

Polyunsaturated fatty acids in maternal diet, breast milk, and serum lipid fatty acids of infants in relation to atopy

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FROM ABSTRACT

BACKGROUND:

The increased consumption of n-6 polyunsaturated fatty acids (PUFA) has been shown to coincide with the increased prevalence of atopic diseases.

We aimed to investigate whether maternal diet and atopic status influence the PUFA composition of breast milk and the serum lipid fatty acids of infants.

METHODS:

Maternal diet was assessed by a food questionnaire. The PUFA composition of breast milk obtained at 3 months from 20 allergic and 20 healthy mothers and of their infants' (10 atopic and 10 nonatopic/group of mothers) serum lipids was analyzed.

RESULTS:

Although no differences in maternal PUFA intake were observed, the breast milk of allergic mothers contained less gamma-linolenic acid (18:3 n-6) than that of healthy mothers.

Similarly, atopic infants had less gamma-linolenic acid in phospholipids than healthy infants, although n-6 PUFA were elevated in other serum lipid fractions in atopic infants.

CONCLUSION:

Our results suggest that dietary n-6 PUFA are not as readily transferred into breast milk or incorporated into serum phospholipids, but may be utilized for other purposes, such as eicosanoid precursors, in allergic/atopic individuals.

Subsequently, high dietary proportions of n-6 PUFA, or reduced proportions of regulatory PUFA, such as gamma-linolenic acid and n-3 PUFA, may be a risk factor for the development of atopic disease.

THESE AUTHORS ALSO NOTE:

"The prevalence of atopic diseases has increased in industrialized countries over the last two decades."

"Breast-feeding has been demonstrated to protect against the development of atopic diseases, although infants may have atopic diseases even during exclusive breast-feeding."

This is a paradoxical phenomenon.

An explanation for this is reduced consumption of saturated fatty acids (SFA) and increased consumption of PUFA has increased over the last 20 years.

Most of the increase in PUFA consumption is due to use of margarine and vegetable oils rich in n-6 PUFA.

Consumption of n-3 PUFA has decreased.

Both linoleic acid (18:2n-6) and alpha-linolenic acid (18:3n-3) are essential PUFA and therefore must be provided by food.

"It has been suggested that man evolved on a diet with a 1:1 ratio of n-6 to n-3. However, in current Western diets, this ratio is approaching 10-25:1, indicating a deficiency in n-3 PUFA."

Linoleic acid (n-6) and alpha-linolenic (n-3) are precursors for longer chain PUFA, and are in continuous competition for the same desaturation and elongation enzymes.

"Due to altered dietary habits, the metabolism of n-6 fatty acids predominates, resulting in the production of longer chain derivatives of linoleic acid such as arachidonic acid (20:4n-6)."

Prostaglandin E2 and other pro-inflammatory eicosanoids (leukotriene B4) derived from arachidonic acid, are important factors promoting atopic inflammation.

"In contrast, n-3 fatty acids and eicosanoids derived from them possess anti-inflammatory properties."

A mother's diet influences the fatty acid composition of her breast milk, which may explain why breast-feeding can influence infant atopic diseases.

In this study, fatty acid composition of serum and breast-milk were assessed from 40 infants and their mothers.

RESULTS

THE MATERNAL DIET AVERAGED:

| | |
|---------------------|-------|
| Protein intake | 15.7% |
| Fat intake | 36.4% |
| Carbohydrate intake | 47.3% |

The proportions of fat intake were:

| | |
|------|-------|
| SFA | 43.1% |
| MUFA | 34.3% |
| PUFA | 15.1% |

The overall n-6 to n-3 ratio of 6.5 to 1.

Mothers having nonatopic children consumed less fat in their diet than mothers whose child suffered from atopic dermatitis.

The intake of n-3 PUFA from marine sources was generally very low, with only 8 mothers consuming oily fish during the recording days.

"When the breast-milk composition was analyzed according to atopic status of the children, the n-6 to n-3 ratio in breast milk from mothers of healthy children was lower than the ratio seen in breast milk from mothers of atopic children."

"A significant correlation was observed between the SFA consumed and the sum of SFA in the breast milk of allergic mothers."

THE INFLUENCE OF BREAST MILK ON SERUM LIPID FATTY ACID PROFILES IN INFANTS:

"Atopic infants had a higher sum of n-6 fatty acids and subsequently a higher n-6 to n-3 ratio."

Nonatopic infants had a higher proportion of docosahexaenoic acid (22:6n-3) and higher sum of n-3 fatty acids in the serum, resulting in a lowered n-6 to n-3 ratio.

DISCUSSION

There are two explanations for why the Western lifestyle is responsible for the increasing susceptibility to atopic disease:

- (1) The hygiene hypothesis
- (2) The dietary hypothesis: the increased prevalence of PUFA in consumption.

The following supports the dietary hypothesis:

- (1) A maternal diet rich in SFA is associated with the atopic status of the children.
- (2) There is a significant correlation between maternal diet and breast-milk content of SFA.
- (3) "It appears that maternal diet during breast-feeding does influence the fatty acid composition of breast milk."
- (4) Breast milk obtained from mothers of infants with atopic dermatitis have more linoleic acid (18:2n-6) and decreased proportions of gamma-linolenic acid (18:3n-6) PUFA than the breast milk of nonatopic infants.
- (5) Breast milk of nonatopic infants contained more gamma-linolenic acid (18:2n-6) and alpha-linolenic acid (18:3n-3) than the breast milk of atopic controls.
- (6) Gamma-linolenic acid (18:3n-6) and the n-6 to n-3 ratio were lower in the breast milk of allergic mothers than in that of healthy mothers.
- (7) "Dietary supplementation of gamma-linolenic acid has been shown to reduce the severity of skin symptoms in children with atopic dermatitis."
- (8) The "proportions of n-3 fatty acids have been demonstrated to possess anti-inflammatory properties and to compete with n-6 PUFA for the same desaturation and elongation enzymes."

Atopic disease prevention emphasis must be placed on the maternal diet during pregnancy.

Gamma-linolenic acid (18:3n-6) is required for normal skin function, and if an infant is exposed through breast milk and prior to immunologic sensitization, such exposure should protect against atopic sensitization.

The authors found that the breast milk of mothers with healthy children tended to contain more n-3 fatty acids.

They suggest that the shortage of regulatory PUFA, such as gamma-linolenic acid and n-3 PUFA, in the breast milk could promote intestinal inflammation, subsequently increasing the permeability of the gut barrier.

There is epidemiologic evidence linking the increase in n-6 PUFA consumption to atopic sensitization.

Atopic subjects have more linoleic acid (18:2n-6) in breast milk and in serum, yet reduced levels of gamma-linolenic acid (18:3n-6).

The conversion of linoleic acid (18:2n-6) to gamma-linolenic acid (18:3n-6) requires delta-6-desaturase enzyme, suggesting a dysfunction of delta-6-desaturase in atopic subjects. [Desaturation means adding a double bond].

This suggests that atopic individuals may have an impaired capacity to metabolize PUFA through desaturation.

In the present study, atopic infants had more n-6 fatty acids and less n-3 fatty acids in serum, indicating that atopic infants (exclusively breast-fed) consumed relatively more n-6 fatty acids.

“The present study demonstrated the carryover effect of dietary fatty acids from the maternal diet via breast milk into infants' serum lipid fatty acids.”

Low maternal consumption of foods rich in n-3 PUFA was noted and evidenced as a high n-6 to n-3 PUFA ratio in the breast milk.

The authors suggest that:

“The excess dietary supply of n-6 PUFA or reduced proportions of regulatory PUFA, such as gamma-linolenic acid and n-3 PUFA, may be a risk factor for the development of atopic disease.”

KEY POINTS FROM DAN MURPHY

- (1) Both n-6 and n-3 PUFA are critical for health, and these fats must come from the diet.
- (2) The optimum ratio of n-6 to n-3 is 1 to 1.
Western diets typically produce a ratio of 10-25 to 1.
- (3) An infant's PUFA n-6 to n-3 ratio is dependent upon the mother's diet.
- (4) An unbalanced maternal n-6 to n-3 ratio will result in an unbalanced ratio in her breast-fed infant.
- (5) Although gamma-linolenic acid (18:3n-6) is an n-6 PUFA, it is considered to be "regulatory" and functions as anti-inflammatory and immuno-regulatory, especially for atopic disease and atopic dermatitis.
- (6) Exposure to gamma-linolenic acid (18:3n-6) should be as early as possible in life, prior to "atopic sensitization."
[The desired Th1 IgG v. the atopic Th2 IgE immune system maturation response.]
- (7) Some mothers and some infants have a dysfunction of the enzyme (delta-6-desaturase) that convert the Western diet rich supply of linoleic acid (18:2n-6) gamma-linolenic acid (18:3n-6).
- (8) Consequently dietary supplementation of gamma-linolenic acid (18:3n-6) may be necessary. [Evening primrose and borage oils are good sources].
- (9) Saturated fats remain high in the maternal diet, and increased maternal saturated fat is correlated to allergic disease.
- (10) Atopic disease prevention emphasis is dependent upon the maternal diet during pregnancy.