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Influence of Short-Term Dietary Measures on Dioxin Concentrations in Human Milk

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Breast-feeding may expose infants to high levels of toxic chlorinated dioxins. To diminish intake of these lipophilic compounds by the baby, two diets were tested for their ability to reduce concentrations of dioxins in human milk. The diets were a low-fat/high-carbohydrate/low-dioxin diet (about 20% of energy intake derived from fat) and a high-fat/low-carbohydrate/low-dioxin diet. These diets were tested in 16 and 18 breast-feeding women, respectively. The test diets were followed for 5 consecutive days in the fourth week after delivery. Milk was sampled before and at the end of the dietary regimen, and dioxin concentrations and fatty acid concentrations were determined. Despite significant influences of these diets on the fatty acid profiles, no significant influence on the dioxin concentrations in breast milk could be found. We conclude that short-term dietary measures will not reduce dioxin concentrations in human milk. *Key words*: dioxins, dibenzofurans, dietary measures, human milk, xenobiotics. *Environ Health Perspect* 102:968–971 (1994)

Chlorinated dioxins and dibenzofurans (henceforth referred to as "dioxins") are highly toxic tricyclic aromatic compounds, whose concentrations in the environment have increased greatly since 1940 (*1*). They are derived from many sources; for example, commercial and technical products like 2,4,5-trichlorophenol and pentachlorophenol, incineration of municipal and hazardous waste, industrial processes, and combustion of leaded gasoline in car engines (*2,3*).

Accumulation in animals takes place readily, whereas uptake in plants is low (*4*). For humans the main source of dioxins is animal fat, especially meat, dairy products, and fish (*5–9*). Pork is relatively low in dioxins (*5,7*); concentrations in oils and fats of vegetable origin are even lower (*5*). Due to their high lipophilicity, dioxins accumulate in human body fat (*10–12*), reaching concentrations far above those in animal fat (*5,7*). These high concentrations are due to the long half-life of dioxins [in humans about 5–8 years (*10,13*)] and the fact that humans are at the top of the food chain and live longer than most species.

In 1984, high dioxin concentrations were detected in the breast milk of women living in the northern part of Sweden and in Western Germany (*14*). Since then, dioxins have been detected in human milk in many other countries (*15*). By breast-feeding, infants may be exposed to high levels of dioxins during a short period. Effects of dioxins on thyroid hormone levels of newborns have already been reported (*16*).

Human milk contains about 2.5–4.5% fat, mainly in the form of triglycerides (*17*). Fatty acids in these lipids are derived from three sources: *de novo* mammary synthesis, dietary lipids, and mobilized adipose or hepatic lipids (*18*). To some extent, the diet influences the composition of fat in breast milk (*19–21*). In women in western countries, the contribution of *de novo* mammary synthesis in humans is low, about 10–20% of the total (*22,23*). It can be enhanced by use of a normo- or hypercaloric low-fat/high-carbohydrate diet as generally consumed in nonindustrialized countries (*19–21*), resulting in an increase of medium-chain fatty acids (MCFAs), in particular dodecanoic acid and tetradecanoic acid, in the milk. The contribution of fatty acids derived from dietary lipids, usually about 30% (*22*), can be enhanced by consuming a high-fat diet (*20*). With normal diets, the majority of fatty acids will be derived from mobilized hepatic or adipose lipids (*23*).

We hypothesized that mobilization of fatty acids from adipose tissue causes release of stored dioxins, which will then be excreted in the breast milk. Consequently, two diets were developed and each diet was tested for its ability to reduce dioxin concentrations in milk by reducing the mobilization of fatty acids from adipose tissue.

Methods

One diet tested was low fat/high carbohydrate/low dioxin. By reducing fat intake to about 20% while offering large amounts of carbohydrates, we tried to stimulate the *de novo* mammary synthesis of fatty acids from glucose. The other diet tested was high fat/low carbohydrate/low dioxin. Fat intake was increased up to about 50%. By offering large amounts of oils and fats of vegetable origin, we tried to promote the excretion of "clean" dietary lipids into the milk. The only meat product allowed was

pork, which has a relatively low content of dioxins (*5,7*). A short description of both test diets is given in Table 1.

To investigate whether the dietary changes were sufficient to change the milk-fat composition, we analyzed the fatty acid pattern of the milk. A low-fat/high-carbohydrate diet will increase the percentage of MCFAs and decrease the percentage $C_{18}:1\omega9$ (= fatty acid with 18 straight-chain carbon atoms, 1-methylene-interrupted double bond and 9 carbon atoms from the terminal methyl group to the nearest double bond) in milk fat (*19–21*). A high-fat/low-carbohydrate diet will decrease the percentage of MCFAs and increase the percentage of $C_{18}:1\omega9$ and long-chain fatty acids in milk fat (*20,21*). The percentage of MCFAs (decanoic acid plus tetradecanoic acid) was calculated as a measure of *de novo* mammary synthesis. Additionally, the percentage of $C_{18}:1\omega9$ fatty acid, derived from the fat stores and from dietary lipids (*21*), was calculated.

In the first 2 weeks after delivery, the fat content of breast milk increases, while it remains quite constant thereafter (*24*). We therefore selected the third and fourth weeks after delivery to minimize possible effects from changes in the fat content of the breast milk.

Thirty-four healthy, well-nourished, totally breast-feeding women entered this study on the basis of informed consent. All were white, between the ages of 23 and 38 years (mean 29.2), and intended to breast-feed for at least 2 months. After delivery, every subject was randomly assigned to one of the diets tested. Group A subjects were assigned to the low-fat/high-carbohydrate/low-dioxin diet; group B subjects were assigned to the high-fat/low-carbohydrate/low-dioxin diet.

In the second week after delivery the women were visited by a dietician, who gave instructions on how to keep the food record and explained the test diet. The food diary recording technique was used. Additionally, a record was kept of the kind of fat (animal or vegetable origin) consumed. The mass of ingredients was estimated by using household measures. The participants received a list of allowed foods. They were encouraged

Table 1. General description of test diets

Food group	Description
Low-fat/high-carbohydrate/low-dioxin diet	
Meat	Lean pork, soya-meat substitutes
Dairy products	Low fat with sugar
Margarine and oils	Minimal use of vegetable oils and low-fat margarine based on vegetable oils
Bread, potatoes	Large amounts
Vegetables, fruits	Large amounts
Beverages	Fruit-juices and sugar-containing drinks
Snack foods	Large amounts of sugar-containing sweets and snacks
High-fat/low-carbohydrate/low-dioxin diet	
Meat	Fatty pork, soya-meat substitutes
Dairy products	Low fat without sugar
Margarine and oils	Large amounts of vegetable oils
Bread, potatoes	As needed
Vegetables, fruits	As needed
Beverages	Sugar-free drinks
Snack-type foods	Large amounts of nuts

to contact the dietician in case of doubt.

In the third week after delivery all women kept a food record of their normal diet for 7 days. On the last day of that week they collected their breast milk (see below). For the next 5 days, during the fourth week, they used the assigned test diet. They could choose their own menus from the articles on the list of allowed foods. During this test diet they continued keeping a food record. On the last day of the test period they collected breast milk again. The breast used, the time of the day, and the method were the same as those used at the end of the third week. During both weeks, daily weights were taken to check for weight loss. At the end of the study the food records were evaluated by the dietician. The calculations were performed using a computer program based on a Dutch nutrient database (Netherlands Nutrition Data Bank tables); TNO voeding (Nutrition and Food Research, Zeist, the Netherlands); and VOVO (Netherlands Bureau for Food and Nutrition Education Nutrition Center, the Hague, the Netherlands).

To estimate the dietary dioxin intake per day, we used data from a Dutch study concerning dioxin concentrations in individual food products (5). The results from this study are considered to be representative for all food products in the Netherlands. Dioxin concentrations in dioxin-rich fat (defined as all animal fats except pork) and pork were assumed to be 1.7 and 0.43 ng toxic equivalents/kg (ng TEQ/kg), respectively. The following formula was used:

$$\text{Daily dioxin intake (ng TEQ)} =$$
$$(\text{DR fat} \times 1.7) + (\text{pork fat} \times 0.43)$$

where DR fat = intake of dioxin-rich fat per day (kg) and pork fat = intake of pork fat per day (kg).

Concentrations of the 17 most toxic congeners (7 dioxins and 10 dibenzofurans) in milk fat were measured. The concentration of each congener was multiplied by its toxicity equivalency factor (TEF), which is the relative toxicity compared to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD), the most toxic congener (25). We calculated the toxicity of a mixture by adding these products and expressed the result in TEQ. In this study chlorinated dioxin and dibenzofuran concentrations ("dioxin" concentrations) are expressed in nanograms TEQ/kilogram milk fat.

While the baby was suckling one breast, the milk sample was taken from the other breast using an electrical breast pump. That breast was emptied as much as possible. The milk sampled was mixed thoroughly. We used 50 ml of milk for analysis and the rest was given to the baby by bottle. From each mother, milk samples collected during one day were pooled. The samples were frozen at -20°C until analyses of dioxins and fatty acids were performed.

To analyze dioxins and total fat, after freeze-drying, the milk samples were soxhlet-extracted with toluene. When the toluene was evaporated, the extracted lipids were determined gravimetrically. Thereafter, a two-step clean-up was done using an activated carbon column (26), followed by two columns filled with $AgNO_3$ on silica gel, and Al_2O_3 respectively. After concentrating the sample, we quantified it using gas chromatography and mass spectrometry (HP 5970 GC, Kratos Concept MS). We used a mixture of ^{13}C-labeled dioxins and dibenzofurans as an internal standard. Fractioning of the medium- and long-chain fatty acids was performed essentially as described by Van der Steege et al. (27).

We used the two-tailed paired Student's *t*-test to determine if the diet tested had a significant influence on total fat, fatty acid composition, and dioxin concentrations in the breast milk. All statistical calculations were performed using the computer program SPSS/PC+ 4.0; *p*-values <0.05 were considered significant.

The study was performed according to a protocol approved by the medical ethical committee of the Academic Medical Centre.

Results

Mean age, weight and Quetelet index (weight/length^2) were the same in both groups (means ± SEM: 29.2 ± 1.1 versus 29.2 ± 0.8 years; 67.7 ± 3.2 versus 70.9 ± 2.9 kg; and 22.7 ± 1.0 versus 22.8 ± 1.1 kg/m^2 for groups A and B, respectively). Descriptive statistics of diet composition of the subjects' usual diets and the test diets of both groups are listed in Tables 2 and 3. In group A, energy intake was significantly decreased during the test diet compared with the usual diet ($p = 0.001$). However, body weights were stable in all subjects during the 2-week period.

Estimated dioxin intake in the low-fat/high-carbohydrate diet was 101 ± 6 and 7 ± 2 pg TEQ/day before and during the

Table 2. Descriptive statistics of mean intake per day of dietary components in group A (low-fat/high-carbohydrate/low-dioxin diet)

	Normal diet		Test diet	
Item (*n* = 16)	Mean	SD	Mean	SD
Total fat (g)	115.6	23.7	46.8	17.6
Animal fat (g)	67.1	18.1	9.5	7.4
Dioxin-rich fat[a] (g)	57.0	14.4	2.3	3.5
Carbohydrates (g)	305.9	52.5	373.3	20.5
Protein (g)	91.9	19.9	79.9	20.4
Alcohol (g)	1.7	3.3	0.1	1.5
Energy intake (kJ)	11,101	1,902	9,446	2,066
Fat (%)[b]	39.2	3.9	18.4	4.8
Carbohydrates (%)[b]	46.5	3.8	67.2	5.8
Protein (%)[b]	13.9	1.6	14.4	3.3
Alcohol (%)[b]	0.4	0.8	0.0	0.5

[a]Dioxin-rich fat = animal fat except pork.
[b]Percent contribution to total energy intake.

Table 3. Descriptive statistics of mean intake per day of dietary components in group B (high-fat/low-carbohydrate/low-dioxin diet)

Item (*n* = 18)	Normal diet		Test diet	
	Mean	SD	Mean	SD
Total fat (g)	105.1	16.0	142.3	44.6
Animal fat (g)	58.7	16.2	31.5	14.0
Dioxin-rich fat[a] (g)	46.9	13.0	2.9	2.6
Carbohydrates (g)	280.1	51.8	206.0	48.4
Protein (g)	83.3	13.7	93.8	22.9
Alcohol (g)	5.5	10.4	2.4	6.7
Energy intake (kJ)	10,238	1,385	10,521	2,586
Fat (%)[b]	38.9	4.4	50.8	5.6
Carbohydrates (%)[b]	45.8	4.8	33.4	5.3
Protein (%)[b]	13.7	1.3	15.0	1.8
Alcohol (%)[b]	1.6	2.7	0.7	1.9

[a]Dioxin-rich fat = animal fat except pork.
[b]Percent contribution to total energy intake.

test diet, respectively (mean ± SEM). In the high-fat/low-carbohydrate diet, dioxin intake was 85 ± 5 and 17 ± 2 pg TEQ/day before and during the test diet, respectively. These differences in dioxin intake before and during both test diets were highly significant ($p<0.001$).

Fat and dioxin content of the breast milk before and during the test diets are listed in Tables 4 and 5. In both groups the fat content of the milk did not decrease during the test diet. Neither did we find a significant change in dioxin concentration in milk fat after use of low-dioxin test diets.

In contrast to total fat content, the percentage MCFAs (dodecanoic acid plus tetradecanoic acid) changed significantly in both dietary groups after using the diet. In the low-fat/high-carbohydrate diet group, the percentage MCFAs increased from 11.9 ± 0.5% before to 15.8 ± 1.6% after using the diet (mean ± SEM, p = 0.042). Conversely, the percentage C_{18}:1ω9 fatty acids decreased from 32.7 ± 0.8 to 29.6 ± 1.1% (p = 0.004). In the high-fat/low-car-

bohydrate diet the percentage MCFAs decreased from 12.3 ± 0.7% before to 9.2 ± 0.7% after the diet (p = 0.001), while the percentage C_{18}:1ω9 fatty acid increased from 31.2 ± 0.7 to 33.2 ± 0.9% (p = 0.046).

Discussion

As expected, changes in diet led to a significant change in the fatty acid composition of breast milk, but not to changes in total fat content. This agrees with results of other studies (*19,20,28,29*). Dietary dioxin intake was significantly reduced by both test diets. However, these dietary measures did not have a significant influence on dioxin concentrations in milk fat.

When the only change in diet is the complete elimination of dioxins from the diet, without changes in carbohydrate and lipid content, the maximum effect of this change on dioxin concentrations in milk fat can be estimated by the following calculation. The mean intake of dioxins, using a normal diet, was about 92.5 pg TEQ/day. If daily breast milk secretion is

assumed to be 0.7 l, with a mean fat content of 34.5 g/l (Table 4) and with a mean dioxin concentration of 27.1 ng TEQ/kg milk fat (Table 5), daily excretion of dioxins in the breast milk is 0.7 × 34.5 × 27.1 = 653 pg TEQ. Thus, daily (dietary) intake is only 14% of the daily excretion. This means that 86% of the dioxins in breast milk are derived from the adipose tissue and that the excretion in the milk would only be reduced by 14%.

In our study, however, the carbohydrate and fat content of the diet was also changed, which resulted in significant changes in the fatty acid composition of the milk. These changes reflect the increased *de novo* mammary synthesis [high-carbohydrate diet (*21*)] or an enhanced uptake of dietary fatty acids [high-fat diet (*20*)]. As a consequence, the mobilization of fatty acids and the subsequent release of dioxins from the adipose tissue will be reduced. Our findings that dietary measures do influence fatty acid composition of breast milk but do not influence dioxin concentrations in milk fat, might be explained by the rapid transfer of dioxins between various lipid compartments in the body (*31*). Depending on the rate of diffusion, dioxin concentration in lipids, formed *de novo* in the mammary gland, may rise to levels existing in other lipid compartments. This equilibrium may take place during the transfer of dietary lipids to the mammary gland and during the period the milk is stored in the mammary gland. This would indicate that the concentration of dioxins in milk fat may be independent of the source of the fatty acids.

Other less likely explanations could be the relatively short test period and the possibility that dietary changes in fat or carbohydrate intake were not large enough. However, Insull et al. (*20*) found that dietary changes had the maximal dietary effect on milk fat composition after 2 days. In our study, both the percentage MCFAs and C_{18}:1ω9 in the breast milk changed significantly after using the diet for 5 consecutive days in both groups. This indicates that the dietary changes and the duration of the test period were sufficient to influence the ratio in which the fatty acids are derived from the different sources. In case of the low-fat/high-carbohydrate diet, the expected decrease in dioxin concentration due to the increased *de novo* synthesis might be antagonized by an increased mobilization of fatty acids from the adipose tissue as a consequence of a negative energy balance during the test diet as compared to the normal diet. However, this was not reflected in the weight of the subjects.

In conclusion, it appears to be difficult to reduce dioxin concentrations in milk fat using short-term dietary measures. The

Table 4. Fat concentration in breast milk during the normal diet and during the test diet (g/l)

Group[a]	n	Normal diet		Test diet		
		Mean	SEM	Mean	SEM	p^b
A	16	34.4	2.3	31.9	2.2	0.29
B	18	34.6	2.1	30.4	2.7	0.10

[a]Group A test diet was low fat/high carbohydrate/low dioxin; group B test diet was high fat/low carbohydrate/low dioxin.
[b]Two-tailed paired Student's *t*-test.

Table 5. Dioxin concentrations in breast milk during the normal diet and during the test diet (ng TEQ/kg milk fat)

Group[a]	n	Normal diet		Test diet		
		Mean	SEM	Mean	SEM	p^b
A	16	30.2	2.4	30.0	2.5	0.91
B	18	24.4	2.1	24.0	2.1	0.73

[a]Group A test diet was low fat/high carbohydrate/low dioxin; group B test diet was high fat/low carbohydrate/low dioxin.
[b]Two-tailed paired Student's *t*-test.

best method to maintain low dioxin concentrations in adipose tissue, and thereby in milk fat, will probably be prolonged consumption (years) of foods with a low dioxin content (5,7). This would prevent dioxins from entering and accumulating in the body. Because human intake of dioxins is largely due to the consumption of animal fats, more attention must be paid to reducing the exposure of animals to these toxic compounds.

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