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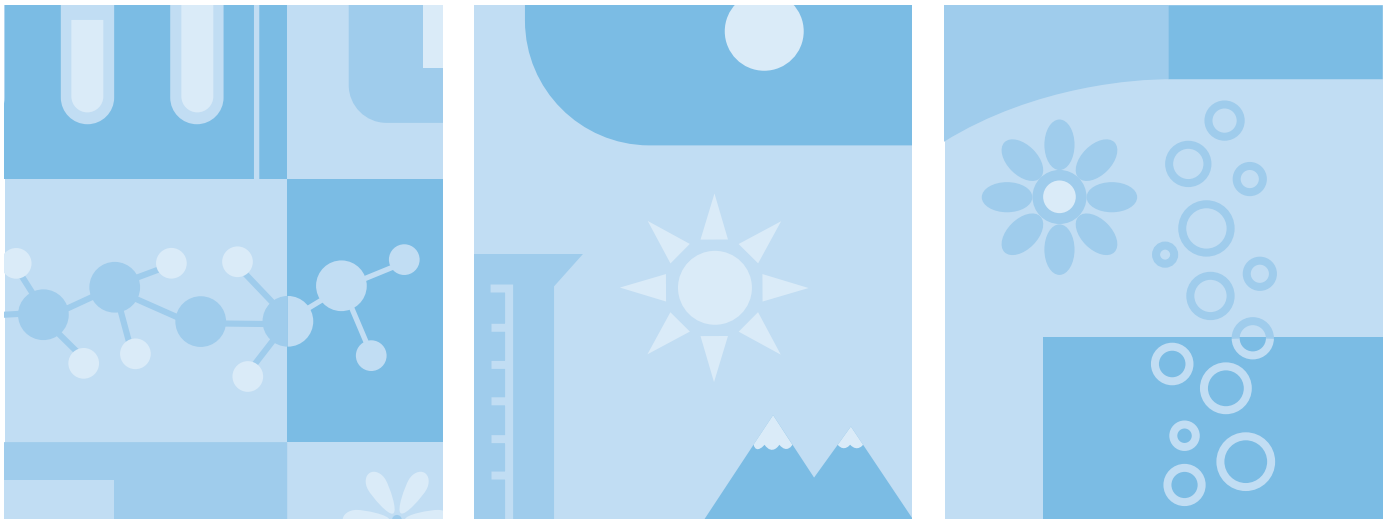

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Preface

The Swedish Chemicals Agency (KemI) has been assigned by the Swedish Government to produce a national action plan for a toxic-free everyday environment: Action plan for a toxic-free everyday environment 2011 – 2014 – protect the children better.

Efforts are now going on in several areas, both in Sweden, within the EU and internationally and often in cooperation with other authorities. Reducing chemical risks in the everyday environment is one step towards attaining the Swedish Parliament´s environment quality objective A Non-Toxic Environment, which is the objective that KemI is responsible for.

Within the framework of the action plan, KemI compiles knowledge in KemI´s report and PM series elaborated by experienced colleagues, researchers or consultants. In this way, KemI presents new and essential knowledge in publications which can be downloaded from the website www.kemikalieinspektionen.se

Chlorinated paraffins are used worldwide in a variety of areas such as plasticisers, additives in cutting oils in the industry, flame retardants and additives in paints. Chlorinated paraffins can thus reach the natural environment through a variety of routes, but also lead to direct exposure of humans. Many chlorinated paraffins are toxic to humans and organisms in the environment, and are persistent and bioaccumulative. The toxicity of chlorinated paraffins is dependent on chain length (short chained are most bioactive) and the degree of chlorination. There are also indications that exposure to chlorinated paraffins in combination with other persistent organic substances provide greater effect on the enzyme and hormone system.

Today we know virtually nothing about the Swedes´, and particularly childrens´, loads of chlorinated paraffins. To learn more about infant exposure to short-and medium-chained chlorinated chlorinated paraffins, the National Food Agency has been commissioned to analyse chlorinated paraffins in Swedish breast milk. The analysis is part of the joint work of the Swedish Chemicals Agency and the National Food Agency to identify sources which contaminate food with dangerous chemical substances.

Content

Abstract

The present CP analyses in pooled Swedish breast milk from 1996-2010 show mean levels for SCCPs and MCCPs of 107 and 14 ng/g fat weight, respectively, although there was a large fluctuation in levels from pools of different years with no obvious time trend. Overall, the levels are of the same magnitude as those earlier found in breast milk from UK and Germany, and the present SCCP/MCCP ratio is similar to that of the UK study. A preliminary assessment of CP intake for the suckling baby in relation to effect levels in experimental studies suggests a reasonable safety margin, but the data base is insufficient. More data on CP levels in food as well as in humans are needed, but this might be dependent on improved methods for CP analysis.

Abstrakt

Den här studien visar att halterna av klorerade paraffiner (CP) i poolade svenska bröstmjölksprover insamlade under1996-2010, ligger på i snitt107 och 14 ng/g fetttvikt för kortkedjiga (SCCP) respektive medelkedjiga (MCCP) klorparaffiner. Det fanns en stor variation i nivåer från olika år, dock utan någon uppenbar tidstrend. Nivåerna är i stort sett av samma storleksordning som de som tidigare uppvisats i bröstmjölk från Storbritannien och Tyskland, och det nuvarande SCCP/MCCP förhållandet liknar det i en brittisk studie. En preliminär bedömning av CP-intaget hos det ammade barnet i förhållande till effektnivåer i experimentella studier tyder på en rimlig säkerhetsmarginal, men databasen är otillräcklig. Fler uppgifter om CP-nivåer i livsmedel och i människor behövs, men detta kan vara beroende av förbättrade metoder för CP analys.

Introduction

Chlorinated paraffins (CPs), or chlorinated alkanes, are a group of synthetic compounds produced by chlorination of straight-chained paraffin fractions. The carbon chain length of commercial CPs varies usually between 10 and 30 carbon atoms, and the chlorine content is usually between 40 and 70 % by weight. CPs are viscous, colorless/yellowish dense oils, except those with long carbon chain with high chorine content, which are solid. There are mainly three different technical CP products, i.e. the short-chain (C_{10}-C_{13}), medium chain (C_{14}-C_{17}) and the long-chain (C_{18}-C_{30}) preparations.

These short chain (SCCP), medium-chain (MCCP) and long-chain (LCCP) CPs all have industrial applications. LCCPs and MCCPs are mainly used as plasticizers in flexible polyvinylchloride (PVC), industrial metalworking fluids for cutting and drilling, and metal stamping in metal manufacturing. They are also used in paints, lathers, textiles and sealing compounds. The SCCPs have similar industrial uses, with focus on metalwork fluids, sealants, flame retardants in rubber and textiles, leather processing, paints and coatings.

The SCCP production in 2007, based on available data from EU, Canada and USA, was between 7 500 and 11 500 tons (Fiedler, 2010). In 2005 two CP producers in EU were noted, in UK (trade name: Cereclor) and in Italy (trade name: Cloparin), and the EU production was estimated to be between 1 500 and 2 500 tons (Fiedler, 2010). However, the main producer today is China. In the 1980s, the CP production increased rapidly due to high demand from the plastic industry, and from that a strong increasing trend in production volumes has led to today's huge production of 600 000 ton (2007). Thus, China is considered the largest CP producer in the world, and there are more than 140 CP factories in China (Yuan et al., 2009; Wang et al., 2009; in Fiedler, 2010).

As a result of the wide industrial applications, CPs have been found as contaminants in the environment. For example, CPs may be released from improperly disposed metal-working fluids, from polymers containing CPs, and by leaching from paints and coatings. Levels of CPs have been analysed in water, sediments and biota. Within biota, fish and aquatic food webs offer the largest environmental dataset for SCCPs, and to a much lesser extent, for MCCPs/LCCPs (Muir, 2010). CPs have been found also in very remote places, e.g. in fish from a lake at Björnöya, in the Arctic. These measured levels (SCCP-MCCP 11-27 and 13-43 ng/g ww in liver, respectively) were however considerably lower than those found for PCBs in the same samples. In Sweden, CPs have been identified in various biotopes. In fish, but also in terrestrial species as elk and hare, measurable CP levels were found (Jansson et al., 1993).

Regarding environmental toxicity of CPs aquatic invertebrates seem to be the most sensitive species; mortality and growth-inhibition is seen at rather low concentrations (Thompson and Madeley, 1983a,b). In mammals, the target organs of toxicity include the liver, kidney, and the thyroid and parathyroid glands, and effects on these organs have been reported for SCCPs at 100 mg/kg bw/day (58% Cl, 14 day, oral gavage) and for MCCP at 25 mg/kg bw/day (52% Cl, 90 days dietary exposure) (El-Sayed Ali and Legler, 2010). However, the most sensitive endpoint for mammalian toxicity seems to be developmental toxicity, with a LOEL (lowest effect level) in rats of 5.7 mg/kg bw/day (Serrone et al., 1987). In this rat study a MCCP mix (52% Cl) was given in the diet 28 days before mating, during mating, and dosing of the females was continued until pups were 21 days old. No effects were seen in the parental generation, but offspring showed adverse effects on body weight and condition, and

haematological parameters. At higher doses, pup survival was also affected. An NTP 2-year study on rats and mice showed increase in tumor development (Bucher et al., 1987), and based on these studies IARC concludes that SCCPs (average chain length 12 and chlorination 60%) are possible carcinogens to humans (IARC 1990).

The presence and persistence of CPs in the environment and the mentioned adverse effects in animal models has prompted regulatory authorities and environmental organizations to decrease and regulate the industrial use of CPs. As an example, the Stockholm Convention has proposed SCCPs as a new POP for the Stockholm Convention list of unwanted chemicals (now 21 POPs) (Stockholm Convention, 2012).

There are few studies on levels of CPs in human samples, and this includes also breast milk data. To our knowledge there are only two studies have reported results for CPs in breast milk. In the UK study, where milk samples were collected from 18 individuals from London and Lancaster in 2001-2002, the median SCCP level was 180 ng/g fat whereas the median MCCP levels was 21 ng/g fat (Thomas et al., 2006). The German study (a short paper) presents results on breast milk samples from six individuals, collected in 2004-2005 (Reth et al., 2005). The mean CP content in milk, as a sum of SCCPs and MCCPs, was 120 ng/g fat.

An important cause behind the general lack of CP data (e.g. levels in environment, food, human samples) could be found in problems concerning CP analysis. In spite of improvements, still today the analysis of CPs in biota is challenging. Very few data have therefore been produced on levels of CPs in humans, illustrated by the very few CP studies presenting breast milk data. As we have learned that effects on offspring are shown after exposure to low levels of MCCPs, it is important to obtain data on the breast milk levels to be able to estimate the CP intake for the little child. In this report, we present for the first time data on CP levels in Swedish pooled breast milk, collected in Uppsala County in 1996-2010.

Materials and methods

Study population and breast milk sampling

From early fall 1996 to May 1999, randomly selected primipara women (18-41 years) from Uppsala County were asked to participate in a study of body burdens of persistent organic pollutants (POPs) (Glynn et al., 2007), and breast milk and serum was obtained from a total of over 200 women. From year 2000 breast milk sampling continued on the same population by sampling of ca 30 breast milk samples randomly every second year, and from 2007 and forwards the sampling was extended to take place every year. The mothers sampled milk while breast feeding their infants during the third week after delivery, using a manual breast pump and/or a passive breast milk sampler. The women were instructed to sample milk both in the beginning and in the end of the breast-feeding sessions. The goal was to sample 500 ml from each mother during the seven days of sampling. The breast milk was kept frozen (-20^{o}C) in acetone-washed glass bottles and the newly sampled milk was poured on top of the frozen milk. Aliquots of individual milk samples were subsequently mixed together to form annual pools. Informed consent was obtained from all participating women. The study was approved by the Ethics Committee of the Medical Faculty at Uppsala University.

Extraction and clean-up

Approximately 30 mL of milk was centrifuged at 3000 rpm for about 10 min. The creamy lipid fraction on top was separated and homogenized with 50 g of Na_2SO_4. ^{13}C labeled chlordane was added as an internal standard, and then the homogenate was cold column extracted with 150 mL of cyclohexane/ethyl acetate 1:1. The extract was concentrated and treated with sulfuric acid before a fractionation on activated silica. A reduction in sample volume and addition of 1,2,3,4 tetrachloro-naphthalene (TCN) as a recovery standard was done prior to analysis on HRGC/HRMS.

The fat content in individual milk samples was determined gravimetrically (Aune et al., 1999), and the calculated mean fat levels of each analysed milk pool were used in estimation of CP levels on fat weight basis.

GC/MS analysis

An HP6890 GC equipped with a column (15 m Restek Rxi 5 ms, 0.25 mm i.d., 0.25 mm filmthickness) and coupled to a VG AutoSpec, high resolution mass spectrometer was used for all of the analyses. The GC was operated in constant flow mode, 1 mL/min (He), with a temperature program starting at 90°C, then ramping to 245°C by 20°C/min, then to 300°C by 50°C/min, holding that temperature for 3 min. The injector temperature was 260°C. The MS was operated in ECNI mode with methane at a pressure of $2´ 10^{-5}$ mbar as reagent gas. To be able to monitor the $[M\text{-}Cl]^-$ ions for each formula group of interest (Table 1), the MS experiment was divided into five time windows. The quantifications were performed according to an earlier described method (Tomy et al., 1997; Zencak et al., 2005).

The extraction, clean-up and analysis were done at NILU whereas the fat determination was performed at NFA.

Statistics

Correlation between SCCP and MCCP values were tested according to Pearson.

Results

Fat-adjusted levels of SCCP and MCCP in annual pools (years 1996-2010) of Swedish breast milk are presented in Table 2. In the same table, the number of individual sample forming each pool and the fat content of the milk pools is presented. There is an obvious difference in levels between SCCP and MCCP, with a mean ratio between the two product groups of 7.9. The fat content of the milk pools lie between 3 and 3.5 % (mean 3.1 % for the whole period), a narrow distribution explained by the fact that the analysed milk represent pooled samples. Moreover, the breast milk pool from 1998 was subjected to duplicate analyses, and the results are given in the Table (they differed by a factor of about three).

In Figure 1, the SCCP and MCCP levels in breast milk are illustrated on an annual time scale, in order to look for possible time trends. The CP analyses show a notable difference in levels between years, but no trend is obvious.

The statistical evaluation of correlation between SCCP and MCCP levels showed no significant correlation (P-value 0.41).

Discussion

For the first time, to our knowledge, measurable levels of CPs in Swedish breast milk are presented. The Swedish milk data could be compared to those from UK and Germany. In the UK study (Thomas et al., 2006), the median levels were 180 (range 49-820) ng/g fat and 21 (range 6.2-320) ng/g fat, for SCCPs and MCCPs, respectively (to be compared with Swedish means of 107 and 14, respectively). The ratio between SCCP and MCCP in the Swedish milk (factor 7.9) is also similar to the ratio found in the UK milk (factor 8.6). In the German short study (Reth et al., 2005), CP levels were presented as total CP concentrations for five individuals, with a mean of 120 ng/g and range of 49-275 ng/g. If the Swedish results on SCCP and MCCP levels are added together, the sum CP mean is 121 and the range 47-187 ng/g fat. Thus, the Swedish CP levels in breast milk were similar to the German data and lower but not too different from the other UK study, and together these the three studied support each other and give us an idea about a "European" level of CPs in breast milk. However, the lack of CP analyses in general, and that of human samples specifically, hampers a conclusion about possible regional differences in CPs levels in humans in Europe, and of CP´s potential health effects.

The levels of CPs in breast milk show no obvious trend over time, but one could possibly notice the low/not quantifiable levels of MCCPs during 1999 to 2002. Comparing human levels (breast milk) and the Swedish industrial CP usage, the Swedish Chemicals Agency reports that the Swedish industrial use of CPs (all chain lengths) has decreased from 5 000 tons/yr to 400 tons/yr during the 90s, while the use after that has been at a rather steady state (a little increase around 2005). From 1994 a 80% reduction of CPs (all product groups) has taken place (SCA, 2012). Of the CPs used during that time, approximately less than 10% was SCCPs, and the rest was both MC and LC preparations. However, in an overview of the global CP production (Fiedler, 2010) the European production 1 500 – 2 500 tone/year (2007) seems to be decreasing, and is modest if you put it in comparison to the Chinese huge and rapidly increasing production. The Swedish CP levels in milk and lack of a visible trend during our study period (1996-2010) may therefore be a consequence of the presence of CPs in imported products rather than a mirror of the decreasing Swedish industrial use of CPs. However, we also have to remember that the CP analyses are difficult to perform and that the presented levels have to be considered as very approximate, as shown by the analyses of the duplicate sample.

As mentioned above, the ratio of SCCP/MCCP in breast milk was about 8-9. At the same time, figures of CP production and use show that higher chain lengths (MC and LC) predominate. The reason to this lack of correspondence between production and biological levels could be found in the fact that the bioavailability decreases with both chain length and degree of chlorination (Tomy et al., 1998; fish - Bengtsson 1979; mice – Darnerud et al., 1982). Also, if Swedish levels are influenced by CPs in imported goods from e.g. Asia, the relation between different CP preparations in these products is not known.

Oral uptake via food is in many cases the major route of intake for POPs, and this may also be true for the CPs. In experimental studies, the oral uptake of CPs, at least up to medium chain length and intermediate chlorination, was effective (Darnerud et al. 1982, Yang et al., 1987). We also know that CPs have been found in fish from Swedish waters (Jansson et al., 1993),

making it probable that fish consumption could be one route of CP intake. We have however very little information on CP levels in food, apart from the Japanese study by Iino et al. (2005) in which a number of food items were analysed for SCCPs. The analyses show that fish and shellfish, but also meat products and in particular fats and oils (both of animal and vegetable origin) contain the highest levels of SCCPs (fats 140 ng/g). In the Japanese study, the median intake was estimated to 100-400 ng/kg bw/day, depending on age (about 6 microgram/day for adult person). In addition to food intake, there are recent publications showing that CPs are found in indoor air and dust, both in Sweden (Fridén et al., 2011), and in France (Bonvallot et al., 2010). In the Swedish study, the intake of CPs from indoor environment was estimated to around 1 microgram per day, both for adults and toddlers.

In the present study, we observe a mean sum CP level in breast milk of 121 ng/g (SCCP: 107; MCCP: 14) and a maximum sum CP level of 187 ng/g (SCCP:157; MCCP:30). If we assume that the breast-feeding baby consumes 0.7 kg milk per day and the milk fat content is 3.1 % (i.e. the mean level of the present study) the daily mean and maximum intake of sum CPs would be about 2 600 and 4 100 ng, respectively. If the weight of the baby is 5 kg, these body weight-adjusted CP intakes would be 520 ng/kg bw/day (mean) or 820 ng/kg bw/day (maximum). These intake levels could be compared to the levels in the Japanese intake study by Iino et al. (2005), in which the estimated median intake for a 1-year old child was about 350 ng/kg bw/day, but with the difference that the estimated intake for the Japanese child was based on levels of CPs in food other than breast milk. As mentioned above, the intake of CPs from other sources than food should be noted, examplified by the Swedish study on CPs in air and dust (Fridén et al., 2011).

As already stated in the Introduction, effects of CPs on experimental mammals (mainly mice and rats) are generally seen at about 100 mg/kg bw for SCCP and at 25 mg/kg bw for MCCP. However, the most sensitive effects seems to be reproductive toxicity, offspring to MCCP-treated rats showed adverse effects on body weight and condition, and on haematological parameters already at a parental dose of 5.7 mg/kg bw in male pups (=LOEL). Based on this lowest effect level, a NOEL of ca 1 mg/kg bw could be assumed. As a worst case scenario we could propose that the sum CP intake via breast milk for the little child is 820 ng/kg bw/day (see above), and that the effects shown for MCCP in the reproductive study in rats are valid for all CPs. Thus, the safety margin between the estimated (worst case) CP exposure in children in this study and NOEL level in rats is a factor around 1 000, which may be regarded as satisfactory. However, a Canadian study reported a TDI for CPs of only 6 microgram/kg bw/day (Gov. Can. 1993; in El-Sayed Ali and Legler, 2010) which is less 10 times above the estimated exposure (although we have no information on the basis for this TDI). Moreover, we know that an additional exposure from indoor dust and air could be significant, but quantitative figures are hard to estimate. The presented risk assessment will most probably be improved when we have access to more information, both regarding effects of CPs and the exposure to specified population groups.

Lastly, chemical analysis of CPs is a difficult and time-consuming activity, and few laboratories have the skill and techniques for performing these studies. During data analysis, the chemist has to do several manual operations that take experience to learn. This fact is a major reason to the relatively few studies on CPs that could be retrieved from open literature. Also, the presented CP data could be regarded as approximations around a “real” value, and therefore the distribution of values could be wide as a consequence of these analytical difficulties. In our study, the duplicate samples from the 1998 milk pool showed a difference in levels by factor three between them (see Table 2). These diverging data of course stress the importance of continuing the development of CP analysis methods in biological samples. In

spite of the diverging results of the duplicate samples, the presented CP data are still of the same approximate magnitude as the German and UK data, and the present Swedish data and the UK data also support each other regarding SCCP/MCCP ratio. The present data on CPs in breast milk are unique by being the first Swedish study to confirm the presence of this compound group in a non-occupationally exposed population, and give important information regarding potential health implications. The present study strengthens and increases the data base on CPs in human samples on a European, as well as global, level. Finally, the presented data give reason to consider future monitoring of human as well as food and indoor dust/air levels of CPs on a regular basis, and to increase the effort to improve the methods for CP analysis.

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Table 1. *Calculated m/z values of $(M\text{-}Cl)^-$ ions monitored for SCCP and MCCP isomers*

SCCP	
	m/z $[M\text{-}Cl]^-$
$C_{10}H_{17}Cl_5$	277.0084
$C_{10}H_{16}Cl_6$	314.9636
$C_{10}H_{15}Cl_7$	346.9275
$C_{10}H_{14}Cl_8$	380.8886
$C_{10}H_{13}Cl_9$	416.8467
$C_{10}H_{12}Cl_{10}$	450.8077
$C_{11}H_{19}Cl_5$	291.0241
$C_{11}H_{18}Cl_6$	326.9822
$C_{11}H_{17}Cl_7$	360.9432
$C_{11}H_{16}Cl_8$	394.9042
$C_{11}H_{15}Cl_9$	430.8623
$C_{11}H_{14}Cl_{10}$	464.8233
$C_{12}H_{20}Cl_6$	340.9978
$C_{12}H_{19}Cl_7$	374.9588
$C_{12}H_{18}Cl_8$	408.9199
$C_{12}H_{17}Cl_9$	444.8779
$C_{12}H_{16}Cl_{10}$	478.8390
$C_{13}H_{21}Cl_7$	388.9745
$C_{13}H_{20}Cl_8$	422.9355
$C_{13}H_{19}Cl_9$	458.8936

MCCP	
	m/z $[M\text{-}Cl]^-$
$C_{14}H_{24}Cl_6$	369.0241
$C_{14}H_{23}Cl_7$	402.9901
$C_{14}H_{22}Cl_8$	436.9512
$C_{14}H_{21}Cl_9$	472.9092
$C_{14}H_{20}Cl_{10}$	506.8703
$C_{15}H_{27}Cl_5$	349.0837
$C_{15}H_{26}Cl_6$	383.0448
$C_{15}H_{25}Cl_7$	417.0058
$C_{15}H_{24}Cl_8$	450.9668
$C_{15}H_{23}Cl_9$	486.9249
$C_{15}H_{22}Cl_{10}$	520.8859
$C_{16}H_{29}Cl_5$	363.0994
$C_{16}H_{28}Cl_6$	397.0604
$C_{16}H_{27}Cl_7$	431.0214
$C_{16}H_{26}Cl_8$	464.9825
$C_{16}H_{25}Cl_9$	500.9405
$C_{16}H_{24}Cl_{10}$	534.9016
$C_{17}H_{29}Cl_7$	445.0371

Table 2. *Levels of SCCPs and MCCPs in pooled breast milk from Uppsala County, sampled 1996-2010*

Sample no.	**Sampling year**	**Fat % (mean)**	**No. of particip. women**	**SCCP (ng/g fat)**[2]	**MCCP (ng/g fat)**[2]
1	1996	3.3	25	120	20
2	1997	3.5	67	112	21
3a[1]	1998	3.3	85	143	7.0
3b[1]	"	"	"	45	2.2
4	1999	3.4	20	120	<1.1
5	2000	3.2	30	115	3.3
6	2002	2.9	31	101	<1.4
7	2004	2.9	31	132	14
8	2006	2.9	29	83	15
9	2007	(3.1)[3]	29	58	17
10	2008	2.9	31	112	26
11	2009	2.9	30	157	30
12	2010	3.0	30	98	18
Mean		3.1		107	14[4]

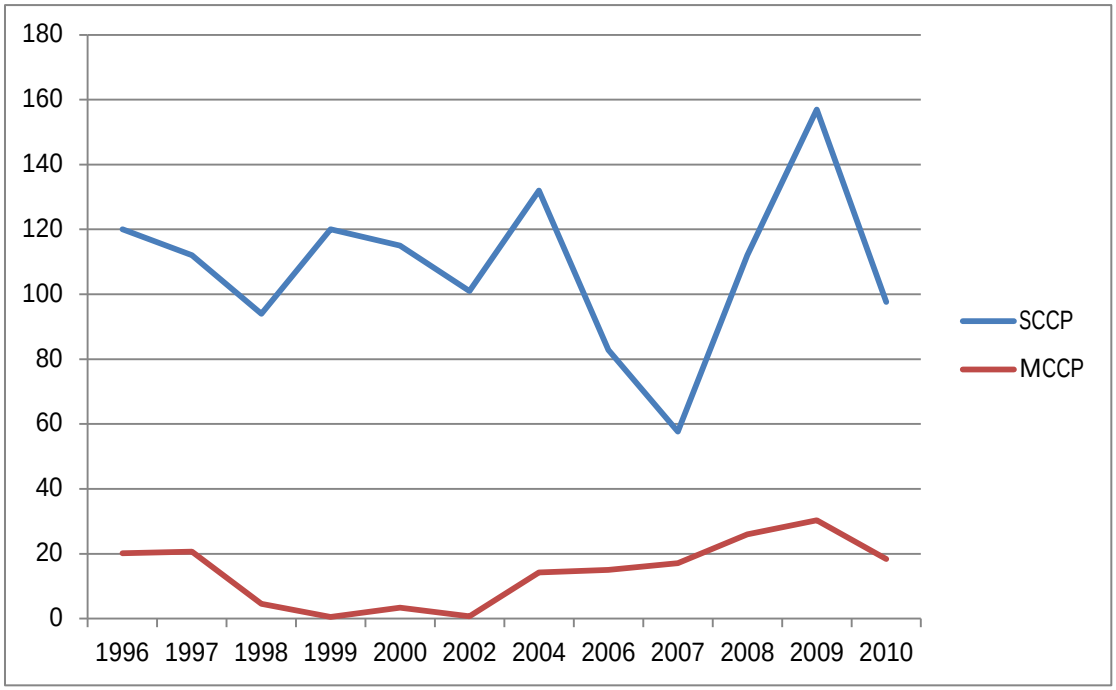
[1] Duplicate samples taken from the same milk pool

[2] The CP levels are expressed based on milk fat weight, by use of the mean fat content of the respective pools

[3] Fat weight data for the individual samples of 2007 years pool are missing; the mean of all other milk pools in this study was used as a surrogate

[4] The mean value for MCCP was calculated by using ½LOQ values when needed (1999 and 2002 milk pools)

Figure 1. *The SCCP and MCCP levels in annual Swedish breast milk pools (in ng /g fat) from the years 1996 to 2010. 1998 level represents mean of duplicate samples. Further details to the figure are given in Table 2*



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