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Trans Fatty Acids in Human Milk in Canada Declined with the Introduction of *Trans* Fat Food Labeling[1]

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Abstract

Trans fatty acids in human milk have raised concerns because of possible adverse effects on infant growth and development. Analyses of human milk in the late 1990s in Canada showed high amounts of *trans* fatty acids from partially hydrogenated oils. Canada introduced labeling of *trans* fatty acids on retail foods in 2003. We analyzed *trans* and *cis* unsaturated and saturated fatty acids in human milk collected from 87 women in 2004–2006 and compared the levels to those in milk collected from 103 women in 1998 and analyzed using similar methods. The total *trans* fatty acids (mean ± SEM, g/100 g fatty acids) in human milk in Canada decreased significantly, from 7.1 ± 0.32 in 1998 to 6.2 ± 0.48, 5.3 ± 0.49, and 4.6 ± 0.32 over 3 consecutive 5-mo periods from November 2004 to January 2006. The milk total *trans* fatty acids were significantly and inversely related to 16:0, 18:2(n-6), 18:3(n-3), 20:4(n-6), 22:4(n-6), and 22:5(n-6) and positively related to 18:0 and conjugated linolenic acids ($P < 0.05$, $n = 190$). The estimated exposures of exclusively breast-fed infants to *trans* fatty acids decreased from a mean and 95th percentile intake of 2.0 and 4.4 g · infant^{-1} · d^{-1} in 1998 to 1.33 and 2.41 g · infant^{-1} · d^{-1}, respectively, in late 2005. The estimated intake of the mothers was 4.0 (range 0.51–12.3) and 2.2 (0.56–7.65) g · person^{-1} · d^{-1} in 1998 and late 2005, respectively. Our studies show *trans* fatty acids have decreased in human milk in Canada, which suggests a concomitant decrease in *trans* fatty acid intake among lactating women and breast-fed infants. J. Nutr. 136: 2558–2561, 2006.

Introduction

Recent interest in the health effects of *trans* fatty acids has centered largely around potential adverse effects of *trans* fatty acids on lipid risk factors for cardiovascular disease, including markers of enhanced inflammatory response (1–5). During growth and development, adverse effects of *trans* fatty acids on the metabolism of the essential (*all-cis*) (n-6) and (n-3) fatty acids are additional concerns (6–8). *Trans* fatty acids are present in the diet from two sources: the industrial partial hydrogenation of fats and oils containing *cis* unsaturated fatty acids, and in the milk and meat of ruminant animals as a result of biohydrogenation of fatty acids in the rumen (9). Estimates of the average daily intake of *trans* fatty acids by adults in the United States, Canada, Europe, and Australia from the 1980s to 2000, based on food usage, food-frequency questionnaires, or duplicate portion analysis, ranged from 3 to 17 g/person (10–15). Whereas *trans* fatty acids can represent as much as 60% of the fatty acids in shortenings, *trans* fatty acids usually represent <5% of dairy and ruminant meat fats (9). Up until 2000, 80–90% of *trans* fatty acids in the diet in Canada and the United States was from partially hydrogenated fats; in the late 1990s about 50% of dietary *trans* fatty acids were consumed in bakery, snack, and fast food products, and about 10% were from table margarines (11,14).

Considerable evidence is available to show that the fatty acid composition of human milk is influenced by the *trans*, (n-6), and (n-3) fatty acid composition of the maternal diet (16–20). In addition, clinical studies that specifically alter *trans* fatty acid intake by lactating women have demonstrated that higher *trans* fat intake results in increased secretion of *trans* fatty acids in human milk (16,17). The fatty acids in human milk are of concern because human milk provides the fatty acids needed for growth and development of the breast-fed infant (20,21). High intake of *trans* fatty acids may have adverse effects during growth and development through inhibition of the desaturation of linoleic acid [18:2(n-6)] and α linolenic acid [18:3(n-3)] to arachidonic acid [ARA[2], 20:4(n-6)] and docosahexaenoic acid [DHA, 22:6(n-3)], respectively, metabolism to unusual fatty acid isomers that are incorporated into membranes, effects on gene expression, or through loss of (n-6) and (n-3) fatty acids from the food supply (6,8,22–27). Previous studies by us and others in Canada and the United States have shown mean total *trans* fatty acid levels in human milk of about 7% total fatty acids, but levels as high as 18% in the milk of some women (18,19,28). In

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[2] Abbreviations used: ARA, arachidonic acid; CLA, conjugated linoleic acid; DHA, docosahexaenoic acid.

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addition, levels of *trans* fatty acids in human milk are generally higher in North America than in Europe (11,18,19,28–31), probably reflecting the lower intake of *trans* fatty acids in Europe than in North America (15).

In 2003, Canada became the first country to introduce food labeling of the *trans* fatty acid content per serving on food labels of packaged foods; this food labeling became mandatory in the retail sector in December 2005 (32). Food industries and manufacturers in Canada responded to the concern over high amounts of *trans* fatty acids on human health, and partially hydrogenated fats have been replaced in many foods such as breads, cookies, crackers, and in frying, with fats and oils containing *cis* unsaturated and saturated fatty acids (33). The objective of this study was to determine whether the introduction of labeling of retail foods' *trans* fat content and removal or reduction of *trans* fatty acids from vegetable oils in many foods has been accompanied by a decline in the *trans* fat concentration of human milk (32,33). To achieve this, we analyzed mature human milk from 87 women giving birth to term infants following the introduction of *trans* fat labeling and compared the results to our previous studies conducted with milk collected from 103 women in 1998 and analyzed using identical methodology (19).

Materials and Methods

Subjects. This study involved women ($n = 87$) who gave birth to full-term (37–41 wk) gestation, single-birth infants between October 2004 and December 2005 and who provided their own milk as the sole source of nutrition to their infants. The women in these studies are part of a larger cohort involving measures of infant development with the same inclusion and exclusion criteria as in our previous studies (19,34). All the women for whom breast milk samples were available were included in this study. These milk samples were compared to samples collected from 103 women in 1998 and analyzed similarly (19). The procedures and methods used in this study were reviewed and approved by the Committee for Ethical Review of Research Involving Human Subjects at the University of British Columbia and the British Columbia's Children's and Women's Hospital Research Coordinating Committee. Informed written consent was obtained from all the women who participated.

Methods. Samples of breast milk (60–100 mL) were collected at 1-mo postpartum during the course of a feeding, at approximately the midpoint of the feeding, and stored at −70°C until analyzed (19). The milk samples were thawed in ice-cold water, directly transmethylated to avoid potential losses of medium-chain saturated fatty acids, and the fatty acid methyl esters separated and quantified by capillary GLC, as in our previous studies (19,34). For the purposes of this report, all fatty acids containing one or more *trans* unsaturated bonds in which all double bonds were methylene interrupted were grouped as *trans* fatty acids. Fatty acids in which one or more of the double bonds were conjugated were grouped and termed conjugated linoleic acids (CLA). As in our previous studies, we did not include specific analytical techniques to confirm the identity of specific *trans* 18:1 isomers, as reported by others (18,28); however, the Δ 9, 10, and 11 isomers, which are the major isomers in partially hydrogenated oils and dairy fats, were all well separated by our GLC methodology (19,34).

Statistical analysis. The data were tested for normality then evaluated by ANOVA and Fisher's LSD test. Potential relations between the levels of total *trans* fatty acids and the saturated, and *cis* monounsaturated and (n-6) and (n-3) fatty acids was examined by using Pearson correlation analysis. A *P*-value ≤0.05 was considered significant. All statistical analyses were performed with the Statistical Package for the Social Sciences (version 7.5; SPSS).

Results

The composition of the major saturated, *cis* (n-9) and (n-7) monounsaturated, (n-6), and (n-3) fatty acids, CLA, and total *trans* unsaturated fatty acids in human milk are shown in **Table 1**. The total *trans* fatty acid level in human milk in western Canada has decreased, such that the level in milk collected between September 2005 and January 2006 was 35% lower than that in milk collected in 1998 ($P < 0.05$). Whereas the mean concentration of total *trans* fatty acids was 7.1 g/100g, with a range of 2.2 to 18.7 g/100g fatty acids in the milk collected in 1998, the mean (range) for milk collected and analyzed in 3 consecutive 5-mo periods from November 2004 to January 2006 was 6.2 (3.4–13.7), 5.3 (3.0–14.5), and 4.6 (2.2–12.2) g/100g milk fatty acids. **Figure 1** illustrates the mean level of *trans* fatty acids, and the 25th–75th percentile range and range of concentrations of *trans* fatty acids in the human samples. The decrease in the mean levels of *trans* fatty acids in human milk (Table 1) is explained by a downward shift in women with high amounts of *trans* fatty acids in their milk, such that the 75th percentile value in the last time period studied approached the mean in milk samples analyzed in 1998 (Figure 1). The level of *cis* monounsaturated or (n-6) or (n-3) polyunsaturated fatty acids did not differ among the sampling times, although the levels of 16:0 were higher and 18:0 and CLA were lower in milk collected between 2004 and 2006 than in samples collected in 1998 (Table 1).

The results of the correlation analyses between the levels of total *trans* fatty acids and the levels of saturated and *cis* unsaturated fatty acids in milk using results for all samples ($n = 96$) are presented in **Table 2**. The levels of total *trans* fatty acids was significantly and inversely related to 16:0, *cis* 18:1(n-9), 18:2(n-6), 20:4(n-6), 22:4(n-6), 22:5(n-6), and 18:3(n-3). Milk total *trans* fatty acids were not associated with 20:5(n-3) ($P = 0.32$), 22:5(n-3) ($P = 0.34$), or 22:6(n-3) ($P = 0.12$) concentrations.

Discussion

Human milk is the sole source of nutrition for the exclusively breast-fed infant and provides all of the fatty acids needed for the

TABLE 1 Total trans fatty acids and major saturated and cis unsaturated fatty acids in human milk collected at different times[1]

	Time of human milk collection			
Fatty acid	1998[2] ($n = 103$)	Nov 04 – Mar 05 ($n = 24$)	Apr 05 – Aug 05 ($n = 24$)	Sept 05 – Jan 06 ($n = 39$)
	g/100 g fatty acid			
trans[3]	7.1 ± 0.32^{c}	6.2 ± 0.48^{bc}	5.3 ± 0.49^{ab}	4.6 ± 0.32^{a}
16:0	19.4 ± 0.28^{a}	19.9 ± 0.53^{ab}	20.7 ± 0.54^{b}	20.5 ± 0.48^{b}
18:0	7.2 ± 0.15^{b}	6.3 ± 0.20^{a}	6.3 ± 0.26^{a}	6.4 ± 0.20^{a}
18:1(n-9)	33.9 ± 0.34	33.4 ± 1.0	33.5 ± 0.87	34.3 ± 0.49
18:2(n-6)	12.1 ± 0.35	12.6 ± 0.62	12.9 ± 0.74	13.1 ± 0.43
20:4(n-6)	0.4 ± 0.01	0.4 ± 0.02	0.4 ± 0.02	0.4 ± 0.01
22:4 (n-6)	0.06 ± 0.01^{a}	0.07 ± 0.01^{ab}	0.09 ± 0.02^{b}	0.07 ± 0.01^{a}
22:5 (n-6)	0.02 ± 0.001	0.02 ± 0.001	0.03 ± 0.01	0.02 ± 0.001
18:3(n-3)	1.4 ± 0.07	1.3 ± 0.11	1.5 ± 0.13	1.4 ± 0.11
20:5(n-3)	0.1 ± 0.01	<0.1 ± 0.02	0.1 ± 0.01	0.1 ± 0.01
22:5(n-3)	0.18 ± 0.02	0.13 ± 0.01	0.14 ± 0.01	0.15 ± 0.01
22:6(n-3)	0.2 ± 0.03	0.2 ± 0.02	0.3 ± 0.03	0.3 ± 0.02
CLA[2]	0.4 ± 0.01^{b}	0.3 ± 0.02^{a}	0.3 ± 0.02^{a}	0.3 ± 0.02^{a}

[1] Values are means ± SEM. Means in a row with superscripts without a common letter differ, $P < 0.05$.
[2] Samples were collected and analyzed in 1998 (19).
[3] Includes all *trans* and *cis-trans* fatty acids, excluding CLA (fatty acids with double bonds are on adjacent carbons).

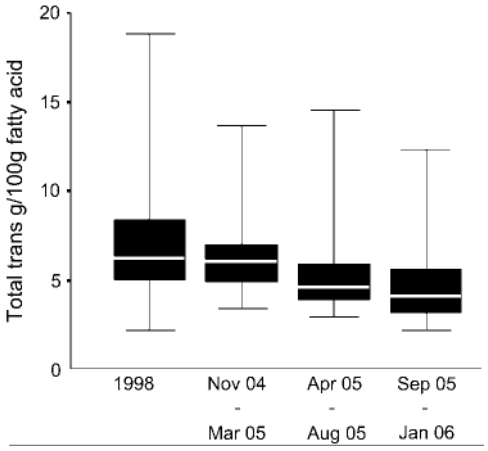

Figure 1 *Trans* fatty acid concentrations in human milk samples collected from women in 1998 ($n = 103$), 11–2004 to 03–2005 ($n = 24$), 04–2005 to 08–2005 ($n = 24$), and 09–2005 to 01–2006 ($n = 39$). The figure illustrates the mean, interquartile range, and range.

infant's growth and development, including fatty acids for synthesis of membrane lipids and adipose tissue (18,19). The presence of high amounts of *trans* fatty acids in human milk has raised concern because of possible adverse effects on growth and development of the recipient breast-fed infant, particularly through interference with the metabolism of the essential (n-6) and (n-3) fatty acids (6,8,17). In addition, high intake of *trans* fatty acids by pregnant and lactating women could have adverse effects on lipoprotein metabolism and inflammatory mediators or (n-6) and (n-3) fatty acid metabolism in the mother, which may also impact transfer of ARA and DHA to the developing fetus and breast-fed infant (1–5,34,35). Previously, we reported a mean of 7.1 g total *trans* fatty acids/100 g in human milk fat in western Canada (19), similar to that recently reported for human milk collected in the United States between 1996 and 2002 (28) and also found by Chen et al. (18) in analyses of human milk collected from different regions of Canada prior to 2000. Using the equation generated by Craig-Schmidt et al. (17) to describe the relation between *trans* 18:1 in the milk and the maternal diet, Chen et al. (18) estimated *trans* fatty acid intakes were 3.9% of total energy, equivalent to 10.1 g/d, for Canadian women with mean total *trans* fatty acid levels of 6.9 g/100 g milk fatty acids. Similarly, applying the equation $Y = 1.49 + 0.42X$, where Y and X represent the percent of *trans* 18:1 in the milk and dietary fat, respectively, and assuming 30% dietary energy from fat in the women's diet (14,18), the estimated mean (range) of intake of *trans* fatty acids by women in the present study was 4.0 (0.51–12.32), 3.4 (1.36–8.72), 2.7 (1.07–9.29), and 2.2 (0.56–7.65) $g \cdot person^{-1} \cdot d^{-1}$ for the women in our studies in 1998 and in the 3 consecutive 5-mo periods from November 2004 to January 2006, respectively.

TABLE 2 Pearson correlation coefficients for relations between trans fatty acids and specific saturated, monounsaturated, polyunsaturated fatty acids, and CLA in human milk[1]

Fatty acid	*r*	*P*
16:00	−0.313	<0.001
18:00	0.306	<0.001
18:1(n-9)	−0.172	0.02
18:2(n-6)	−0.339	<0.001
20:4(n-6)	−0.197	0.007
22:4(n-6)	−0.156	0.033
22:5(n-6)	−0.212	0.004
18:3(n-3)	−0.234	0.001
20:5(n-3)	−0.073	0.323
22:5(n-3)	−0.071	0.336
22:6(n-3)	−0.115	0.116
CLA	0.360	<0.001

[1] $n = 190$.

A similar approach can be taken to estimate the exposure of breast-fed infants to *trans* fatty acids derived from the maternal diet. On average, mature human milk provides 37 g fat/L, with 50% of the energy in the milk as fat (18). Using the results in the present study, the mean and 95th percentile of intake (percent total energy intake) of breast-fed infants to *trans* fatty acids from human milk, estimated from the analyses of human milk and assuming an average intake of 780 mL/day of milk was 2.05 and 4.40 g/d (1.02 and 2.2%), 1.79 and 3.80 g/d (0.89 and 1.90%), 1.53 and 3.77 g/d (0.76 and 1.88%), and 1.33 and 2.41 g/d (0.66 and 1.21%), for milk samples analyzed in 1998 and the 3 5-mo periods beginning in November 2004. However, we note that because these estimates are based on the average fat content of human milk and the average milk intake of the fully breast-fed infant, individual intakes may be much different.

Fatty acids secreted in human milk are derived from synthesis in the mammary gland and by uptake from maternal plasma. Due to the mammary gland enzyme, thioesterase II, fatty acid synthesis in the mammary gland is terminated at the level of myristic acid (14:0) rather than palmitic acid (16:0), as in other tissues (18). Our results show that the total *trans* fatty acids in the milk ($n = 187$) was significantly and inversely associated with the levels of 16:0, and *cis* 18:1(n-9), 18:2(n-6), and 18:3(n-3) are consistent with the replacement of partially hydrogenated fats and oils in retail foods with unhydrogenated soybean and canola oils or more saturated fats, such as palm and palm kernel oil. Similarly, the positive association between 18:0 and *trans* fatty acids in milk shown in our study is not unexpected because 18:0 is higher in hydrogenated oils than in their unhydrogenated counterparts. The lower CLAs in human milk in late 2005 than in 1998 shown in the present study, however, may suggest that the intake of dietary fats derived from ruminant animals, which are also a source of *trans* fatty acids CLA and 18:0, may have decreased among women in our population. On the other hand, available estimates suggest that endogenous conversion of *trans* 11–18:1 (vaccenic acid) to *cis* 9, trans 11–18:2 (CLA) contributes ~25% of the CLA in humans (36), suggesting the lower CLA in human milk collected in 2005 than in 1998 may be explained at least in part by a lower intake of *trans* 11–18:1. In addition, ruminant meats and dairy fats contain 2–5% *trans* fatty acids (9) and contribute about 10% of the total dietary *trans* fatty acids/d (14). This suggests that a decrease in the intake of *trans* fatty acids from animal foods is not likely to explain the estimated 45% decrease in *trans* fat intake among breast-feeding women from 1998 to 2005 suggested by our studies. Consistent with our results, Mosley et al. (28) recently reported a positive relation between total *trans* fatty acids and 18:0, as well as CLA in human milk collected from women in the United States. Whether or not women with higher intake of ruminant fats are also those with higher intake of foods more likely to contain partially and fully hydrogenated vegetable oils cannot be determined from our data. The results of the present study also show a significant inverse association between *trans* fatty acids in human milk and the levels of ARA and its longer

chain metabolites, 22:4(n-6) and 22:5(n-6). Several studies have raised concern that *trans* fatty acids may inhibit the desaturation of dietary linoleic acid and α linolenic acid, and evidence of an inverse association between ARA and *trans* fatty acids in maternal and newborn plasma has been reported (8,34). We found no evidence of an inverse relation between *trans* fatty acids and DHA in human milk. Whether these results are explained by differences in dietary intake or differences in the possible interactions between *trans* fatty acids and the (n-6) and (n-3) series of fatty acids is unclear.

In summary, our results show a decrease in *trans* fatty acids in human milk and provide evidence that the intake of *trans* fatty acids has decreased among women, particularly those women with higher intake of *trans* fatty acids, following the introduction of labeling of *trans* fat on foods sold at retail and the decrease in partially hydrogenated fats and oils in foods such as breads, snack foods, and fried foods in Canada (32,33). New food-labeling laws were also recently adopted in the United States (37). Our studies suggest that changes in the exposure to *trans* fatty acids derived from the industrial partial hydrogenation of vegetable oil has decreased in Canada. Future studies will need to address the possible benefits to human growth, development, and health.

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