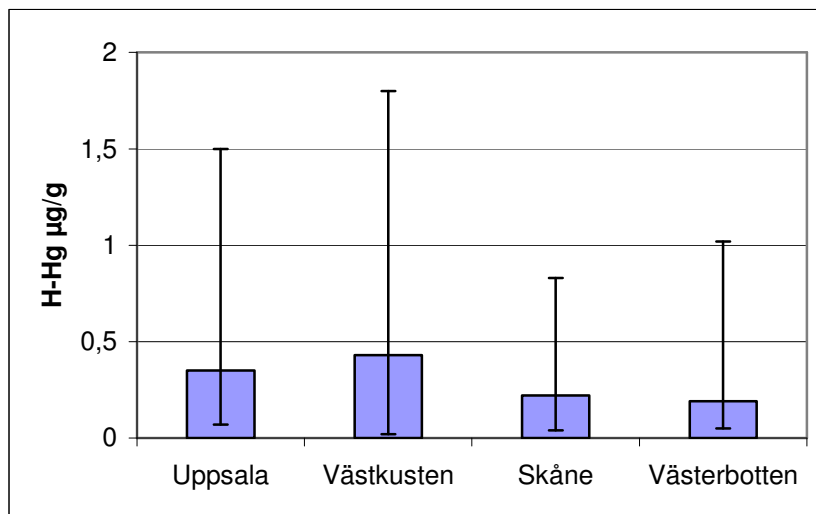


Figure 3. Hg concentration in the hair of pregnant women from Uppsala 1996-99 [38], Västkusten 2001-02 [19], Skåne 2002-03 [20] and Västerbotten 2003-04 [21]. The bars show the median value and the lowest and highest concentrations recorded.

High consumers of fish

High consumers of fish, professional fishermen and individuals who eat large amounts of fish from contaminated waters have a higher average Hg load than the average fish consumer in Sweden (Figure 4). It is important to note that the variation in MeHg exposure is considerable in these groups. In women who reported high consumption of fish the median concentration of MeHg in blood was 1.7 µg/L (min-max 0.30-14 µg/L) [40], while in women with high consumption of fish from Lake Vättern the blood concentration was 7.9 µg/L (min-max 0.9-31 µg/L) [23], and in older sport anglers with high consumption of fish it was 8.6 µg/L (min-max 4.4-24 µg/L) [26]. In professional fishermen the median concentration in blood was 4.3 µg/L [28].

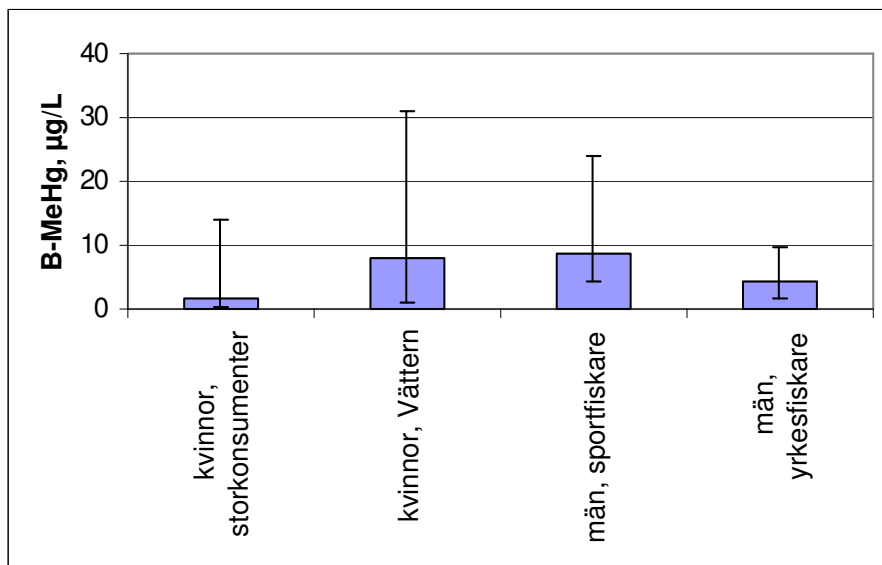


Figure 4. MeHg concentration in the blood of groups of individuals who eat large amounts of fish [40], [23], [26], [28]. The bars show the median value and the lowest and highest concentrations recorded.

Note to Figure 4:

kvinnor. storkonsumenter: women, high consumers

kvinnor. Vättern: women, Vättern

män. sportfiskare: men, sport anglers

män. yrkesfiskare: men, professional fishermen

The median concentration in the hair of women who ate fish 8 times per month was 0.7 µg/g (variation 0.08-6.6 µg/g) [41]. The median concentration of Hg in the hair of sport anglers with connections to Värmland was 0.9 µg/g (min-max 0.1-18.5 µg/g), while the median value for concentration in hair in those who reported consumption of freshwater fish at least once a week was 1.8 µg/g [25]. The average Hg concentration in fish in the area was 0.7 mg/kg. The calculated median value for Hg concentration in the hair of women who ate large amounts of fish from Lake Vättern was approx. 2 µg/g, based on MeHg concentration in blood and a blood/hair ratio of 1:250 [23]. In an investigation from 1985, the Hg concentration in hair in individuals with high consumption of freshwater fish was on average 3.2 ± 2.3 (SD) (min-max 0.3-10.8 µg/g) [42].

Children and young people

The average Hg concentration in blood in 245 Swedish 17-year-olds from Uppsala and Trollhättan, sampling years 1995/96, was 1.1 µg/L (min-max <0.7-5.8) [32]. No correlation was found between amalgam fillings and Hg concentration in blood, despite the fact that 39 % of these young people had such fillings.

Temporal trends

Blood samples collected from 600 randomly selected adult individuals (25-74 years) in Västerbotten and Norrbotten during the period 1990-1999 were analysed with respect to mercury [43]. The concentration of Hg in red blood cells was positively correlated to fish consumption, age and alcohol consumption. Linear regression showed an annual decrease in Hg concentration in the blood cells of men and women of 5.8 % (CI 4.4-7.0 %) after correction for age and fish consumption. However, this decrease in Hg concentration could have been due to a lower concentration in locally caught fish or to a change in the type of fish eaten.

Effects in humans

The scientific documentation on the effects of MeHg in humans and in animal models is very extensive. Comprehensive reviews have been published in the international risk assessments carried out recently [44-47]. The following describes the studies that have had the greatest significance for the final evaluation in the international risk assessments. For a more detailed description see the above-mentioned risk assessments. In Japan and Iraq, events occurred during the 1950s, 1960s and 1970s that led to a large number of people being affected by MeHg poisoning [48, 49]. The effects reported were in many cases very serious. In adults the symptoms appeared as damage to the central and peripheral nervous system. At lower exposure effects were also observed on the central nervous system of foetuses without any effects being observed in the pregnant women.

Effects of prenatal exposure on the central nervous system

A lowest exposure level that did not cause effects on the central nervous system (CNS) of the foetus could not be established and it was considered important to identify such a level. Therefore, during the 1980s and 1990s a number of epidemiological studies were carried out world-wide on population groups that consume large amounts of fish and therefore have higher chronic exposure to MeHg than other population groups. Indications that increased prenatal exposure to MeHg leads to effects on neurological status (for example muscle reflexes, muscle tension, fine motor activity) have been observed in some studies [50-52], while others have not found any such correlation [53-55]. In neurophysiological studies of children on the Faroe Islands and Madeira, a correlation has been reported between increased MeHg exposure and depressed activity in the brain in response to visual or aural stimuli [56, 57]. However, it should be noted that measurement of depressed activity in the brain (so-called brain auditory evoked potential or visual evoked potential) is an area that is relatively unexplored. Nevertheless, an advantage with such parameters is that they are not affected by e.g. socioeconomic factors, as is the case with many neuropsychological empirical methods. Sight and hearing were not correlated to exposure to MeHg in children on the Faroe Islands [51], while indications of a slight hearing impairment effect were observed in a smaller study of children in Ecuador [58]. A correlation between early exposure to MeHg and delayed motor development (sitting, crawling, walking) was observed in the Faroe Islands (postnatal exposure) [59]

while no such correlation has been reported in children in the Seychelles [60, 61] and in Peru [62].

The epidemiological studies that have had the greatest significance in the risk assessment of MeHg have been carried out in New Zealand, the Faroe Islands and the Seychelles. In New Zealand, development was compared in 74 children whose mothers had high exposure to MeHg ($> 6 \mu\text{g/g}$ in hair) during pregnancy (variation 6-86 $\mu\text{g/g}$, most in the concentration range 6-10 $\mu\text{g/g}$) and children who had lower prenatal exposure to MeHg [63]. At 4 years of age, the group with high prenatal MeHg exposure had worse test results in terms of motor, neurological and cognitive functions than the group with low prenatal MeHg exposure. At 6 years of age extensive tests were carried out on the children's development but with more children in the control group than in the previous study [64]. The outcome varied somewhat depending on the choice of statistical method used for analysis, but in general a correlation was found between increased prenatal exposure to MeHg and impaired general cognitive and linguistic ability [64, 65]. However, the study in New Zealand has received criticism because e.g. it was not subjected to external scientific review and the number of mother-child pairs was small.

In the Faroe Islands, a prospective study was carried out on 917 mother-child pairs, which were followed from birth of the babies in 1986-87 onwards [51]. Approximately half the women ate fish three times or more per week, mostly consisting of fish species with low concentrations of Hg. However, they often ate whale meat in particular and this can contain high concentrations of Hg, which had greater significance for MeHg exposure than consumption of fish [66]. They also ate whale blubber, which contains elevated concentrations of PCBs [67]. The Hg concentration in maternal hair at parturition was on average 4.3 $\mu\text{g/g}$, and the interquartile spread (25th-75th percentile) was 2.6-7.7 $\mu\text{g/g}$ [51]. In 15 % of the mothers the Hg concentration in hair was $< 10 \mu\text{g/g}$, and the highest concentration recorded in hair was 351 $\mu\text{g/g}$. The concentration of MeHg in umbilical cord blood was used as a measure of foetal exposure, and the mean concentration in umbilical cord blood was 22.8 $\mu\text{g/L}$. At 7 years of age the neurophysiological function and neurological status of the children was measured, and also their cognitive abilities. A correlation was found between MeHg exposure and impaired test results in terms of concentration, language and memory, and also to a certain extent motor and visuospatial function. These differences remained when children whose mothers had a hair content over 10 $\mu\text{g Hg/g}$ were excluded from the calculations. A potential source of error in the study is that there was simultaneous exposure to PCBs in whale blubber, which could itself have affected the test results. However after correction for PCB exposure in approx. half the children studied, it was found that the statistical differences in test results between the groups with low and high MeHg exposure remained [68]. In a later analysis of the material it was concluded that MeHg is probably a more important factor in

explaining the central nervous system effects observed than PCB, but that simultaneous PCB exposure could possibly enhance the MeHg-induced effects at high MeHg exposure, although it is emphasised that interpretation is difficult [69]. The effects of simultaneous exposure to MeHg and PCBs have also been studied in a group of 212 children in the USA [70]. The median Hg concentration in maternal hair during pregnancy was 0.50 µg/g, and in the American children a weak interaction was found between prenatal exposure to MeHg and PCB, but the relationship was difficult to interpret. Fish consumption was low, with those who ate large amounts of fish reporting on average 15 fish meals per year. The interaction effects observed at 38 months of age were not observed at 54 months of age. Further studies are probably required to clarify any interactions between PCB and MeHg in prenatal exposure.

In a similar longitudinal study on the Seychelles group of islands, 779 mother-child pairs were monitored [71]. Approximately 85 % of the population on the Seychelles eat marine fish every day, and the women who took part in the study reported that they ate fish approx. 12 times per week. The median concentration of Hg in fish varied between 0.5-0.25 mg/kg depending on fish species. The concentration of Hg in maternal hair during pregnancy was selected as a measure of exposure to MeHg, and the mean concentration was 6.8 µg/g ± 4.5 (variation 0.9-25.8 µg/g). This study was preceded by a pilot study that encompassed 804 children whose mothers had median Hg concentrations in hair of 6.6 µg/g during pregnancy [72]. The pilot study found that the results in a development test carried out between 1 and 25 months of age were more often abnormal or doubtful in children whose mothers had Hg concentrations in hair above 12 µg/g than in children whose mothers had lower Hg concentrations in hair [72]. At 9 years of age, 87 of the children from the pilot study were tested with regard to cognitive ability, vision and motor ability [73]. No negative relationship was observed between children's cognitive ability/neurological function and prenatal MeHg-exposure, although there was a positive relationship in boys. In the so-called main study no correlation was found between prenatal exposure to MeHg and mental and psychomotor development in approx. 740-780 children at 6.5, 19 and 29 months of age [55, 74]. At approx. 5 years of age, 711 of the children were tested with regard to e.g. general cognitive ability, social behaviour, language, mathematical and spatial ability [75]. No negative correlation was found between early exposure to MeHg and child development, but there was a positive correlation between prenatal MeHg exposure and the results of language ability and problem solving, and in boys also spatial ability. At 9 years of age, 21 different parameters for development of the central nervous system in children were investigated [76]. Improved test results for hyperactivity index with increasing prenatal MeHg exposure and a negative relationship with the outcome of one of the tests in boys were reported, but overall the results were deemed not to be of biological relevance. The results from tests carried out at 11 years of age

have recently been published [77]. No relationship was observed between prenatal MeHg-exposure and intelligence quotient, reading, mathematical ability, social behaviour and memory.

The results from a large number of animal studies on both rodents and primates also show that the effects on the central nervous system during development after prenatal or early postnatal exposure, similar to those observed in humans, are sensitive parameters.

Cardiovascular effects

The first publication reporting a correlation between MeHg-exposure and risk of heart attack/mortality came from Finland and was based on a cohort of 1833 men in eastern Finland [78]. Fish consumption was high, on average 46.5 g/day (min-max 0-619 g/day) and some of the most common fish species were vendace, rainbow trout, pike and perch. The Hg concentration in hair was on average 1.92 (min-max 0-15.67 µg/g) and was correlated to fish consumption. Individuals who ate >30 g fish per day had a risk ratio (RR) for heart attack of 1.87 (CI 1.13-3.09) compared with individuals with lower fish consumption, and similarly the risk of heart attack was significantly elevated in those who had a Hg concentration in hair > 2 µg/g compared with those with < 2 µg/g (RR 1.69; CI 1.03-2.76). Mortality from heart attack and total mortality were also correlated to Hg concentration in hair. Two follow-up studies were later carried out on the cohort. These investigated the relationship between serum concentrations of n-3-fatty acids and morbidity in heart disease (197 cases of 'coronary events' according to the authors) [79]. In the group that had the 20 % highest serum concentrations of n-3-fatty acids, the risk of contracting disease was significantly lower (47 %) than in those who had the 20 % lowest serum concentrations of the fatty acids docosahexaenoic acid (DHA) and docosapentaenoic acid (DPA). In the one-third who had a Hg concentration in hair that exceeded >2 µg/g there was no clear protective effect of the n-3-fatty acids. However in the others the relationship between serum concentrations of n-3-fatty acids and the risk of heart disease was dose-related, with a protective effect with increasing serum concentrations. In a later follow-up study [80] similar results were reported. In total, 282 cases of acute coronary events being contracted were included in the study. The risk of contracting the disease was 1.07 (CI 0.77-1.49) in the one-third whose Hg concentrations in hair were in the range 0.84-2.03 µg/g compared with the risk in those with the lowest Hg concentrations in hair (< 0.84 µg/g). The one-third who had Hg concentrations in hair > 2.03 µg/g had a risk of 1.66 (CI 1.20-2.29) of contracting the disease. At Hg concentrations in hair < 2.03 µg/g a decreased risk of contracting the disease was found with increasing serum concentrations of

DHA+DPA (RR=0.69; CI 0.52-0.91), while no such correlation was observed at Hg concentrations in hair > 2.03 µg/g.

A Swedish prospective case-control study investigated the relationships between fish consumption, MeHg exposure and plasma levels of long-chain polyunsaturated fatty acids (P-PUFA), and the risk of suffering an acute heart attack [35]. The study included 78 cases and 156 matched controls. Those who ate fish less than once a week had a median Hg concentration in red blood cells (Ery-Hg) of 3.3 ng/g, while those who ate fish once a week or more often had a median value of 5.2 ng/g. Compared with the risk of morbidity in those who had Ery-Hg < 3 ng/g, the risk was 0.91 (CI 0.49-1.69) in those who had Ery-Hg 3-6 ng/g, while it was 0.43 (CI 0.19-0.95) for Ery-Hg > 6 ng/g. Ery-Hg and P-PUFA co-varied. A strong positive correlation was found between both P-PUFA and MeHg exposure and decreasing risk of suffering acute heart attack.

In a cross-sectional study of men who had suffered a heart attack and matched controls from several countries in Europe (Sweden not included), a correlation was found between Hg concentration in nails (N-Hg) and concentration of DHA in fatty tissue biopsies [81]. The risk of acute heart attack increased with increasing N-Hg, and decreased with increasing fatty tissue DHA.

A prospective study of 1014 men from eastern Finland found a significant relationship between thickening of the carotid artery over a 4-year period (a measure of progressive arteriosclerosis) and Hg concentration in hair [82]. The effect was observed in the group that had Hg concentrations in hair of > 2.81 µg/g compared with others. The Hg concentration in hair was on average 1.8 µg/g and the highest concentration 23.3 µg/g.

Effects on blood pressure have been reported in a study of 917 seven-year-old children from the Faroe Islands who were exposed to MeHg to varying degrees during the foetal stage [83]. The Hg concentration in umbilical cord blood was on average 31.77 µg/L (SD 29.02; min-max approx. 1-300 µg/L). Systolic and diastolic blood pressure increased by 14.6 and 13.9 mm Hg, respectively, when the Hg concentration in umbilical cord blood increased from 1 to 10 µg/L, i.e. in those with the lowest exposure levels; the effect was strongest in children with a birth weight < 3700 gram. No such effect was observed at higher exposure levels. The Hg concentration in maternal hair at parturition was also correlated to increasing blood pressure, but not as strongly as the Hg concentration in umbilical cord blood. In Swedish studies no relationship has been observed between MeHg exposure and blood pressure [35]. Cardiac rhythm variability at 7 years of age was also correlated to prenatal MeHg exposure and Hg concentration in hair at 7 years of age in children on the Faroe Islands [83]. At 14 years of age a similar relationship was also observed, but only with regard to the prenatal MeHg

exposure [57, 84]. These changes were considered to be an effect of changes to the brain stem.

Epidemiological data indicate that there is a relationship between high MeHg exposure and increased risk of acute heart attack, but the conclusions regarding the shape of the dose-response curve are partly uncertain. However, the Finnish studies indicate the risk of contracting cardiovascular disease can increase if the Hg concentration in hair exceeds 2 µg/g. In the Swedish studies that have been carried out no such relationship has been found, and this can probably be explained by the MeHg exposure having been considerably lower compared with e.g. that in the Finnish studies. On the contrary, the Swedish studies have found a positive relationship between MeHg exposure and decreasing risk of heart attack. This can be interpreted as the protective effect of the long-chain polyunsaturated fatty acids (and possibly also selenium) dominating up to a certain intake level after which the MeHg-induced negative effects take over.

There is extremely limited data support for evaluating whether a relationship exists between MeHg and stroke. Swedish data, based on a study population with low MeHg-exposure, do not indicate any positive relationship between MeHg and the risk of stroke. There are certain indications that exposure to MeHg during the foetal stage or childhood is correlated to increased blood pressure and minor changes in cardiac rhythm variability. The significance of these findings is unclear, but could be related to incidence of acute heart attack. In Swedish studies on adults no such relationship between exposure to MeHg and blood pressure was observed, but the exposure levels were low. More studies are needed to clarify this.

Immune system

Studies on humans

Knowledge of effects of exposure to methyl mercury on the immune system in humans is very limited. In a study of neonates in Quebec who had been exposed prenatally to both PCBs and MeHg, changes were found in the composition of T-, B- and NK-cells in umbilical cord blood and in cytokine response [85, 86].

Animal studies and *in vitro* models

The effects of MeHg on the immune system have been studied in a number of *in vivo* studies on rats and mice and in *in vitro* experiments. These studies vary in design and in the immunological parameters studied. It was not possible in any of the *in vivo* studies to identify a NOAEL (no observed adverse effect level), i.e. a dose at which no effects could be observed, while otherwise only one dose was

used. The effects reported after exposure of adult animals to MeHg include decreased thymus weight, reduced NK-cell activity, decreased resistance to virus infection, changes in the composition of T- and B-cells, and changes in mitogen response and autoimmunity [87-93]. Effects on development of the immune system from exposure to MeHg during the foetal period or immediately after birth have also been reported in some studies. Effects on thymus and spleen weight, changes in number of lymphocytes in the blood, NK-cell activity, mitogen response and composition of T- and B-cells have been reported [88, 94, 95]. Altered levels of essential trace elements such as iron, calcium, manganese and zinc have also been observed in mice exposed to MeHg in a virus infection model [96]. The sensitivity of the immunological response varies between different strains of mice, which could perhaps be explained by differences between different strains in demethylation of methyl mercury to inorganic mercury, which then affects the immunological system [97]. The studies on rodents that have been reported show that MeHg has the potential to affect the immune system in adults and foetuses. However these studies do not provide an unequivocal answer regarding the most sensitive immunological parameters, or regarding the direction in which MeHg influences the respective parameters. The extent to which MeHg exerts effects on the immune system at lower exposure levels and also the highest exposure level that does not give rise to effects are still unclear.

Risk assessments

Up until 2003, the provisional tolerable weekly intake (PTWI) for MeHg was set at 3.3 µg/kg body weight and week by the Joint FAO/WHO Expert Committee on Food Additives and Contaminants (JECFA) [45]. This assessment was based on neurotoxic effects in adults but it was considered that PTWI was not regarded as protecting pregnant women (i.e. the foetus) from effects. Based on new epidemiological studies from the Faroe Islands and the Seychelles on the effects on child development, JECFA has revised its risk assessment and now specifies a PTWI of 1.6 µg/kg body weight and week [46]. At this exposure level pregnant women and their foetuses are considered not to be at risk of any neurotoxic effects. In 2000, the American National Research Council (NRC) carried out an assessment of the risks of effects from exposure during the foetal stage based on the study from the Faroe Islands [44], which resulted in a reference dose (RfD) of 0.1 µg/body weight/day, which is equivalent to 0.7 µg/kg body weight and week. The assessments by NRC and JECFA thus resulted in highest tolerable exposure levels of 0.7 and 1.6 µg/kg body weight and week respectively, which corresponds to a mercury concentration in hair of 1.2 and 2.2 µg/g respectively, and in blood of 4.8 and 8.8 µg/L respectively.

PTWI and RfD are stated as amount of MeHg/kg body weight, but are based on epidemiological studies in which the measure of exposure was Hg concentration in hair or umbilical cord blood. To convert from concentration in hair or blood, a kinetic formula has been used, which means that a range of assumptions have been made [44, 46]:

$$d = C \times b \times V / (A \times f \times bw)$$

where:

C = concentration of mercury in blood (µg/L), poss. via conversion of hair/blood ratio

b = elimination constant (0.014/day)

V = blood volume

A = dose absorbed (0.95)

f = fraction of dose absorbed distributed in blood (0.05)

bw = body weight

d = dose (µg/day)

The PTWI produced by JECFA has been calculated on the basis of the exposure level at which effects were not observed in the Seychelles and Faroe Islands

studies. On the Seychelles, no effects were observed in children at a maternal hair concentration of 15.3 µg/g during pregnancy in the group with the highest exposure level [75], while the study on the Faroe Islands reported a so-called benchmark dose¹ (BMDL) of 12 µg/g [44, 98]. The mean value of these two studies is 14 µg/g, which has been used in calculation of PTWI [46].

NRC has calculated separate BMDLs for the Seychelles and Faroe Islands and a study from New Zealand, plus an integrated BMDL for all three studies, but elected to base the RfD on the Faroe Islands study alone (BMDL 12 µg/g) [44].

The fact that different assessments have arrived at different conclusions on the tolerable level is mainly due to a use of different uncertainty factors. NRC has applied an uncertainty factor of 10 in total, in order to take account of variation in kinetics between individuals and the indications of cardiovascular-related, immunotoxicological and late neurological effects. JECFA on the other hand has used a safety factor of 2 for variation in hair/blood ratio between individuals and a factor of 3.2 for other variation in kinetics between individuals, i.e. a total uncertainty factor of 6.4.

During 2006, JECFA investigated whether the current PTWI should include others in the population in addition to pregnant women, i.e. children and the elderly. For adult individuals apart from women of childbearing age, it has been estimated that exposure can be in the order of twice PTWI without any danger of health effects arising [47]. For children up to around 17 years of age, it is assumed that sensitivity is not greater than in the foetus but the possibility cannot be ruled out that the sensitivity is greater than in adults and therefore a PTWI of 1.6 µg/kg body weight and week is recommended for this age group.

Table 3 shows NOEL and BMDL values for the three studies in the Faroe Islands, Seychelles and New Zealand based on Hg concentration in maternal hair during pregnancy. The researchers who carried out the study on the Faroe Islands have reported benchmark calculations on their data and determined a lowest BMDL at a Hg concentration of ~5 µg/L in umbilical cord blood, which corresponded to a T-Hg concentration in maternal hair of ~1 µg/g [98]. The BMDL varied between 1.6–12.7 µg Hg/L in umbilical cord blood for five different test parameters (e.g. motor ability, concentration, language) on assumption of a logarithmic dose-response model. With the starting point of concentrations measured in hair, the BMDL is instead 2.2-6.8 µg/g on assumption of a logarithmic dose-response model and 9.4-14 µg/g on assumption of a linear dose-response model.

¹ The so-called benchmark dose (BMD) concept has been proposed to replace NOAEL in risk assessment. The BMD is calculated from a dose-response model that has been fitted to data. The lower confidence limit of the BMD, the BMDL, is used for considering uncertainties in the data.

The authors point out that the BMDL varies depending on the dose-response model used in the analysis. In the NRC assessment a different dose-response model (linear) was assumed for the Faroe Islands study, which resulted in a representative BMDL for a Hg concentration in hair of 12 µg/g [44]. The choice of dose-response model and other assumptions made that were important for the outcome, has been discussed [44, 99].

Table 3. Summary of NOEL and BMDL in the most important epidemiological studies of the relationship between prenatal exposure to MeHg and effects on the CNS during childhood. The measure of exposure is maternal Hg concentration in hair (µg/g) during pregnancy.

Study	NOEL	BMDL	Source
Faroe Islands		2.2-6.8*	[98]
		9.4-14**	[98]
		12	[44]
Seychelles	15.3	25 (19-30)	[75]
			[100]
New Zealand		7.4-10	[65]
Faroe Islands + Seychelles + New Zealand		7	[44]

Note: the BMDL corresponds to a 5 % response (BMDL₀₅), except for the Seychelles study where the BMDL corresponds to a 10 % response (BMDL₁₀).

* logarithmic dose-response.

** linear dose-response.

Risk characterisation

The risk assessment from NRC [44] and that from JECFA [46] report tolerable intakes that differ by a factor of 2.3. The difference consists of the assumptions regarding uncertainty and variability made by both expert committees in their assessments. With the uncertainties occurring in these assessments, it is not unreasonable to assume that the tolerable intake lies somewhere in the range between the PTWI and the RfD. Therefore in the risk characterisation below, comparisons are made of exposure against both PTWI and RfD.

Comparisons between intake/exposure and tolerable intake

The most sensitive group in the population is pregnant women, due to the greater sensitivity of effects in the foetus. In the latest investigations of exposure in a random selection of pregnant women in Sweden, the blood and hair concentrations corresponding to the JECFA PTWI value of 1.6 µg/kg body weight/week were not exceeded, while 0-4 % exceeded the American reference dose of 0.7 µg/kg body weight/week. When this is translated to the entire population, according to the calculations there may be up to approx. 2400-8500 pregnant women/year who exceed the reference dose somewhat [19]. Theoretical calculations of intake indicate that the JECFA PTWI value can be exceeded by pregnant women with high consumption of fish with elevated concentrations of MeHg [13].

On the basis of the JECFA and NRC assessments, it is not likely that any negative effects will arise in the foetus at these actual exposure levels, but the safety margin is smaller for those with higher MeHg exposure.

For men and older women, PTWI can be exceeded by a factor of 2 according to the JECFA assessment. In targeted studies of exposure in high consumers of fish the average exposure has been below such a level, but the individual variation is very great and some cases of PTWI being exceeded have been reported. No supporting data are available for establishing how frequently it is exceeded at present. In theoretical calculations, a value twice the PTWI is exceeded by 0-0.3 % of men and older women [13]. For the vast majority of men and older women the MeHg exposure is at a safe level.

Calculations based on the National Food Administration study on children indicate that 0.5-3 % and 0.1-0.4 % of children in the ages 4, 8-9 and 11-12 years can exceed the American reference dose and the JECFA PTWI. This can occur in children who eat fish daily, 40-150 g, or who regularly eat pike or other fish with elevated MeHg concentrations several times a week.

Comparison between exposure on the Faroe Islands and in Sweden

The study from the Faroe Islands is the most complete study that reports the relationship between prenatal exposure to MeHg and effects on development of the nervous system. It can therefore be interesting to compare the exposure levels in Sweden with those reported from the Faroe Islands. Table 4 shows the Hg concentrations in umbilical cord blood in pregnant women on the Faroe Islands [101] and in Sweden [38]. The concentrations are approx. 20 times lower in the Swedish women compared with the concentrations in the women from the Faroe Islands. Table 5 presents the Hg concentrations in the hair of pregnant women on the Faroe Islands [38, 101] and in Sweden [38], and those in high consumers of fish [41]. The average Hg concentrations in hair are 10-20 times lower in pregnant women in Sweden compared with the Faroe Islands. Women in Sweden who eat large amounts of fish or fish with high mercury concentrations have on average approx. 2-6 times lower Hg concentration in hair than women on the Faroe Islands.

Table 4. Comparison between reported MeHg concentrations in umbilical cord blood from women on the Faroe Islands 1986-1987 [102] and Sweden 1996-1999 [38].

MeHg in umbilical cord blood (µg/L)	Pregnant women, Faroe Islands	Pregnant women, Sweden
25th percentile	13.4	
Median	22.9	1.3
75th percentile	41.3	
Max	351	5.7

Table 5. Mercury concentration in hair of pregnant women in the Faroe Islands and Sweden, and of women in Sweden who eat large amounts of fish.

Hg concentration in hair ($\mu\text{g/g}$)	Pregnant women, Faroe Islands [101]	Pregnant women, Sweden [19-21, 38]	High consumption, women Sweden [41]	High consumption, women Sweden Vättern* [23]
<i>n</i>	914	422	126	37
Median	4.27	0.19-0.43	0.70	2
75th percentile	7.7			
90th percentile			1.6	
Max	39	0.8-1.8	6.6	7.8

*Calculated Hg concentration in hair.

Comparisons between exposure in Sweden and effect levels

From the three studies in New Zealand, the Faroe Islands and the Seychelles, several benchmark doses have been calculated (Table 3). The BMDL varies between studies and also depending on the dose-response model used. The lowest BMDL values reported from the Faroe Islands are in the range 2.2-6.8 $\mu\text{g/g}$ in maternal hair, as well as the NRC's combined BMDL of 7 $\mu\text{g/g}$ for all three studies. The NRC assessment was based on a BMDL of 12 $\mu\text{g/g}$ in maternal hair based only on the Faroe Islands study. The highest BMDL of 25 $\mu\text{g/g}$ in maternal hair is based on the Seychelles study. Comparing the average Hg concentrations (0.19-0.43 $\mu\text{g/g}$; Table 5) reported in recent studies on pregnant Swedish women, the margin to the BMDL is 5- to 36-fold based on the most conservative modelling of data from the Faroe Islands study. With a different assumption of the dose-response model according to NRC, the margin is 27-63-fold between exposure in pregnant women in Sweden and the BMDL based on data from the Faroe Islands. The margin to the BMDL from the Seychelles study is in the range 58-130-fold.

Table 6. Margin between the BMDL, based on the Faroe Islands and Seychelles studies, and average exposure in pregnant women in Sweden. Data taken from Tables 3 and 5.

Hg in hair ($\mu\text{g/g}$) Pregnant women, Sweden*	BMDL**	Margin
<i>Median</i>		
0.19-0.43	2.2-6.8	5-36
	12	27-63
	25	58-130

*see Table 5 for references to the exposure studies.

**see Table 3 for references to BMDL.

The limited data that are available on exposure in women in Sweden who eat large amounts of fish indicate that there may be groups that lie close to or at the level of the most conservative BMDLs.

The average Hg concentrations in hair reported in recently performed Swedish studies targeting high consumers of fish with elevated concentrations [23, 25] lie close to the level in the Finnish studies in which a correlation has been observed between exposure to MeHg and morbidity from heart attack [79, 80, 103]. The supporting data are very limited but the possibility cannot be excluded that there are groups in the Swedish population with high consumption of fish with elevated Hg concentrations who have such a high MeHg intake that the risk of MeHg-induced cardiovascular disease may be elevated. It is currently not possible to quantify the size of this group, but it is probably limited to those who very frequently eat self-caught fish with elevated MeHg concentrations.

Against the background of these studies and the calculations of MeHg exposure that have been made, it can be concluded that under Swedish conditions there is a certain risk of elevated intake of MeHg if the consumption pattern is altered in favour of fish with higher concentrations of MeHg.

Conclusions

Concentrations in fish

The major source of exposure to methyl mercury (MeHg) is fish. Mercury in fish occurs mainly, 75-100 %, as MeHg and the remainder as inorganic Hg. The mercury concentration varies greatly in the domestic fish species that are of particular interest for calculation of mercury exposure and depends e.g. on catch site. Fish in nutrient-poor forest lakes have higher mercury concentrations than fish in nutrient-rich plains lakes. Predatory fish have higher Hg concentrations than other fish species, but the concentration also increases with fish size. For Swedish conditions, it is primarily pike, pike-perch, perch, burbot and eel that can accumulate MeHg, but other species can also exhibit elevated Hg concentrations depending on catch site, for example salmonids caught in Lakes Vänern and Vättern. The concentrations of Hg in fish in inland waters are generally markedly elevated as a result of emissions of Hg to the air and long-distance transport. Local emissions of Hg also affect the Hg concentrations in fish, as can be seen e.g. in northern parts of Lake Vänern. Coastal pike in areas without local influences have relatively low Hg concentrations.

Exposure

Current exposure

Around 2-3 % of the adult population report that they eat pike, perch, pike-perch or burbot 1-3 times per month. Further, 1-3 % state that they eat such fish once a week, while 1 % of men and older women eat such fish a few times per week. Others eat such fish never or only a few times per year.

Among children, 1-2.7 % and 0.1 % (4-12 year-olds) report that they eat pike, perch, pike-perch or burbot 2-3 times a month and once a week respectively, while only 0.1-0.4 % state that they eat such fish more than once a week. Others never eat such fish (56-66 %) or only a few times per year (1-4 times/yr, 26-30 %; 5-8 times/yr, 4-7 %; 9-12 times/yr, 3-6 %).

Calculations of MeHg intake indicate that mean intake for adults and children is approx. 0.1-0.2 µg/kg body weight and week. Lean sea fish that has low average Hg concentrations is the dominant source of MeHg exposure for most individuals. For those who eat pike the intake is considerably higher and for these individuals pike is a very large source of MeHg exposure, particularly if the catch site is such that the Hg concentration in fish is greatly elevated. If the Hg concentration in pike is at the maximum limit level (1.0 mg/kg), then the calculations imply that

intake is 0.8 µg/kg body weight and week for an adult individual. In children who eat large amounts of fish (95th percentile) MeHg exposure varied between approx. 0.4-0.6 µg/kg body weight and week.

The exposure studies carried out among pregnant women in Sweden point in the same direction as the theoretical intake calculations; exposure to MeHg is generally low. Studies directed at groups who eat large amounts of fish show a higher exposure to MeHg than in pregnant women, and with considerable inter-individual variation.

A survey of recreational fishing from 2005 indicates that 1-2 % of the population eat self-caught fish at least once a week. Perch and pike caught in small lakes and tarns represent the greatest catch out-take among recreational anglers.

Effects of MeHg

Based on observations from early environmental disasters and later studies on the effects in children exposed to prenatal MeHg exposure, it has been concluded that the critical effect of exposure to MeHg is the impact on central nervous system during the foetal stage. The effects that have been observed in some studies consist of delayed development of the central nervous system in the form of impaired cognitive ability. These effects are so small that they cannot be distinguished at an individual level.

Pregnant women are therefore a risk group in the population. Furthermore, the possibility cannot be ruled out that children up to teenage years can be more sensitive to effects than adults. At high exposures to MeHg in adults, effects are also observed on the nervous system but higher levels than in the foetus are required before these occur. Results from epidemiological studies indicate a correlation between high exposure to MeHg in adults and incidence of cardiovascular disease, while no such effects are observed at low exposure to MeHg. Animal models and *in vitro* systems indicate that MeHg has an immunotoxic effect, but the significance of this from a risk assessment perspective has still not been fully investigated.

Risk assessments

Tolerable intake

The FAO/WHO expert group JECFA specifies a provisional tolerable weekly intake (PTWI) of 1.6 µg/kg body weight. This is considered to protect the most sensitive group, i.e. pregnant women and their foetuses, from effects. This assessment is based on epidemiological studies on the Faroe Islands and the Seychelles. The PTWI applies in principle to all groups in the population, but for adults other than pregnant women JECFA considers a value twice the PTWI, i.e.

approx. 3.2 µg/kg body weight/week, to be a safe exposure level. The latest American risk assessment, which is based on the study on the Faroe Islands, specifies a reference dose of 0.1 µg/kg body weight/day, which is equivalent to 0.7 µg/kg body weight/week.

Lowest observed effect levels

Benchmark dose modelling based on the latest epidemiological studies on the relationship between prenatal exposure to MeHg and effects on development of the nervous system in children has resulted in concentrations that vary between 2.2-6.8 µg/g in maternal hair during pregnancy in the most conservative modelling of Faroe Island data. When a different dose-response curve was chosen the modelling of data from the Faroe Islands resulted in a BMDL of 12 µg/g in maternal hair during pregnancy. The BMDL based on the Seychelles study is approx. 25 µg/g in maternal hair during pregnancy. The FAO/WHO expert committee JECFA based their calculations on an average value of 14 µg/g in maternal hair during pregnancy based on the studies from the Faroe Islands and the Seychelles.

Risk characterisation

Exceeding tolerable intake and effect levels

Based on exposure studies, a small proportion (0-4 %) of pregnant women in Sweden can be assumed to exceed the American reference dose (0.7 µg/kg body weight/week), while none appear to exceed the JECFA PTWI (1.6 µg/kg body weight/week). In studies of exposure in groups who eat large amounts of fish, the limit is exceeded more frequently than in pregnant women, but there are no supporting data to quantify the size of this group. Theoretical calculations of intake in adults indicate that the JECFA PTWI value can also be exceeded by pregnant women with high consumption of fish with elevated concentrations of MeHg.

The margin between the exposure levels given as BMDL based on the Faroe Islands study and the average exposure in pregnant women in Sweden is 5- to 36-fold if the most conservative BMDLs are used, or 27- to 63-fold based on another assumption regarding the dose-response model. Using the JECFA assessment instead, the margin is 32- to 73-fold between the average exposure in pregnant women in Sweden and NOEL/BMDL.

The limited data available on exposure to MeHg in women in Sweden who eat large amounts of fish indicate that there can be groups that lie close to or within the vicinity of the most conservative BMDLs.

Exposure studies directed at men and older women who are high consumers of fish show that exposure generally lies below a value twice the PTWI, but that it is

exceeded in some cases. The supporting data are very limited but the possibility cannot be excluded that in the Swedish population there are groups with high consumption of fish with elevated Hg concentrations who have such a high MeHg intake that the risk of MeHg-induced cardiovascular disease can be elevated. It is currently not possible to quantify the size of this group, but it is probably limited to those who very often eat self-caught fish with elevated MeHg concentrations.

Trends

A number of new fish species have been introduced onto the Swedish market in recent years. In certain cases the Hg concentrations in such fish may be unclear. Certain marine fish species in particular, e.g. swordfish and tuna (not canned tuna) which as a rule have higher Hg concentrations than other marine fish species have become increasingly common in supermarkets. What this means for MeHg exposure in the population is not known. However, the temporal trend study carried out in Västerbotten (1990-1999) indicates that exposure is generally decreasing.

For nutritional reasons, the National Food Administration recommends eating fish 2-3 times a week, of which one portion is fatty fish. With the current dietary habits, this means that it is desirable for fish consumption to generally increase. Such a general increase in fish consumption will also mean an increase in the average intake of MeHg. The level is currently so low that there is considered not to be a problem with such an increase in fish consumption. However, it is desirable that those who already eat large amounts of fish do not increase their consumption further, since the safety margins decrease.

Potential for exceeding tolerable intake

The mercury concentration in fish varies greatly, and in fish caught in certain lakes it is considerably higher than in fish caught in other lakes. Fish that is sold commercially is probably caught in waters with known low Hg concentrations. However the conclusion that can be drawn from available environmental monitoring data (which are not associated with commercial fish but are only intended to reflect the environmental situation) is that it is common for the Hg concentration in pike to exceed the maximum level for commercial pike. The mercury concentration in perch in such studies has also been above the maximum level for sale. This is therefore primarily of importance for those who catch their own consumption fish, which is in the order of 1-2 % of the population. It is therefore important that information on the local mercury situation is available and that it is known to consumers who catch their own fish.

If consumption habits were to change so that consumers more frequently selected fish species with elevated Hg concentrations, the fraction of the population that

exceeded tolerable intake would increase. The safety margins to the levels at which effects could be expected would therefore also decrease.

Need for exposure-limiting dietary advice

Overall, it is estimated that exposure to MeHg in the absolute majority of the Swedish population is at a level that can currently be assumed to be safe with regard to the tolerable intake based on the JECFA and NRC assessments. This applies to all age groups in the population. Tolerable intake or possible effect levels are observed to be exceeded in individuals who regularly eat fish with elevated concentrations rather than other fish, and in individuals who eat all types of fish very often.

For the great majority of the Swedish population, with current consumption habits, there is therefore no need for restrictive dietary advice to reduce exposure to MeHg. A few percent of the population report that they regularly eat pike, perch, pike-perch and burbot, i.e. species that can have elevated concentrations. It is mainly in this group that restriction of exposure may be necessary. Very biased consumption of fish, even with low Hg-concentrations, can also result in tolerable intakes being exceeded. Information on consumption of certain marine predatory fish with high Hg concentrations is limited, but these species should also be included in dietary advice. However, a change should perhaps be considered in the dietary advice on eel. Consumption of eel is extremely limited and the National Food Administration's comprehensive investigations have shown that the Hg concentrations in eel are generally lower than in pike, perch, pike-perch and burbot. However, there are exceptions; eel from certain waters may have higher concentrations.

Dietary advice has probably resulted in decreased fish consumption generally in pregnant women. It is still important that this group in the population is informed of the importance of limiting consumption of certain fish species but continues to consume fish with low Hg concentrations.

The way in which dietary advice should be formulated should be based on the risk-benefit assessment currently being carried out at the National Food Administration, although dietary advice should be targeted more clearly than at present at those who eat certain fish species often. However, it should be pointed out that the dietary advice provided to date was based on a lack of scientific data to establish a safe exposure level to protect the foetus from the effects of MeHg. Current dietary advice to pregnant and breastfeeding women and women planning a pregnancy is therefore that they should abstain completely from consumption of pike, pike-perch, burbot, perch, eel and some marine predatory fish; giant halibut, fresh/frozen tuna, swordfish, shark and ray. With the new epidemiological studies

available, it has been possible to establish a tolerable intake that also protects the foetus, according to the JECFA assessment. It may eventually be possible to establish a safe consumption frequency for the fish species for which the National Food Administration currently recommends full abstinence. The experience of the National Food Administration from contacts with pregnant women and antenatal nurses is that many pregnant women are greatly concerned about having eaten occasional portions of the species listed before they knew they were pregnant and were given the information on dietary advice in the antenatal clinic. It would be an advantage for many pregnant women if such unnecessary anxiety could be avoided.

Dietary advice to date has been specifically targeted at pregnant and breastfeeding women and women planning a pregnancy, in addition to the general public. The half-life of MeHg in the body is a few months. This means that high MeHg exposure in the mother in the period immediately before and in the first few months of a pregnancy can lead to the foetus being exposed to undesirable amounts of MeHg. The current structure of the advice presumes that women of childbearing age plan their pregnancies and also that they adopt the targeted advice before their first visit to the antenatal clinic. Even though the advice *per se* is based on scientific grounds, targeting the advice at women of childbearing age for information reasons should be considered.

Dietary advice to decrease exposure to MeHg has previously not been directed at children. However, based on the intake calculations made recently by the National Food Administration, exposure to MeHg in children in Sweden is at a safe level according to JECFA and NRC. The National Food Administration's current dietary advice on consuming perch, pike, pike-perch and burbot at most once a week should remain.

Overall, there is a need for exposure-limiting dietary advice that is clearly targeted at those who eat large amounts of fish of certain fish species (in particular pike, pike-perch, burbot and perch and certain marine predatory fish). It is particularly important that information on such dietary advice reaches pregnant and breastfeeding women.

It is also important that those who catch their own fish have access to information on the mercury concentrations in fish in the waters in which they fish, and that consumption of self-caught fish that exceeds the maximum limit is restricted.

References

1. Westöö, G. and M. Rydälv, *Kvicksilver och metylkvicksilver i fisk och kräftor*. Vår Föda, 1969. **3**.
2. Lasorsa, B. and S. Allen-Gil, *The methylmercury to total mercury ratio in selected marine, freshwater and terrestrial organisms*. Water Air Soil Pollut, 1995. **80**: p. 905-13.
3. Storelli, M.M., et al., *Total mercury and methylmercury content in edible fish from the Mediterranean Sea*. J Food Prot, 2003. **66**(2): p. 300-3.
4. Andersson, T., et al., *Kvicksilver i svenska sjöar*. Naturvårdsverket, 1987. **Rapport 3291**.
5. Meili, M., P. Kärrhage, and H. Borg, *Kvicksilver i fisk och födodjur i 10 skånska sjöar 2002*. Rapport 2004:19. Länsstyrelsen i Skåne län.
6. Lindeström, L. and C. Grotell, *Metaller och stabila organiska ämnen i Vänernfisk 1996/97*. Vänerns vattenvårdsförbund. Rapport 5., 1998.
7. Sundström, B., et al., *Mercury in fish, mainly from the Baltic Sea and Swedish waters*. Poster. Presenterad vid Second international IUPAC symposium. Trace Elements in Food. Bryssel, 7-8 Oktober 2004.
8. Lindeström, L., *Mercury in sediment and fish communities of Lake Vänern, Sweden; recovery from contamination*. Ambio, 2001. **30**: p. 538-544.
9. Greyerz, E., et al., *Kvicksilver i gäddor från Norrlandskusten*. Naturvårdsverket, Livsmedelsverket, Länsstyrelserna i Gävleborg, Norrbottens, Västerbottens och Västernorrlands län. Rapport tryckt av Länsstyrelsen i Luleå. 2000.
10. Ohlin, B., *Kvicksilverhalter i fisk i allmänna handeln [In Swedish]*. Vår Föda, 1993. **8-9**: p. 390-397.
11. Forsyth, D.S., et al., *Methylmercury levels in predatory fish species marketed in Canada*. Food Addit Contam, 2004. **21**(9): p. 849-56.
12. Bignert, A., *Comments concerning the national Swedish monitoring programme in fresh water biota 2001*. Rapport från Naturhistoriska Riksmuseet. 2002.
13. Ankarberg, E. and K. Petersson Grawé, *Intagsberäkning för dioxiner (PCDD/PCDF), dioxinlika PCBer och metylkvicksilver via livsmedel*. Livsmedelsverket, Uppsala. Rapport 25-2005, 2005.
14. Lindeström, L., *Mälarfisk. Innehåll av metaller och stabila organiska ämnen 2001*. ÅF 01/35:2. Mälarens vattenvårdsförbund. 2001.
15. SNT. Statens näringsmiddelstilsyn, N., *Kartlegging av tungmetaller og klororganiske miljøgifter i marin fisk fanget i Sør-Norge*. Rapport 4/1999.
16. Gutenmann, W.H., Lisk, D. J., *Higher average mercury concentration in fish filets after skinning and fat removal*. J Food Safety, 1991. **11**: p. 99-103.
17. Morgan, J.N., Berry, M. R., Graves, R. L., *Effects of commonly used cooking practices on total mercury concentration in fish and their impact on exposure assessments*. J Expo Anal Environ Epidemiol, 1997. **7**: p. 119-33.
18. Becker W, P.M., *Riksmaten 1997-98. Befolkningens kostvanor och näringsintag. Metod- och resultatanalys*. Livsmedelsverket, Uppsala. 2002.
19. Rödström, A., et al., *Hg i hår och blod hos gravida kvinnor i Västsverige*. Sakrapport till Naturvårdsverket, 2004. 2004.
20. Gerhardsson, L., T. Lundh, and H. Welinder, *Metallmätningar hos gravida kvinnor*. Rapport till miljöövervakningsenheten, Naturvårdsverket, kontrakt nr 2150204. 2005. 2005.
21. Bergdahl I, S., M. Lundh T., *Metallmätningar hos gravida kvinnor i Västerbotten*. Rapport till Miljöövervakningsenheten, Kontrakt nr 215 0305, Naturvårdsverket. 2006.
22. Glynn, A., et al., *Studie av förstföderskor. Organiska miljögifter hos gravida och ammande - Del 1 Serumnivåer*. SLV Rapport, 2006. **4**: p. 1-62.
23. Helmfrid, I., et al., *Miljögifter i blod hos högkonsumenter av Vätternfisk*. Rapport 74. Vätternvårdsförbundet. 2003.
24. Björnberg Ask, K. and M. Berglund, *Studie av storfiskkonsumenter, del I. Delrapport till Naturvårdsverket. Överenskommelse Nr 215 0510*. 2006.

25. Johnsson, C., et al., *Hair mercury levels versus freshwater fish consumption in household members of Swedish angling societies*. Environ Res, 2004. **96**(3): p. 257-63.
26. Johnsson, C., A. Schutz, and G. Sallsten, *Impact of consumption of freshwater fish on mercury levels in hair, blood, urine, and alveolar air*. J Toxicol Environ Health A, 2005. **68**(2): p. 129-40.
27. Rylander, L., U. Stromberg, and L. Hagmar, *Agreement between reported fish consumption obtained by two interviews and its impact on the results in a reproduction study*. Eur J Epidemiol, 1998. **14**(1): p. 93-7.
28. Svensson, B.G., et al., *Fish consumption and exposure to persistent organochlorine compounds, mercury, selenium and methylamines among Swedish fishermen*. Scand J Work Environ Health, 1995. **21**(2): p. 96-105.
29. Fiskeriverket, *Fiske 2005 en undersökning om svenskarnas fritidsfiske. Fiskeriverket i samarbete med SCB*. 2005.
30. Enghardt Barbieri, H., M. Pearson, and W. Becker, *Riksmaten - barn 2003. Livsmedels- och näringsintag bland barn i Sverige. Livsmedelsverket, Uppsala*. 2006.
31. Concha, G., et al., *Svensk intagsberäkning av dioxiner (PCDD/PCDF), dioxinlika PCBer och metylkvicksilver för barn baserad på aktuella analysdata samt kostundersökningen 2003. Resultatrapport, november 2006*. 2006.
32. Barany, E., et al., *Mercury and selenium in whole blood and serum in relation to fish consumption and amalgam fillings in adolescents*. J Trace Elem Med Biol, 2003. **17**(3): p. 165-70.
33. Berglund, M., et al., *Inter-individual variations of human mercury exposure biomarkers: a cross-sectional assessment*. Environ Health, 2005. **4**: p. 20.
34. Stern, A.H., *Estimation of the interindividual variability in the one-compartment pharmacokinetic model for methylmercury: implications for the derivation of a reference dose*. Regul Toxicol Pharmacol, 1997. **25**(3): p. 277-88.
35. Hallgren, C.G., et al., *Markers of high fish intake are associated with decreased risk of a first myocardial infarction*. Br J Nutr, 2001. **86**(3): p. 397-404.
36. Lindberg, A., et al., *Exposure to methylmercury in non-fish-eating people in Sweden*. Environ Res, 2004. **96**(1): p. 28-33.
37. Vahter, M., et al., *Longitudinal study of methylmercury and inorganic mercury in blood and urine of pregnant and lactating women, as well as in umbilical cord blood*. Environ Res, 2000. **84**(2): p. 186-94.
38. Bjornberg, K.A., et al., *Methyl mercury and inorganic mercury in Swedish pregnant women and in cord blood: influence of fish consumption*. Environ Health Perspect, 2003. **111**(4): p. 637-41.
39. Oskarsson, A., et al., *Mercury levels in the hair of pregnant women in a polluted area in Sweden*. Sci Total Environ, 1994. **151**(1): p. 29-35.
40. Ask Björnberg, K., et al., *Methyl mercury exposure in Swedish women with high fish consumption*. Science of The Total Environment, 2005. **341**(1-3): p. 45-52.
41. Bjornberg, K.A., et al., *Methyl mercury exposure in Swedish women with high fish consumption*. Sci Total Environ, 2005. **341**(1-3): p. 45-52.
42. Oskarsson, A., et al., *Mercury Levels in Hair from People Eating Large Quantities of Swedish Fresh-Water Fish*. Fd Add Contam, 1990. **7**: p. 555-562.
43. Wennberg, M., et al., *Time trends in burdens of cadmium, lead, and mercury in the population of northern Sweden*. Environ Res, 2006. **100**(3): p. 330-8.
44. NRC., *Toxicological effects of methylmercury*, N.A. Press, Editor. 2000, National Research Council: Washington DC. p. 344.
45. WHO, *Methylmercury. Fifty-third meeting of the Joint FAO/WHO Expert Committee on Food Additives and Contaminants. Safety evaluation of certain Food Additives and Contaminants*. Food Additives Series 44. World Health Organization, Geneva, 2000: p. 313-391.
46. WHO, *Methylmercury. Sixty-first meeting of Joint FAO/WHO Expert Committee on Food Additives and Contaminants. Safety evaluation of certain Food Additives and Contaminants. Food Additives Series, 52*. World Health Organization, Geneva. 2004.
47. WHO, *Methylmercury. Joint FAO/WHO Expert Committee on Food Additives and Contaminants. Sixty-seventh meeting. Summary and conclusions. Safety evaluation of certain Food Additives and Contaminants*. JECFA/67/SC. World Health Organization, Geneva., 2006.
48. Harada, M., *Minamata disease: methylmercury poisoning in Japan caused by environmental pollution*. Crit Rev Toxicol, 1995. **25**(1): p. 1-24.
49. Bakir, F., et al., *Methylmercury poisoning in Iraq*. Science, 1973. **181**(96): p. 230-41.

50. Steuerwald, U., et al., *Maternal seafood diet, methyl mercury exposure, and neonatal neurologic function*. J Pediatr, 2000. **136**: p. 599-605.
51. Grandjean, P., *Mercurial uncertainties in environmental health*. Ann N Y Acad Sci, 1997. **837**: p. 239-45.
52. Cordier, S., et al., *Neurodevelopmental investigations among methylmercury-exposed children in French Guiana*. Environ Res, 2002. **89**(1): p. 1-11.
53. Marsh, D.O., et al., *The Seychelles study of fetal methylmercury exposure and child development: introduction*. Neurotoxicology, 1995. **16**(4): p. 583-96.
54. Myers, G.J., et al., *Neurodevelopmental outcomes of Seychellois children sixty-six months after in utero exposure to methylmercury from a maternal fish diet: pilot study*. Neurotoxicology, 1995. **16**(4): p. 639-52.
55. Myers, G.J., et al., *Summary of the Seychelles child development study on the relationship of fetal methylmercury exposure to neurodevelopment*. Neurotoxicology, 1995. **16**(4): p. 711-16.
56. Murata, K., et al., *Evoked potentials in Faroese children prenatally exposed to methylmercury*. Neurotoxicol Teratol, 1999. **21**(4): p. 471-2.
57. Murata, K., et al., *Delayed brainstem auditory evoked potential latencies in 14-year-old children exposed to methylmercury*. J Pediatr, 2004. **144**(2): p. 177-83.
58. Counter, S.A., et al., *Blood mercury and auditory neuro-sensory responses in children and adults in the Nambija gold mining area of Ecuador*. Neurotoxicology, 1998. **19**(2): p. 185-96.
59. Grandjean, P., P. Weihe, and R.F. White, *Milestone development in infants exposed to methylmercury from human milk*. Neurotoxicology, 1995. **16**(1): p. 27-33.
60. Myers, G.J., et al., *Effects of prenatal methylmercury exposure from a high fish diet on developmental milestones in the Seychelles Child Development Study*. Neurotoxicology, 1997. **18**(3): p. 819-29.
61. Axtell, C.D., et al., *Semiparametric modeling of age at achieving developmental milestones after prenatal exposure to methylmercury in the Seychelles child development study*. Environ Health Perspect, 1998. **106**(9): p. 559-63.
62. Marsh, D.O., et al., *Fetal methylmercury study in a Peruvian fish-eating population*. Neurotoxicology, 1995. **16**(4): p. 717-26.
63. Kjellström, T., et al., *Physical and Mental Development of Children with Prenatal Exposure to Mercury from Fish. Stage 1. Preliminary tests at age 4. Solna, National Swedish Environmental Board. Rapport 3080*. 1986.
64. Kjellström, T., et al., *Physical and Mental Development of Children with Prenatal Exposure to Mercury from Fish. Stage 2. Interviews and psychological tests at age 6. Solna, National Swedish Environmental Board. Rapport 3642*. 1989.
65. Crump, K.S., et al., *Influence of prenatal mercury exposure upon scholastic and psychological test performance: benchmark analysis of a New Zealand cohort*. Risk Anal, 1998. **18**(6): p. 701-13.
66. Grandjean, P., et al., *Impact of maternal seafood diet on fetal exposure to mercury, selenium, and lead*. Arch Environ Health, 1992. **47**(3): p. 185-95.
67. Weihe, P., et al., *Health implications for Faroe islanders of heavy metals and PCBs from pilot whales*. Sci Total Environ, 1996. **186**(1-2): p. 141-8.
68. Budtz-Jorgensen, E., et al., *Methylmercury neurotoxicity independent of PCB exposure*. Environ Health Perspect, 1999. **107**(5): p. A236-7.
69. Grandjean, P., et al., *Neurobehavioral deficits associated with PCB in 7-year-old children prenatally exposed to seafood neurotoxins*. Neurotoxicol Teratol, 2001. **23**(4): p. 305-17.
70. Stewart, P.W., et al., *Cognitive development in preschool children prenatally exposed to PCBs and MeHg*. Neurotoxicol Teratol, 2003. **25**(1): p. 11-22.
71. Myers, G.J., et al., *Main neurodevelopmental study of Seychellois children following in utero exposure to methylmercury from a maternal fish diet: outcome at six months*. Neurotoxicology, 1995. **16**(4): p. 653-64.
72. Myers, G.J., et al., *A pilot neurodevelopmental study of Seychellois children following in utero exposure to methylmercury from a maternal fish diet*. Neurotoxicology, 1995. **16**(4): p. 629-38.
73. Davidson, P.W., et al., *Neurodevelopmental outcomes of Seychellois children from the pilot cohort at 108 months following prenatal exposure to methylmercury from a maternal fish diet*. Environ Res, 2000. **84**(1): p. 1-11.

74. Davidson, P.W., et al., *Longitudinal neurodevelopmental study of Seychellois children following in utero exposure to methylmercury from maternal fish ingestion: outcomes at 19 and 29 months*. Neurotoxicology, 1995. **16**(4): p. 677-88.
75. Davidson, P.W., et al., *Effects of prenatal and postnatal methylmercury exposure from fish consumption on neurodevelopment: outcomes at 66 months of age in the Seychelles Child Development Study*. Jama, 1998. **280**(8): p. 701-7.
76. Myers, G.J., et al., *Prenatal methylmercury exposure from ocean fish consumption in the Seychelles child development study*. Lancet, 2003. **361**(9370): p. 1686-92.
77. Davidson, P.W., et al., *Methylmercury and neurodevelopment: Longitudinal analysis of the Seychelles child development cohort*. Neurotoxicol Teratol, 2006. **28**(5): p. 529-35.
78. Salonen, J.T., et al., *Intake of mercury from fish, lipid peroxidation, and the risk of myocardial infarction and coronary, cardiovascular, and any death in eastern Finnish men*. Circulation, 1995. **91**(3): p. 645-55.
79. Rissanen, T., et al., *Fish oil-derived fatty acids, docosahexaenoic acid and docosapentaenoic acid, and the risk of acute coronary events: the Kuopio ischaemic heart disease risk factor study*. Circulation, 2000. **102**(22): p. 2677-9.
80. Virtanen, J.K., et al., *Mercury, fish oils, and risk of acute coronary events and cardiovascular disease, coronary heart disease, and all-cause mortality in men in eastern Finland*. Arterioscler Thromb Vasc Biol, 2005. **25**(1): p. 228-33.
81. Guallar, E., et al., *Mercury, fish oils, and the risk of myocardial infarction*. N Engl J Med, 2002. **347**(22): p. 1747-54.
82. Salonen, J.T., et al., *Mercury accumulation and accelerated progression of carotid atherosclerosis: a population-based prospective 4-year follow-up study in men in eastern Finland*. Atherosclerosis, 2000. **148**(2): p. 265-73.
83. Sorensen, N., et al., *Prenatal methylmercury exposure as a cardiovascular risk factor at seven years of age*. Epidemiology, 1999. **10**(4): p. 370-5.
84. Grandjean, P., et al., *Cardiac autonomic activity in methylmercury neurotoxicity: 14-year follow-up of a Faroese birth cohort*. J Pediatr, 2004. **144**(2): p. 169-76.
85. Bilrha, H., et al., *In vitro activation of cord blood mononuclear cells and cytokine production in a remote coastal population exposed to organochlorines and methyl mercury*. Environ Health Perspect, 2003. **111**(16): p. 1952-7.
86. Belles-Isles, M., et al., *Cord blood lymphocyte functions in newborns from a remote maritime population exposed to organochlorines and methylmercury*. J Toxicol Environ Health A, 2002. **65**(2): p. 165-82.
87. Ilback, N.G., J. Sundberg, and A. Oskarsson, *Methyl mercury exposure via placenta and milk impairs natural killer (NK) cell function in newborn rats*. Toxicol Lett, 1991. **58**(2): p. 149-58.
88. Ilback, N.G., et al., *Effects of methyl mercury on cytokines, inflammation and virus clearance in a common infection (coxsackie B3 myocarditis)*. Toxicol Lett, 1996. **89**(1): p. 19-28.
89. Ortega, H.G., et al., *Lymphocyte proliferative response and tissue distribution of methylmercury sulfide and chloride in exposed rats*. J Toxicol Environ Health, 1997. **50**(6): p. 605-16.
90. Ortega, H.G., et al., *Neuroimmunological effects of exposure to methylmercury forms in the Sprague-Dawley rats. Activation of the hypothalamic-pituitary-adrenal axis and lymphocyte responsiveness*. Toxicol Ind Health, 1997. **13**(1): p. 57-66.
91. Thompson, S.A., et al., *Alterations in immune parameters associated with low level methylmercury exposure in mice*. Immunopharmacol Immunotoxicol, 1998. **20**(2): p. 299-314.
92. King, M.D., et al., *Neurotoxicity and immunotoxicity assessment in CBA/J mice with chronic Toxoplasma gondii infection and multiple oral exposures to methylmercury*. J Parasitol, 2003. **89**(4): p. 856-9.
93. Haggqvist, B., et al., *The immunosuppressive effect of methylmercury does not preclude development of autoimmunity in genetically susceptible mice*. Toxicology, 2005. **208**(1): p. 149-64.
94. Thuvander, A., J. Sundberg, and A. Oskarsson, *Immunomodulating effects after perinatal exposure to methylmercury in mice*. Toxicology, 1996. **114**(2): p. 163-75.
95. Wild, L.G., et al., *Immune system alteration in the rat after indirect exposure to methyl mercury chloride or methyl mercury sulfide*. Environ Res, 1997. **74**(1): p. 34-42.
96. Ilback, N.G., et al., *Trace element distribution in heart tissue sections studied by nuclear microscopy is changed in Coxsackie virus B3 myocarditis in methyl mercury-exposed mice*. Biol Trace Elem Res, 2000. **78**(1-3): p. 131-47.

97. Hultman, P. and H. Hansson-Georgiadis, *Methyl mercury-induced autoimmunity in mice*. Toxicol Appl Pharmacol, 1999. **154**(3): p. 203-11.
98. Budtz-Jorgensen, E., et al., *Benchmark dose calculations of methylmercury-associated neurobehavioural deficits*. Toxicol Lett, 2000. **112-113**: p. 193-9.
99. Budtz-Jorgensen, E., N. Keiding, and P. Grandjean, *Benchmark dose calculation from epidemiological data*. Biometrics, 2001. **57**(3): p. 698-706.
100. Crump, K.S., et al., *Benchmark concentrations for methylmercury obtained from the Seychelles Child Development Study*. Environ Health Perspect, 2000. **108**(3): p. 257-63.
101. Grandjean, P., et al., *Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury*. Neurotoxicol Teratol, 1997. **19**(6): p. 417-28.
102. Grandjean, P., et al., *Umbilical cord mercury concentration as biomarker of prenatal exposure to methylmercury*. Environ Health Perspect, 2005. **113**(7): p. 905-8.
103. Salonen, J.T., K. Nyyssonen, and R. Salonen, *Fish intake and the risk of coronary disease*. N Engl J Med, 1995. **333**(14): p. 937; author reply 938.

Appendix

Appendix

County	Municipality	Catch year Sampling date	Fish species	Number	Age (yr)	Length (cm)	Weight (g)	Hg mg/kg wet wt					Minimum limit exceeded by, %
								Mean	SD	Median	Min	Max	
Blekinge	Karlskrona	1991	Pike	57		48 (39-66)	1101 (560-2990)	0.9	0.4	0.8	0.1	2.2	35
Dalarna	Malung	1993	Pike	6			1152 (860-1590)	0.8	0.4	0.7	0.6	1.7	17
Dalarna	Malung	1994	Pike	5		55 (51-60)	1120 (950-1300)	1.0	0.1	1.0	0.9	1.1	40
Dalarna	Avesta	1990	Pike	18		53 (43-70)	997 (520-1900)	0.4	0.2	0.4	0.0	0.8	0
Dalarna	Falu	1990	Pike	22		47 (40-61)	893 (460-1900)	0.6	0.3	0.5	0.2	1.2	9.1
Dalarna	Hedemora	1990	Pike	23		49 (42-56)	995 (580-1330)	0.6	0.4	0.6	0.1	1.3	13
Dalarna	Rättvik	1990	Pike	5			980 (800-1350)	0.8	0.3	0.8	0.4	1.1	20
Dalarna	Gagnef, Falu, Avesta	1991	Pike	32			898 (350-1800)	0.4	0.2	0.4	0.1	0.8	0
Dalarna	Hedemora	1991	Pike	15			724 (260-2120)	0.5	0.3	0.4	0.2	1.2	6.7
Dalarna	Ludvika	1991	Pike	11			1067 (740-1450)	1.0	0.2	1.0	0.7	1.2	45
Dalarna	Malung	1991	Pike	20			953 (450-1450)	0.6	0.4	0.5	0.1	1.5	15
Dalarna	Avesta	1992	Pike	3			1417 (1000-2050)	1.0	0.2	1.1	0.8	1.2	67
Dalarna	Falu	1992	Pike	36			851 (450-1540)	0.5	0.2	0.5	0.2	1.2	2.8
Dalarna	Ludvika	1992	Pike	5			1258 (790-1670)	0.5	0.1	0.6	0.4	0.7	0
Dalarna	Rättvik	1992	Pike	14		62 (54-92)	1443 (1100-2800)	0.8	0.4	0.6	0.3	1.7	36
Dalarna	Avesta	1993	Pike	1		56	930	0.9					
Dalarna	Falu, Vansbro	1993	Pike	51		52 (34-68)	927 (240-1760)	0.5	0.2	0.5	0.2	1.0	0
Dalarna	Gagnef	1993	Pike	5		47 (40-64)	715 (350-1660)	0.8	0.2	0.7	0.7	1.2	20
Dalarna	Malung	1993	Pike	28		50 (41-66)	932 (330-1740)	0.7	0.3	0.6	0.3	1.7	7.1
Dalarna	Avesta	1994	Pike	8			1103 (360-1600)	0.3	0.2	0.3	0.1	0.5	0
Dalarna	Falu	1994	Pike	18		51 (38-71)	1213 (380-2060)	0.6	0.3	0.5	0.3	1.4	5.6
Dalarna	Ludvika	1994	Pike	4		53 (50-56)	938 (680-1130)	0.8	0.2	0.8	0.6	1.1	25
Dalarna	Malung	1994	Pike	20		54 (47-60)	1018 (680-1320)	0.7	0.2	0.8	0.3	1.1	10
Dalarna	Rättvik	1994	Pike	9		62 (55-68)	1378 (1000-1900)	1.0	0.2	1.0	0.7	1.3	44

Appendix

County	Municipality	Catch year Sampling date	Fish species	Number	Age (yr)	Length (cm)	Weight (g)	Hg mg/kg wet wt					Minimum limit exceeded by, %
								Mean	SD	Median	Min	Max	
Dalarna	Säter	1994	Pike	30		48 (36-66)	1256 (600-3000)	0.4	0.2	0.4	0.1	1.1	3.3
Dalarna	Falu	1995	Pike	5		47 (43-53)	694 (500-1030)	0.6	0.2	0.5	0.4	0.9	0
Dalarna	Gagnef	1995	Pike	6		51 (43-55)	877 (455-1130)	0.7	0.2	0.7	0.5	1.1	17
Dalarna	Ludvika	1995	Pike	10		52 (47-60)	825 (560-1270)	0.6	0.2	0.5	0.3	1.1	10
Dalarna	Malung	1995	Pike	17		53 (43-64)	825 (300-1300)	0.6	0.3	0.5	0.3	1.1	12
Dalarna	Avesta, Leksand	1996	Pike	33		52 (40-75)	1004 (385-2750)	0.6	0.2	0.6	0.2	1.0	0
Dalarna	Falu	1996	Pike	11		55 (42-67)	1133 (480-2080)	0.7	0.2	0.7	0.5	1.1	9.1
Dalarna	Malung	1996	Pike	15		57 (49-64)	990 (205-1480)	0.7	0.3	0.7	0.3	1.2	6.7
Dalarna	Avesta	1997	Pike	9		51 (38-60)	1096 (335-1800)	0.4	0.3	0.3	0.2	1.2	11
Dalarna	Falu, Älvdalen	1997	Pike	46		51 (35-71)	912 (273-2580)	0.3	0.2	0.3	0.1	0.8	0
Dalarna	Malung	1997	Pike	15		55 (48-62)	1130 (751-1500)	0.7	0.2	0.64	0.44	1.2	20
Dalarna	Falu, Malung	1998	Pike	20			754 (0,44-1510)	0.5	0.2	0.4	0.2	1.0	0
Dalarna	Ludvika (Marnästjärnen)	1998	Pike	5				2.2	0.7	1.9	1.5	3.1	100
Dalarna	Älvdalen	1998	Pike	32	5,6 (3-9)	53 (42-73)	944 (468-2172)	0.4	0.3	0.3	0.2	1.2	3.1
Dalarna	Hedemora, Ludvika, Falu, Malung, Älvdalen	1999	Pike	57		53 (37-85)	1127 (344-3920)	0.4	0.2	0.4	0.1	1.0	0
Dalarna	Avesta, Falu	2000	Pike	14			1601 (691-5600)	0.6	0.2	0.6	0.3	1.0	0
Dalarna	Avesta, Ludvika	2001	Pike	15		54 (38-67)	1135 (320-1860)	0.5	0.2	0.4	0.2	0.9	0
Dalarna	Falu	2001	Pike	5	4 (4-4)	52 (50-55)	938 (774-1105)	0.7	0.2	0.7	0.5	1.1	20
Dalarna	Malung	2001	Pike	30		52 (42-63)	983 (520-1640)	0.5	0.2	0.5	0.2	1.1	6.7
Dalarna	Malung	2002	Pike	15		53 (45-65)	996 (509-1850)	0.6	0.2	0.6	0.4	1.1	6.7
Dalarna	Ludvika	2002	Pike	5			1002 (800-1240)	0.3	0.0	0.3	0.2	0.3	0
Dalarna	Falu (Grycken)	2004	Pike	5		3,4 (3-4)	1,2 (0,73-1,4)	1.4	0.3	1.3	1.0	1.7	80
Dalarna	Malung	2004	Pike	10		52 (44-60)	1,0 (0,47-1,6)	0.7	0.2	0.6	0.5	1.1	10
Dalarna	Avesta	1994	Pike-perch	2		32, 40	360, 920	0.1, 0.1					0

Appendix

County	Municipality	Catch year Sampling date	Fish species	Number	Age (yr)	Length (cm)	Weight (g)	Hg mg/kg wet wt					Minimum limit exceeded by, %
								Mean	SD	Median	Min	Max	
Gävleborg	Gävle	1990	Pike	5				0.7	0.2	0.7	0.5	1.0	0
Gävleborg	Sandviken	1990	Pike	5		51 (44-69)	1550 (960-3210)	1.0	0.2	1.1	0.7	1.2	60
Gävleborg	Nordanstig, Hudiksvall, Söderhamn, Gävle	1999	Pike	62	5.4 (4-10)	54 (43-68)	1027 (510-2240)	0.2	0.1	0.2	0.1	0.6	0
Gävleborg	Hudiksvall (Källsjön)	2003	Pike	20	6.9 (4-12)	57 (42-71)	968 (195-1820)	1.4	0.7	1.1	0.5	3.2	55
Gävleborg	Ljusdal	2003	Pike	20	7.2 (4-9)	54 (41-68)	934 (375-1710)	0.9	0.3	0.8	0.4	1.6	30
Gävleborg	Gävle	2005	Pike	10		47 (43-52)	1083 (715-1500)	0.6	0.2	0.6	0.2	0.8	0
Gävleborg	Bollnäs	2005	Pike	17		53 (39-62)	1032 (360-1710)	0.8	0.5	0.6	0.3	1.5	35
Gävleborg	Hofors	2005	Pike	18		47 (38-54)	664 (312-1194)	0.5	0.3	0.5	0.1	1.1	5.6
Gävleborg	Ockelbo	2005	Pike	16		52 (41-58)	816 (423-1023)	0.9	0.2	0.9	0.7	1.3	25
Gävleborg	NorrHälsinge	2005	Pike	14		54 (44-63)	1260 (696-1713)	1.1	0.6	0.9	0.3	2.4	36
Gävleborg	Ovanåker, Bollnäs, Sandviken	2006	Pike	30		50 (34-73)	947 (280-2570)	0.5	0.2	0.5	0.1	0.9	0
Gävleborg	Ljusdal	2006	Pike	11		53 (42-61)	1139 (445-1830)	0.5	0.3	0.4	0.3	1.1	9.1
Gävleborg	Söderhamn	2005	Pike	16		50 (43-58)	974 (490-1718)	1.1	0.3	1.0	0.6	1.5	50
Gävleborg	Söderhamn	2006	Pike	29		46 (35-60)	719 (292-1282)	0.5	0.3	0.4	0.2	1.6	3.4
Halland	Falkenberg	1990	Pike	10		47 (42-53)	1007 (740-1360)	0.7	0.3	0.7	0.2	1.1	10
Halland	Hylte	1990	Pike	9		54 (44-78)	1574 (880-4120)	1.2	0.3	1.2	0.8	1.7	89
Halland	Falkenberg, Hylte	1991, 1993, 1995-2000, 2002, 2003	Pike	80		55 (46-68)	1128 (576-2274)	0.4	0.1	0.4	0.2	0.8	0
Jönköping	Aneby	1990, 1994	Pike	28			1070 (370-2600)	0.4	0.2	0.4	0.1	0.8	0
Jönköping	Eksjö	1990	Pike	25		50 (42-58)	1134 (760-1650)	0.8	0.3	0.7	0.3	1.4	16
Jönköping	Eksjö	1991	Pike	19			883 (530-1200)	0.6	0.4	0.5	0.2	1.6	26
Jönköping	Eksjö	1993	Pike	107			50 (25-64)	0.5	0.3	0.4	0.1	1.5	6.5
Jönköping	Eksjö	1994	Pike	54	6 (4-11)	53 (39-75)	1000 (380-2225)	0.6	0.4	0.5	0.2	2.7	13

Appendix

County	Municipality	Catch year Sampling date	Fish species	Number	Age (yr)	Length (cm)	Weight (g)	Hg mg/kg wet wt					Minimum limit exceeded by, %
								Mean	SD	Median	Min	Max	
Jönköping	Eksjö	1998	Pike	2		50, 70	984, 2109	0.2, 1.1	0.7				50
Jönköping	Eksjö	1992, 1995, 2000, 2002	Pike	25		51 (40-62)	906 (420-1559)	0.6	0.2	0.6	0.2	1.0	0
Jönköping	Gislaved	1991	Pike	40		52 (43-66)	935 (440-1860)	0.5	0.2	0.5	0.1	0.9	0
Jönköping	Gislaved	1993	Pike	30	5.5 (3-10)	52 (40-68)	945 (380-1778)	0.6	0.3	0.5	0.3	1.6	13
Jönköping	Gislaved	1994	Pike	53	4.6 (3-7)	49 (32-76)	914 (390-2000)	0.5	0.3	0.4	0.1	1.5	3.8
Jönköping	Gislaved	1995	Pike	12	5.5 (3-7)	54 (47-62)	1001 (600-1734)	0.7	0.2	0.7	0.4	1.3	8.3
Jönköping	Gislaved / Gnosjö	2002	Pike	5		48 (38-62)	631 (284-1047)	1.3	0.6	0.9	0.8	2.1	40
Jönköping	Gislaved / Gnosjö	2003	Pike	9		49 (43-63)	699 (451-1591)	0.8	0.2	0.8	0.6	1.1	11
Jönköping	Gnosjö	1990, 1994, 2002	Pike	24		49 (38-61)	854 (451-1750)	0.5	0.2	0.5	0.2	0.8	0.0
Jönköping	Gnosjö	1991	Pike	44		54 (44-73)	1177 (600-2550)	0.8	0.4	0.7	0.2	1.8	27
Jönköping	Gnosjö	1992	Pike	45		57 (44-76)	1116 (465-2100)	0.8	0.4	0.7	0.3	1.9	27
Jönköping	Gnosjö	1993	Pike	18		53 (39-70)	1093 (510-2000)	0.9	0.3	0.8	0.6	1.8	22
Jönköping	Gnosjö	1995	Pike	33	5.5 (3-9)	53 (41-66)	1047 (620-1700)	0.6	0.5	0.5	0.1	2.5	6.1
Jönköping	Gnosjö	2002	Pike	5		54 (49-57)	843 (632-1008)	0.5	0.0	0.5	0.4	0.5	0.0
Jönköping	Gnosjö	2003	Pike	18	5.9 (3-9)	52 (36-68)	967 (263-2174)	1.1	0.6	0.8	0.4	2.3	44
Jönköping	Habo	1994	Pike	8	6 (3-11)	55 (48-65)	1103 (634-2000)	0.5	0.1	0.5	0.3	0.6	0
Jönköping	Habo	2002	Pike	5		50 (41-57)	773 (394-1218)	1.0	0.2	1.0	0.7	1.3	40
Jönköping	Habo/Mullsjö	2004	Pike	5		51 (39-68)	862 (511-1447)	1.1	0.2	1.0	0.8	1.4	40
Jönköping	Jönköping	1990, 1992, 1995	Pike	25		51 (46-62)	1060 (640-2050)	0.4	0.3	0.3	0.1	1.0	0
Jönköping	Jönköping	1993	Pike	10		53 (44-64)	962 (501-1618)	0.6	0.5	0.5	0.2	1.6	10
Jönköping	Jönköping	1994	Pike	89	5.5 (3-10)	51 (30-72)	936 (284-2400)	0.4	0.3	0.3	0.1	1.4	3.4
Jönköping	Jönköping	1997	Pike	4	5.3 (4-7)	52 (47-56)	998 (770-1110)	0.7	0.3	0.7	0.4	1.2	25
Jönköping	Jönköping	1998	Pike	5	4.4 (4-5)	52 (47-58)	838 (673-1196)	0.8	0.4	0.7	0.5	1.4	20
Jönköping	Jönköping	2003	Pike	5		53 (47-63)	983 (597-1572)	0.7	0.2	0.7	0.4	0.9	0

Appendix

County	Municipality	Catch year Sampling date	Fish species	Number	Age (yr)	Length (cm)	Weight (g)	Hg mg/kg wet wt					Minimum limit exceeded by, %
								Mean	SD	Median	Min	Max	
Jönköping	Nässjö	1990, 1993-1996	Pike	88	5.8 (3-9)	51 (39-67)	948 (370-2250)	0.5	0.2	0.4	0.0	1.0	0
Jönköping	Nässjö	2003	Pike	10		50 (40-73)	783 (349-2040)	0.6	0.6	0.5	0.5	2.1	10
Jönköping	Nässjö	2004	Pike	5		49 (46-55)	713 (566-919)	0.6	0.1	0.6	0.5	0.8	0
Jönköping	Sävsjö	1992	Pike	8		56 (42-67)	1154 (680-2100)	0.6	0.1	0.6	0.5	0.7	0
Jönköping	Sävsjö	1993	Pike	85		53 (37-86)	1034 (350-2500)	0.6	0.4	0.4	0.1	2.2	15
Jönköping	Sävsjö	2002	Pike	13		54 (34-69)	1296 (298-2133)	0.3	0.3	0.3	0.1	1.0	0
Jönköping	Sävsjö	2003	Pike	15		53 (30-64)	972 (456-1544)	0.7	0.6	0.5	0.1	1.7	33
Jönköping	Tranås	1991, 1994	Pike	52	5.2 (3-10)	49 (36-64)	857 (253-1747)	0.3	0.1	0.2	0.1	0.6	0
Jönköping	Vaggeryd	1991	Pike	2		48, 37	590	0.3, 0.2					0
Jönköping	Vaggeryd	1993	Pike	17	6.4 (8-10)	53 (38-63)	1005 (488-1750)	0.7	0.4	0.9	0.1	1.7	12
Jönköping	Vaggeryd	1994	Pike	24	6.3 (3-12)	52 (42-65)	1038 (475-2000)	0.6	0.4	0.5	0.1	1.6	12
Jönköping	Vaggeryd	1996	Pike	1		43		0.4					
Jönköping	Vaggeryd	1999	Pike	4		25 (0-51)	900 (700-1100)	1.0	0.5	0.9	0.5	1.4	50
Jönköping	Vetlanda	1993, 1994	Pike	29	5 (3-8)	51 (39-64)	915 (300-1680)	0.5	0.2	0.4	0.1	0.9	0
Jönköping	Värnamo	1990, 1993, 2002, 2003	Pike	31		55 (36-72)	1127 (296-2273)	0.5	0.3	0.6	0.2	1.0	0
Jönköping	Värnamo	1994	Pike	56	6 (3-10)	49 (39-65)	977 (345-2000)	0.5	0.3	0.4	0.1	1.5	3.6
Jönköping	Värnamo	2004	Pike	5	7 (7-8)	58 (54-62)	1060 (823-1270)	0.9	0.3	1.0	0.5	1.3	40
Kronoberg	Lessebo	1990	Pike	13			565 (480-615)	0.7	0.1	0.6	0.5	1	0
Kronoberg	Tingsryd	1990	Pike	12		50 (38-59)	846 (313-1502)	0.8	0.3	0.8	0.4	1.2	25
Kronoberg	Uppvidinge (Säljen)	1990	Pike	6		47 (39-52)	1013 (690-1270)	1.3	0.3	1.3	1.0	1.7	83
Kronoberg	Lessebo	1991	Pike	5		56 (45-66)	1135 (469-1738)	0.6	0.1	0.6	0.6	0.8	0
Kronoberg	Tingsryd	1991	Pike	11		49 (37-63)	784 (305-1461)	0.8	0.5	0.8	0.3	1.9	27
Kronoberg	Växjö, Ljungby	1993, 2002	Pike	20		60 (45-77)	1465 (634-2800)	0.4	0.2	0.4	0.2	0.8	0
Kronoberg	Ljungby	1994	Pike	10	7 (4-10)	54 (44-66)	1145 (476-2400)	0.3	0.2	0.3	0.2	0.7	0

Appendix

County	Municipality	Catch year Sampling date	Fish species	Number	Age (yr)	Length (cm)	Weight (g)	Hg mg/kg wet wt					Minimum limit exceeded by, %
								Mean	SD	Median	Min	Max	
Kronoberg	Tingsryd (Viren)	1997	Pike	1			967	1.2					
Kronoberg	Tingsryd (Viren)	1999	Pike	5			1100 (800-1500)	0.6	0.1	0.6	0.4	0.8	0
Kronoberg	Alvesta	2004	Pike	21	6 (4-11)	59 (46-78)	1417 (670-3280)	0.7	0.3	0.6	0.4	1.3	19
Norrbottn	Arjeplog, Boden, Kiruna, Övertorneå, Arvidsjaur, Piteå, Överkalix, Pajala, Haparanda, Gällivare, Jokkmokk, Kalix	1992	Pike	310	6 (3-14)	52 (37-76)	929 (253-3161)	0.3	0.2	0.2	0.0	0.8	0
Norrbottn	Älvsbyn	1992	Pike	35	8.7 (6-14)	55 (38-75)	1130 (305-2630)	0.4	0.2	0.4	0.1	1.1	2.9
Norrbottn	Arjeplog	1997	Pike	30	5 (4-8)	59 (50-67)	1283 (825-1865)	0.5	0.2	0.5	0.2	1.1	3.3
Norrbottn	Boden	1997	Pike	49	4.2 (1-7)	53 (38-61)	861 (290-1290)	0.6	0.4	0.6	0.0	1.8	8.2
Norrbottn	Haparanda	1997	Pike	20	4.8 (1-12)	58 (38-87)	1273 (320-4400)	0.8	0.4	0.7	0.3	1.5	25
Norrbottn	Arvidsjaur, Gällivare, Jokkmokk, Kiruna	1997	Pike	59	5.1 (2-9)	54 (46-70)	1006 (565-2180)	0.3	0.2	0.3	0.1	0.8	0
Norrbottn	Haparanda, Kalix	1999	Pike	29	5.3 (3-8)	58 (46-66)	1225 (660-1880)	0.3	0.1	0.3	0.2	0.4	0
Stockholm	Nynäshamn	1990	Pike	5		46 (42-50)	1004 (730-1350)	0.1	0.0	0.1	0.1	0.2	0
Stockholm	Tyresö	2004	Pike	23	7.4 (5-10)	49 (38-73)	929 (325-2930)	1.0	0.3	0.9	0.6	1.8	35
Södermanland	Gnesta	1993	Pike	5		60 (55-68)	1236 (960-1830)	0.9	0.4	0.8	0.5	1.4	40.0
Södermanland	Eskiltuna, Flen	1993	Pike	67		54 (43-69)	933 (520-1850)	0.4	0.2	0.3	0.1	0.9	0
Södermanland	Gnesta, Strängnäs	1996	Pike	10		51 (40-58)	875 (370-1250)	0.3	0.1	0.3	0.2	0.4	0
Södermanland	Eskiltuna, Flen, Strängnäs, Trosa, Nyköping, Katrineholm, Vingåker, Gnesta	1997	Gädda	143		54 (40-84)	1016 (375-3400)	0.3	0.2	0.3	0.1	1	0
Södermanland	Vingåker	2000	Pike	5			900 (700-1200)	0.7	0.1	0.8	0.5	0.8	0
Södermanland	Nyköping	1993	Pike (freshwater)	72		55 (43-69)	954 (520-1830)	0.4	0.3	0.3	0.1	1.4	2.8

Appendix

County	Municipality	Catch year Sampling date	Fish species	Number	Age (yr)	Length (cm)	Weight (g)	Hg mg/kg wet wt					Minimum limit exceeded by, %
								Mean	SD	Median	Min	Max	
Södermanland	Nyköping	1997, 1998	Pike (freshwater)	16		55 (45-67)	1047 (561-1700)	0.1	0.1	0.1	0.1	0.3	0
Södermanland	Gnesta	1996, 1997	Pike (freshwater)	23		52 (40-62)	888 (370-1760)	0.4	0.2	0.4	0.1	0.9	0
Södermanland	Örebro	2001	Pike	6				0.8	0.4	0.8	0.4	1.3	33
Södermanland	Flen, Nyköping	2001	Pike	5				0.6	0.1	0.6	0.4	0.7	0
Södermanland	Finspång	2002	Pike	5				1.0	0.3	0.9	0.7	1.3	40
Södermanland	Flen, Katrineholm	2002	Pike	7				0.8	0.1	0.8	0.6	0.9	0
Södermanland	Vingåker	2003	Pike	2				0.2. 0.3				0	
Värmland	Arvika	1993	Pike	10		53 (45-63)	984 (600-1750)	0.4	0.1	0.4	0.3	0.6	0
Värmland	Arvika	1995	Pike	86		53 (37-79)	1004 (400-1600)	0.7	0.3	0.7	0.3	1.6	13
Värmland	Arvika	1996	Pike	30		55 (30-92)	1307 (250-4999)	0.8	0.6	0.6	0.3	2.8	27
Värmland	Arvika	1997	Pike	40		53 (45-63)	995 (550-1690)	0.7	0.2	0.6	0.4	1.4	8
Värmland	Arvika	1998	Pike	102		52 (34-75)	1002 (352-2990)	1.0	0.2	0.7	0.3	1.7	11
Värmland	Arvika	1999	Pike	5		50 (38-62)	1268 (550-2170)	1.0	0.6	0.6	0.5	1.9	40
Värmland	Eda	1990	Pike	14		52 (38-70)	1322 (590-2600)	1.1	0.4	1.0	0.5	1.9	36
Värmland	Eda	1991	Pike	13			804 (300-1300)	0.5	0.4	0.4	0.1	1.3	15
Värmland	Eda	1995	Pike	5		57 (51-61)	1970 (975-5320)	1.0	0.5	0.7	0.6	1.9	20
Värmland	Eda	1992, 1994, 1997, 1999	Pike	34		52 (40-78)	958 (315-2650)	0.5	0.2	0.5	0.1	1	0
Värmland	Eda (Skårsjön)	1998	Pike	5			2380 (600-5100)	1.6	1.3	1.1	0.5	3.8	60
Värmland	Eda (Askesjön)	2003	Pike	1			12000	4.7					
Värmland	Filipstad	1993	Pike	5			990 (700-1450)	1.0	0.5	0.8	0.7	1.9	20
Värmland	Filipstad	1994, 1996	Pike	30		55 (48-65)	1094 (700-1600)	0.6	0.1	0.6	0.4	0.9	0
Värmland	Filipstad	1995	Pike	37		52 (36-65)	961 (600-1500)	0.6	0.3	0.7	0.1	1.2	2.7
Värmland	Filipstad	1997	Pike	8		52 (44-59)	874 (630-1155)	0.6	0.4	0.4	0.2	1.3	25

Appendix

County	Municipality	Catch year Sampling date	Fish species	Number	Age (yr)	Length (cm)	Weight (g)	Hg mg/kg wet wt					Minimum limit exceeded by, %
								Mean	SD	Median	Min	Max	
Värmland	Forshaga (Vågsjöarna)	1999	Pike	5		49 (43-59)	750 (443-1280)	2.8	0.9	3.0	1.7	3.6	100
Värmland	Forshaga (Emsen)	2000	Pike	5		51 (42-56)	1294 (700-1570)	0.2	0.1	0.2	0.1	0.3	0
Värmland	Grums	1996	Pike	6		48 (39-52)	725 (500-900)	0.5	0.1	0.4	0.4	0.7	0
Värmland	Grums	1997	Pike	10		47 (41-54)	842 (480-1270)	0.5	0.3	0.5	0.3	1.2	10
Värmland	Hagfors	1990	Pike	40		46 (32-63)	875 (310-1600)	0.9	0.3	0.9	0.3	1.9	25
Värmland	Hagfors	1991	Pike	5			696 (610-980)	0.7	0.4	0.7	0.2	1.1	20
Värmland	Hagfors	1992	Pike	25			1047 (205-1820)	0.7	0.4	0.7	0.2	1.8	12
Värmland	Hagfors	1993	Pike	15			952 (600-1300)	1.1	0.5	1	0.4	1.8	40
Värmland	Hagfors	1994	Pike	15			961 (635-1740)	0.9	0.2	0.9	0.5	1.1	33
Värmland	Hagfors	1996	Pike	29			1102 (700-1700)	0.6	0.2	0.7	0.3	1.4	3.4
Värmland	Hagfors	1997, 1998	Pike	24			951 (730-1610)	0.4	0.1	0.4	0.3	0.7	0
Värmland	Karlstad	1994, 1997, 2000	Pike	39		53 (42-75)	1165 (521-2503)	0.5	0.2	0.4	0.2	0.9	0
Värmland	Karlstad	1995	Pike	13		54 (46-68)	1100 (560-1770)	0.9	0.4	0.8	0.3	1.4	38
Värmland	Karlstad	1996	Pike	5		46 (41-54)	610 (373-946)	1.0	0.3	1.0	0.6	1.4	40
Värmland	Kristinehamn	1993	Pike	10		57 (51-63)	1045 (700-1250)	1.0	0.4	1.0	0.7	1.6	50
Värmland	Kristinehamn	1994, 1997	Pike	15		53 (40-68)	871 (505-1320)	0.4	0.1	0.4	0.2	0.6	0
Värmland	Kristinehamn	1998	Pike	20		53 (46-66)	875 (571-1351)	0.8	0.4	0.6	0.5	2.0	10
Värmland	Kristinehamn	2002	Pike	10		55 (47-63)	980 (650-1300)	1.0	0.2	0.9	0.7	1.3	30
Värmland	Sunne	1990, 1995, 2000	Pike	25		53 (47-59)	1071 (780-1310)	0.7	0.2	0.7	0.2	1.0	0
Värmland	Sunne	1992	Pike	10			766 (370-2180)	0.5	0.3	0.4	0.2	1.4	10
Värmland	Sunne	1993	Pike	15			1070 (645-1690)	1.1	0.3	1	0.6	1.7	47
Värmland	Sunne	1996	Pike	23		57 (40-75)	1174 (250-2610)	0.8	0.5	0.7	0.3	2.3	22
Värmland	Sunne	1998	Pike	25		55 (43-65)	1015 (481-1830)	0.6	0.2	0.5	0.3	1.1	4.0
Värmland	Sunne (Gårdsjön)	2001	Pike	4		50 (46-57)	1300 (1030-1730)	1.4	0.3	1.6	1.0	1.6	75

Appendix

County	Municipality	Catch year Sampling date	Fish species	Number	Age (yr)	Length (cm)	Weight (g)	Hg mg/kg wet wt					Minimum limit exceeded by, %
								Mean	SD	Median	Min	Max	
Värmland	Säffle	1991	Pike	50		59 (52-71)	1186 (900-1700)	0.8	0.2	0.8	0.4	1.3	10
Värmland	Säffle	1992	Pike	10			1270 (800-2700)	1.1	0.3	1.1	0.8	1.9	50
Värmland	Säffle	1993	Pike	28			1151 (472-1888)	0.8	0.3	0.8	0.3	1.4	29
Värmland	Säffle	1994	Pike	79		58 (50-67)	1174 (600-1800)	0.6	0.3	0.7	0.1	1.2	5
Värmland	Säffle	1995	Pike	26			957 (500-1700)	0.6	0.2	0.6	0.2	1.1	3.8
Värmland	Säffle	1996, 1998, 1999, 2001-03	Pike	74		55 (49-64)	1117 (761-1480)	0.4	0.1	0.4	0.2	0.7	0
Värmland	Säffle	1997	Pike	22			1043 (600-1800)	0.5	0.3	0.5	0.2	1.1	4.5
Värmland	Säffle	2000	Pike	20		51 (34-64)	957 (238-1700)	0.4	0.2	0.3	0.1	1.1	5
Värmland	Torsby	1991	Pike	9			971 (540-1780)	0.8	0.4	0.7	0.5	1.6	22
Värmland	Torsby	1993	Pike	15		56 (49-66)	1089 (700-1650)	0.6	0.3	0.7	0.2	1.1	7
Värmland	Torsby	1995	Pike	15		54 (42-70)	1049 (490-1850)	1.0	0.5	0.8	0.5	2.1	33
Värmland	Torsby	1996	Pike	15		56 (49-62)	1093 (650-1480)	0.6	0.3	0.5	0.3	1.2	13
Värmland	Årjäng	1990	Pike	4		44 (39-50)	860 (610-1140)	0.9	0.1	0.9	0.8	1.1	25
Värmland	Årjäng (Rinen, Södra Yxesjön)	1991	Pike	4			1063 (1000-1100)	1.2	0.2	1.2	1	1.5	75
Värmland	Årjäng	1992	Pike	29		44 (31-66)	971 (325-2760)	0.8	0.2	0.7	0.4	1.4	10
Värmland	Årjäng	1993	Pike	58			957 (450-1354)	0.8	0.4	0.9	0.3	2.6	31
Värmland	Årjäng	1994	Pike	119		46 (35-62)	865 (323-1870)	0.6	0.3	0.5	0.2	1.5	9
Värmland	Årjäng	1995	Pike	35			1000 (1000-1000)	0.6	0.1	0.6	0.4	0.8	0
Värmland	Årjäng	1996	Pike	42			980 (160-2050)	0.8	0.7	0.6	0.4	4.8	17
Värmland	Årjäng	1997	Pike	9		53 (36-80)	1068 (290-3050)	0.6	0.3	0.7	0.2	1.1	11
Värmland	Årjäng	1998	Pike	20		50 (40-59)	722 (365-1120)	0.7	0.2	0.7	0.5	1.3	10
Västerbotten	Sorsele, Skellefteå	1990	Pike	79		47 (33-79)	1011 (360-3117)	0.7	0.3	0.6	0.2	1.3	10
Västerbotten	Sorsele, Bjurholm	1991	Pike	20		56 (36-67)	1277 (620-2170)	0.7	0.4	0.7	0.3	1.6	25

Appendix

County	Municipality	Catch year Sampling date	Fish species	Number	Age (yr)	Length (cm)	Weight (g)	Hg mg/kg wet wt					Minimum limit exceeded by, %
								Mean	SD	Median	Min	Max	
Västerbotten	Dorotea, Sorsele	1992-1994, 1996-2001, 2003	Pike	82	5.4 (3-9)	60 (39-70)	1479 (388-2318)	0.3	0.1	0.3	0.2	0.6	0
Västernorrland	Örnsköldsvik	1990, 1991	Pike	10		50 (40-59)	981 (540-1575)	0.5	0.2	0.5	0.2	0.8	0
Västernorrland	Timrå (Aspen)	1991	Pike	6		50 (44-57)	815 (502-1375)	1.5	0.4	1.6	0.8	1.9	83
Västernorrland	Sundsvall, Timrå	1992	Pike	28		51 (44-60)	893 (484-1565)	0.6	0.2	0.6	0.3	1.0	0
Västernorrland	Örnsköldsvik	1992	Pike	16		50 (39-60)	828 (332-1475)	0.7	0.2	0.6	0.3	1.1	13
Västernorrland	Sollefteå (Graningesjön, Simsjön)	1992	Pike	8		54 (44-62)	1078 (556-1725)	1.2	0.3	1.2	0.9	1.6	75
Västernorrland	Härnösand (Lill-Roten)	1992	Pike	5		50 (46-55)	779 (568-1016)	1.1	0.3	1.2	0.8	1.5	60
Västernorrland	Ånge (Skärvingen)	1992	Pike	6		56 (53-60)	1052 (846-1205)	1.3	0.3	1.3	0.9	1.7	67
Västmanland	Fagersta	1990	Pike	8		48 (40-56)	839 (550-1200)	1.0	0.3	0.9	0.7	1.4	25
Västmanland	Norberg	1990	Pike	5		45 (42-52)	890 (640-1490)	0.9	0.2	0.9	0.7	1.1	20
Västmanland	Skinnskatteberg	2003	Pike	22	6 (3-11)	56 (25-91)	1111 (88-3750)	1.1	1.1	0.8	0.3	5.6	41
Västra Götaland	Bengtfors, Mellerud, Åmål	1990, 1991, 1994	Pike	13		50 (30-64)	1075 (134-2080)	0.5	0.2	0.5	0.3	0.9	0
Örebro	Örebro	1990	Pike	3			1027 (900-1130)	0.6	0.5	0.3	0.3	1.1	33
Örebro	Örebro	1992	Pike	6			925 (650-1188)	0.5	0.3	0.3	0.2	1.1	17
Örebro	Örebro	1996	Pike	5			1076 (780-1180)	0.6	0.7	0.3	0.2	1.8	20
Örebro	Örebro	1997	Pike	1			900	0.6					
Örebro	Örebro	1999	Pike	11				0.5	0.4	0.3	0.2	1.2	9.1
Örebro	Örebro	2000	Pike	22			845 (600-1700)	1.0	0.8	0.8	0.2	3.6	27
Örebro	Örebro	2001	Pike	45			946 (600-1650)	0.9	0.5	0.7	0.3	2.7	36
Örebro	Örebro	2002	Pike	10			925 (600-1550)	0.9	0.4	0.8	0.6	1.7	30
Örebro	Örebro	2003	Pike	20			1081 (650-1800)	0.3	0.2	0.3	0.1	0.9	0
Örebro	Örebro	2004	Pike	40			1005 (600-1750)	0.9	0.6	0.6	0.2	2.5	38
Örebro	Ljusnarsberg	1991, 1992	Pike	14		42 (34-71)	789 (240-1960)	0.5	0.2	0.5	0.3	0.9	0

Appendix

County	Municipality	Catch year Sampling date	Fish species	Number	Age (yr)	Length (cm)	Weight (g)	Hg mg/kg wet wt					Minimum limit exceeded by, %
								Mean	SD	Median	Min	Max	
Örebro	Hallsberg	1995, 1997	Pike	9		52 (50-56)	824 (700-1140)	0.3	0.3	0.2	0.1	0.9	0
Örebro	Nora	1990	Pike	1			880	1.3					
Örebro	Nora	1992	Pike	1			840	1.7					
Örebro	Nora (Gryttjärn)	1993	Pike	14		49 (45-55)	849 (480-1580)	1.4	0.6	1.6	0.4	2.2	64
Örebro	Nora	1998	Pike	7		49 (46-53)	828 (670-1060)	0.4	0.1	0.4	0.3	0.6	0
Örebro	Nora (Sången, Vikern)	1999	Pike	9		56 (45-65)	1167 (620-1700)	1.4	0.5	1.4	0.7	2.1	78
Örebro	Nora	2000	Pike	13		54 (45-66)	1368 (736-2690)	1.0	0.6	0.8	0.3	2.1	46
Örebro	Nora	2001	Pike	16		50 (40-61)	1151 (580-2100)	1.1	0.3	1.1	0.6	1.6	50
Örebro	Nora	2002	Pike	14		61 (52-75)	1606 (950-2050)	0.9	0.2	0.9	0.6	1.5	21
Örebro	Lekeberg	1999	Pike	1				1.2					
Örebro	Lekeberg	2002	Pike	3		43 (40-45)	893 (680-1051)	1.0	0.1	1.0	0.9	1.1	33
Örebro	Lindesberg	1992	Pike	10			1013 (700-1390)	0.8	0.4	0.9	0.2	1.4	50
Örebro	Lindesberg	1993	Pike	8			970 (690-1350)	0.7	0.3	0.6	0.4	1.1	25
Örebro	Lindesberg	1995	Pike	4			985 (800-1220)	1.0	0.3	1.0	0.6	1.3	50
Örebro	Lindesberg	1996	Pike	2			1390, 1060	0.5; 1.3	0.3	0.1	0.3	0.2	0
Örebro	Lindesberg	1997, 2000	Pike	14			744 (600-900)	0.3	0.1	0.3	0.2	0.6	0
Örebro	Laxå	1992	Pike	2		54, 52	863, 807	0.6; 0.5					
Örebro	Laxå	1994	Pike	1			1220	1.7					
Örebro	Laxå	1996	Pike	2			1100, 980	0.7; 1.1					
Örebro	Laxå	2000	Pike	10		55 (48-58)	1116 (850-1370)	0.9	0.2	0.8	0.7	1.1	20
Örebro	Laxå	2001	Pike	10		55 (44-63)	1119 (580-1740)	1.2	0.7	1.1	0.4	2.5	50
Örebro	Karlskoga	1990	Pike	9		47 (44-52)	1023 (800-1486)	1.1	0.4	1.2	0.4	1.6	67
Örebro	Karlskoga	1992	Pike	2			951, 951	1.2; 1.2					
Örebro	Karlskoga	1997	Pike	13		62 (51-77)	1487 (870-2570)	0.8	0.3	0.7	0.4	1.3	23

Appendix

County	Municipality	Catch year Sampling date	Fish species	Number	Age (yr)	Length (cm)	Weight (g)	Hg mg/kg wet wt					Minimum limit exceeded by, %
								Mean	SD	Median	Min	Max	
Örebro	Karlskoga	1998	Pike	10		53 (41-66)	899 (390-1560)	1.0	0.3	0.9	0.7	1.6	40
Örebro	Karlskoga (Kärmen, Våtsjön)	2001	Pike	15				1.2	0.6	1.4	0.3	2.1	67
Örebro	Karlskoga	1995, 2002	Pike	17			1026 (770-1300)	0.7	0.2	0.7	0.4	1	0
Örebro	Degerfors	1990	Pike	1			1350	1.7					
Örebro	Degerfors	1991	Pike	1			840	1.6					
Örebro	Degerfors	1992	Pike	1			770	1.5					
Örebro	Degerfors	1994	Pike	10		56 (45-66)	988 (600-1300)	0.7	0.2	0.7	0.4	0.9	0
Örebro	Hällefors	1990	Pike	4			744 (480-1025)	1.1	0.3	1.0	0.8	1.5	50
Örebro	Hällefors (Stora Hällsjön, Stora Änsjön)	1991	Pike	4			775 (460-1210)	1.3	0.4	1.2	0.9	1.9	75
Örebro	Hällefors	1992	Pike	9			822 (630-990)	1.2	0.5	1.0	0.6	2.1	44
Örebro	Hällefors	1993	Pike	1			850	1.8					
Örebro	Hällefors	1996	Pike	7		54 (50-58)	1070 (680-1750)	0.9	0.4	1.1	0.4	1.4	57
Örebro	Hällefors	1997	Pike	9		59 (49-65)	1281 (770-1950)	1.0	0.5	0.8	0.5	2.0	33
Örebro	Hällefors	1998	Pike	3				1.0	0.3	0.9	0.7	1.3	33
Örebro	Hällefors	1999	Pike	1			1300	0.8					
Örebro	Askersund	1990	Pike	1			610	1.6					
Örebro	Askersund	1991	Pike	1			760	1.6					
Örebro	Askersund	1992	Pike	2			920, 790	1.6; 1.1					
Örebro	Askersund	1994, 1995, 1999	Pike	18		55 (43-76)	1224 (500-3460)	0.5	0.2	0.5	0.3	1.0	0
Örebro	Askersund	1996	Pike	1			1140	1.1					
Örebro	Askersund	1997	Pike	9		54 (44-65)	1013 (520-1820)	1.2	0.7	0.9	0.5	2.8	44
Örebro	Askersund	1998	Pike	15		52 (45-57)	932 (620-1500)	0.9	0.3	0.9	0.6	1.6	27
Örebro	Askersund	2000	Pike	5		52 (49-57)	1259 (964-1580)	1.0	0.3	0.9	0.8	1.5	20

Appendix

County	Municipality	Catch year Sampling date	Fish species	Number	Age (yr)	Length (cm)	Weight (g)	Hg mg/kg wet wt					Minimum limit exceeded by, %
								Mean	SD	Median	Min	Max	
Örebro	Askersund	2001	Pike	10		48 (40-56)	962 (680-1250)	0.9	0.3	0.7	0.5	1.4	30
Örebro	Lindesberg, Hällefors, Hallberg, Degerfors, Nora, Askersund, Laxå	2005	Pike	7				0.6	0.2	0.6	0.3	0.9	7
Örebro	Hällefors	2006	Pike	6				0.8	0.2	0.7	0.5	1.1	17
Örebro	Hällefors, Filipstad, Degerfors, Laxå	2006	Pike	3				0.4	0.1	0.5	0.3	0.5	0
Östergötland	Motala	1990	Pike	1		49	1120	1.7					
Östergötland	Finspång, Söderköping	1990, 1997	Pike	10		51 (46-54)	1091 (950-1520)	0.4	0.1	0.4	0.3	0.6	0
Östergötland	Motala	2001	Pike	5		47 (40-56)	916 (680-1130)	1.1	0.3	1.2	0.6	1.4	60
Blekinge	Karlskrona ¹	1991	Perch	12		31 (27-37)	698 (360-990)	1.1	0.7	0.9	0.3	2.9	75
Dalarna	Ludvika	1993	Perch	5			143 (115-165)	0.1	0.0	0.1	0.1	0.1	0
Dalarna	Gagnef	2000-2004	Perch	86	1.7 (1-4)	59 (5,3-137)	17 (1,3-42)	0.1	0.0	0.1	0.1	0.3	0
Dalarna	Avesta	1994	Pike-perch	2		32, 40	360, 920	0.1, 0.1					0
Gävleborg	Gävle (Vittersjön)	1990	Perch	5				1.0	0.3	0.8	0.8	1.4	100
Gävleborg	Hudiksvall	1999, 2000, 2001, 2002, 2004	Perch	101	2.1 (1-6)	27 (4.6-116)	8.3 (0.9-22)	0.2	0.1	0.2	0.1	0.5	0
Gävleborg	Hudiksvall	2003	Perch	39	4.6 (2-9)	130 (78-199)	31 (4.4-89)	0.3	0.2	0.3	0.1	0.8	10
Gävleborg	Ljusdal	1997-2004	Perch	190	2.9 (1-10)	30 (7-117)	26 (3-75)	0.1	0.1	0.1	0.0	0.5	0
Halland	Falkenberg	1990-1992, 1994	Perch	45	4.3 (3-6)	14 (13-24)	43 (21-260)	0.2	0.0	0.2	0.1	0.3	0
Halland	Falkenberg	1993	Perch	10	4.9 (3-6)	15 (13-18)	40 (28-62)	0.4	0.2	0.4	0.1	0.8	10
Halland	Halmstad	1997, 2001	Perch	29	3 (1-7)	12 (7-18)	23 (3-70)	0.2	0.1	0.2	0.1	0.4	0
Halland	Halmstad	1999	Perch	20	1.8 (1-4)	10 (7-12)	9.1 (3-17)	0.4	0.1	0.4	0.3	0.8	10
Halland	Halmstad	2000	Perch	20	2 (1-5)	9.7 (7-13)	9.5 (3-18)	0.4	0.2	0.3	0.1	0.8	15
Halland	Halmstad	2003	Perch	20	3 (1-5)	88 (64-112)	7 (2.3-15)	0.3	0.1	0.3	0.2	0.7	15

Appendix

County	Municipality	Catch year Sampling date	Fish species	Number	Age (yr)	Length (cm)	Weight (g)	Hg mg/kg wet wt					Minimum limit exceeded by, %
								Mean	SD	Median	Min	Max	
Halland	Halmstad	2004	Perch	20	3 (2-5)	96 (80-112)	9.4 (5-15)	0.3	0.1	0.2	0.1	0.6	5
Jämtland	Strömsund	2000-2003	Perch	40	6 (4-10)	17 (16-19)	54 (44-71)	0.2	0.1	0.1	0.1	0.4	0
Jönköping	Gislaved	1999-2004	Perch	119	1.8 (1-5)	40 (5-133)	9 (0.9-24)	0.1	0.1	0.1	0.1	0.1	0
Jönköping	Gislaved (Hagsjön)	2003	Perch	4		23 (17-26)	148 (50-211)	0.6	0.1	0.6	0.4	0.8	75
Kalmar	Västervik, Vimmerby	1991-1994, 1997, 1999	Perch	60	4.7 (4-6)	15 (12-19)	37 (18-70)	0.3	0.1	0.3	0.2	0.5	0
Kalmar	Vimmerby	1990	Perch	10	4.7 (4-6)	14 (13-16)	29 (23-39)	0.4	0.1	0.4	0.3	0.7	10
Kalmar	Vimmerby	1995	Perch	10	4.8 (4-6)	14 (13-15)	26 (22-29)	0.4	0.1	0.4	0.2	0.6	20
Kalmar	Emmaboda	1999	Perch	21	1.4 (0-4)	9 (5-12)	7 (1-14)	0.5	0.2	0.5	0.3	1.1	29
Kalmar	Emmaboda	2000	Perch	26	2.3 (1-4)	11 (8.7-14)	13 (5.7-27)	0.4	0.2	0.4	0.2	0.7	23
Kalmar	Emmaboda	2001	Perch	14	2.2 (1-3)	10 (9.4-11)	11 (8.3-13)	0.5	0.1	0.5	0.3	0.7	29
Kalmar	Emmaboda	2002	Perch	20	2.5 (0-7)	10 (5-14)	11 (0.9-30)	0.5	0.2	0.4	0.2	0.9	35
Kalmar	Vimmerby	2002	Perch	10		17 (16-18)	46 (38-56)	0.5	0.1	0.4	0.3	0.8	30
Kalmar	Emmaboda	2003	Perch	20	2.4 (2-5)	91 (76-126)	7.5 (4-20)	0.4	0.2	0.3	0.2	1.1	15
Kalmar	Vimmerby	2003	Perch	10	6 (5-7)	19 (18-20)	74 (57-85)	0.5	0.1	0.5	0.3	0.7	10
Kalmar	Emmaboda	2004	Perch	20	3.8 (2-9)	99 (81-122)	10 (5-18)	0.4	0.2	0.4	0.2	1.0	15
Kronoberg	Alvesta, Ljungby, Uppvidinge	1998-2004	Perch	197	3.4 (0-12)	36 (5-192)	39 (1-600)	0.1	0.1	0.1	0.0	0.4	0
Norrbottn	Haparanda, Luleå, Piteå, Gällivare, Pajala	2004	Perch	7	5 (4-7)	17 (16-19)	55 (45-76)	0.1	0.1	0.1	0.1	0.3	0
Norrbottn	Haparanda, Luleå, Piteå, Gällivare, Pajala	2005	Perch	7	6 (5-9)	19 (18-19)	65 (61-69)	0.1	0.1	0.1	0.1	0.3	0
Skåne	Ystad	2000-2003	Perch	40	3.3 (2-8)	17 (16-19)	55 (28-82)	0.1	0.1	0.1	0.0	0.3	0
Stockholm	Haninge, Södertälje, Tyresö	1999-2004	Perch	300	2 (1-8)	29 (5-132)	15 (1-83)	0.2	0.1	0.2	0.0	0.5	0
Stockholm	Södertälje	2000	Perch	10				0.3	0.1	0.2	0.2	0.6	10
Stockholm	Södertälje	2002	Perch	10	6 (4-8)	18 (17-19)	63 (53-73)	0.4	0.1	0.4	0.1	0.6	20

Appendix

County	Municipality	Catch year Sampling date	Fish species	Number	Age (yr)	Length (cm)	Weight (g)	Hg mg/kg wet wt					Minimum limit exceeded by, %
								Mean	SD	Median	Min	Max	
Stockholm	Tyresö	2003	Perch	38	3.8 (2-7)	14 (8-25)	47 (5-170)	0.3	0.2	0.3	0.1	1.0	11
Stockholm	Vallentuna (Tärnan)	2002	Perch	10	8 (5-9)	18 (17-19)	55 (51-60)	0.5	0.2	0.6	0.2	0.9	60
Stockholm	Haningen	2003	Perch	40	2 (1-5)	11 (7-14)	13 (3-26)	0.3	0.2	0.4	0.1	0.6	2.5
Stockholm	²	2004	Perch	21		10 (9-11)		0.1	0.0	0.0	0.0	0.2	0
Värmland	Årjäng (Furskogtjärnet)	1993	Perch	4		28 (24-32)	321 (230-430)	0.7	0.2	0.6	0.5	0.9	75
Värmland	Arvika	1998	Perch	131		27 (18-37)	266 (80-1070)	0.5	0.3	0.5	0.2	1.5	33
Värmland	Hagfors	1995	Perch	5				0.1	0.0	0.1	0.1	0.2	0
Värmland	Säffle, Hammarö	1996	Perch	18		17 (15-20)	63 (31-108)	0.1	0.1	0.1	0.1	0.3	0
Värmland	Hammarö	1997	Perch	7		20 (18-21)	68 (47-89)	0.2	0.0	0.1	0.1	0.2	0
Värmland	Säffle, Hammarö	1998	Perch	20		19 (17-21)	80 (51-117)	0.1	0.1	0.1	0.1	0.3	0
Värmland	Eda, Säffle	1999	Perch	13		21 (17-28)	108 (56-260)	0.1	0.1	0.1	0.1	0.3	0
Värmland	Årjäng, Hammarö, Säffle	2000-2003	Perch	119		19 (16-21)	69 (47-100)	0	0	0.2	0.1	0.5	0
Västerbotten (sjö)	Skellefteå (Björnsjön, Stor Löwattnet)	1990	Perch	15		21 (15-24)	217 (60-970)	0.6	0.2	0.5	0.3	1.0	47
Västerbotten (hav)	Umeå	1991, 1995-1998, 2000-2004	Perch	115	3.9 (0.0-7)	18 (16-23)	75 (52-138)	0.1	0.0	0.1	0.0	0.2	0
Västernorrland	Örnsköldsvik	2000, 2002, 2003	Perch	40		18 (15-20)	57 (36-84)	0.2	0.1	0.2	0.0	0.4	0
Västmanland	Skinnskatteberg	1999, 2000, 2002, 2004	Perch	260	1.8 (0-13)	30 (4-127)	13 (0.6-72)	0.1	0.1	0.1	0.0	0.5	0
Västmanland	Skinnskatteberg	2001	Perch	70	2.5 (1-13)	11 (7-20)	16 (3.7-64)	0.2	0.1	0.2	0.1	0.6	1.4
Västmanland	Skinnskatteberg	2003	Perch	76	2.6 (1-8)	12 (6-25)	24 (2.6-177)	0.2	0.2	0.1	0	0.8	5.3
Västra Götaland	Tanum, Lerum, Mellerud, Mariestad, Gullspång	1996, 1998-2004	Perch	478	1.6 (1-6)	30 (4-124)	30 (0.4-115)	0.1	0.1	0.1	0.0	0.4	0
Västra Götaland	Tanum	1997	Perch	10	6 (4-9)	17 (15-18)	50 (41-62)	0.4	0.2	0.4	0.2	0.7	20
Västra Götaland	Mellerud, Mariestad	1997	Perch	19		20 (18-21)	81 (54-104)	0.1	0.0	0.1	0.1	0.1	0

Appendix

County	Municipality	Catch year Sampling date	Fish species	Number	Age (yr)	Length (cm)	Weight (g)	Hg mg/kg wet wt					Minimum limit exceeded by, %
								Mean	SD	Median	Min	Max	
Örebro	Lindesberg, Hällefors, Degerfors, Nora, Askersund, Ljusnarsberg, Kumla, Laxå	1991-1992, 1999-2004	Perch	143	1.5 (0-4)	34 (6-126)	9 (2-28)	0.2	0	0.2	0	0.3	0
Örebro	Karlskoga	1997	Perch	5		36 (32-43)	686 (390-1180)	0.4	0.1	0.4	0.3	0.6	20
Örebro	Kumla	1997	Perch	3		21 (21-22)	104 (89-129)	0.3	0.1	0.3	0.2	0.3	0
Östergötland	Valdemarsvik	1990-2004	Perch	182	4.6 (3-7)	19 (15-29)	84 (37-278)	0.04	0.02	0.03	0.01	0.1	0

¹Björkerydssjön, Iglasjön, Kiljasjön, Långasjön, Stora Åsjön, Älten

²Tyresö, Norrtälje, Sigtuna, Österåker, Botkyrka, Nynäshamn, Upplands_Bro, Stockholm, Sollentuna, Vallentuna, Södertälje, Värmdö, Nykvarn, Haningen, Salem

1. Mikroprofil Gris – Kartläggning av mikroorganismer på slaktkroppar av M Lindblad.
2. Nyckelhålet för spannmålsprodukter av A Laser Reuterswärd.
3. Interkalibrering av laboratorier. Mikrobiologi – Livsmedel, januari 2006 av C Normark och K Mykkänen.
4. Studie av förstföderskor – Organiska miljögifter hos gravida och ammande. Del 1 Serumnivåer av A Glynn, M Aune, P O Darnerud, S Atuma, S Cnattingius, R Bjerselius, W Becker och Y Lind.
5. Kontroll av rests substanser i levande djur och animaliska livsmedel – Resultat 2005 av I Nordlander, H Green och I Nilsson.
6. Proficiency Testing – Food Chemistry, Nutritional Components of Food, Round N-37, by L Merino and M Åström.
7. Proficiency Testing – Food Chemistry, Trace Elements in Food, Round T-12 by C Åstrand and L Jorhem.
8. Krav på livsmedelsföretagarna – Utbildning i livsmedelshygien.
9. Interkalibrering av laboratorier. Mikrobiologi – Livsmedel, april 2006 av C Normark och K Mykkänen.
10. Interkalibrering av laboratorier. Mikrobiologi – Dricksvatten 2006:1, mars av T Šlapokas och C Gunnarsson.
11. Rapportering om livsmedelstillsyn 2005 – Tillsynsmyndigheternas rapportering om livsmedelstillsyn av D Rosling.
12. Rapportering av dricksvattentillsyn 2005 – Tillsynsmyndigheternas rapportering om dricksvattentillsyn av D Rosling.
13. The Swedish Monitoring of Pesticide Residues in Food of Plant Origin: 2005, EC and National Report by A Andersson, A Jansson and A Hellström.
14. Kontroll av svenska musselodlingar av I Nordlander.
15. Studie av förstföderskor – Organiska miljögifter hos gravida och ammande. Del 2 Bröstmjölksnivåer samt korrelationer mellan serum- och bröstmjölksnivåer av S Lignell, A Glynn, M Aune, P O Darnerud, R Bjerselius och W Becker.
16. Proficiency Testing – Food Chemistry, Nutritional Components of Food, Round N-38 by L Merino and M Åström.
17. Proficiency Testing – Food Chemistry, Vitamins in Foods, Round V-4 by H S Strandler and A Staffas.
18. Förslag till framtidens nyckelhålmärkning i storhushåll – certifieringssystem och nya kriterier av U Bohman och A L Reuterswärd.
19. Fiskkonsumtion – risk och nytta. Risk- och nyttovärdering baserad på innehållet av dioxin/PCB, metylkvicksilver och vissa näringsämnen i fisk.
20. Svenska barns matvanor 2003 – resultat av enkätfrågor av W Becker och H Enghardt Barbieri.
21. Interkalibrering av laboratorier. Mikrobiologi – Dricksvatten 2006:2, september av T Šlapokas, C Gunnarsson och M Foucard.
22. Proficiency Testing – Food Chemistry, Trace Elements in Food, Round T-13 by C Åstrand and L Jorhem.
23. Interkalibrering av laboratorier. Mikrobiologi – Livsmedel, oktober 2006 av C Normark, K Mykkänen, I Tillander och C Gunnarsson.

1. Algtoxiner i avsaltat dricksvatten.
2. Nationellt tillsynsprojekt 2006 om livsmedelsmärkning.
3. Indikatorer för bra matvanor av W Becker.
4. Interkalibrering av laboratorier. Mikrobiologi – Livsmedel, januari 2007 av C Normark och K Mykkänen.
5. Proficiency Testing – Food Chemistry, Nutritional Components of Food, Round N-39 by L Merino and M Åström.
6. Nutrient Analysis of Dairy Foods and Vegetarian Dishes by M Arnemo, S Johansson, L Jorhem, I Mattisson, S Wretling and C Åstrand.
7. Proficiency Testing – Food Chemistry, Trace Elements in Food, Round T-14 by C Åstrand and L Jorhem.
8. Riskprofil – *Yersinia enterocolitica* av S Thisted Lambertz.
9. Riskvärdering av persistenta klorerade och bromerade miljöföroreningar i livsmedel av E Ankarberg, M A, G Concha, P O Darnerud, A Glynn, S Lignell och A Törnkvist.
10. Risk Assessment of Methylmercury in Fish by K Petersson-Grawé, G Concha and E Ankarberg.

