Consumption of fish, butter and margarine during pregnancy and development of allergic sensitizations in the offspring: Role of maternal atopy

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Consumption of fish, butter and margarine during pregnancy and development of allergic sensitizations in the offspring: role of maternal atopy

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It has been suggested that changes in dietary habits, particularly increased consumption of ω-6 polyunsaturated fatty acids (PUFA) and decreased consumption of ω-3 PUFAs may explain the increase in atopic disease seen in recent years. Furthermore, it seems possible that it is mainly prenatal or very early life environmental factors that influence the development of allergic diseases. It has also been suggested that intrauterine risk factors may act differently if mother themselves suffer from allergic disease.

The aim of this study was to investigate whether the consumption of fish, butter and margarine during pregnancy might influence the development of allergic sensitizations in the offspring. The study population was divided into the offspring of allergic and non-allergic mothers. This was a retrospective cohort study enrolling 295 offspring of allergic mothers and 693 of non-allergic mothers. Information regarding maternal intake of fish, butter and margarine during pregnancy as well as other prenatal and perinatal confounding factors were retrospectively assessed by parental report via a standardized questionnaire. Atopy was determined by skin-prick tests (SPT) to eight prevalent inhalant allergens and two foods.

In the allergic mothers’ group there is no clear correlation between maternal intakes of fish, butter and margarine and sensitizations to food or inhalants.

In the non-allergic mothers’ group there was no correlation between butter and margarine intake and food or inhalant sensitizations. On the contrary, a protective effect of fish intake on SPT positivity was observed. In particular, frequent maternal intake (>2–3 times/wk or more) of fish reduced the risk of food sensitizations by over a third (aOR 0.23; 95% CI: 0.08–0.69). A similar trend, even if not significant, was found for inhalants. Finally, even in the whole study population, i.e. allergic group plus non-allergic group, there was a similar trend between increased consumption of fish and decreased prevalence of SPT positivity for foods.

This study shows that frequent intake of fish during pregnancy may contrast the development of SPT sensitizations for foods in the offspring of mothers without atopic disease. Therefore, larger prospective studies are needed, enrolling mothers with and without allergic disease, to confirm these results.

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Key words: fish; butter; margarine; allergic sensitizations; prenatal; allergic mothers

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It has been suggested that the changing lifestyle in western countries may explain the dramatic increase seen in the prevalence of atopic disease in recent years. Among various possible explanations, Black and Sharpe proposed that changes in dietary habits, particularly the increased use of margarine and vegetable oils, containing $\omega$-6 polyunsaturated (PUFA) fatty acids and the decreased consumption of oily fish, which contains $\omega$-3 PUFAs, may lead to the development of allergic sensitizations; this might explain the recent increase in the prevalence of asthma, eczema and allergic rhinitis (1).

This hypothesis was later supported by several observational studies showing that the higher the fish intake, the lower the prevalence of atopic disease (2–4) even though experimental studies in asthma patients have not demonstrated an improvement in asthma after supplementation with fish oil (5), and up to now there is no conclusive evidence that dietary supplementation with fish oil is beneficial (6).

Similarly, other studies have supported the hypothesis that the increased intake of margarine and $\omega$-6 PUFA may increase the risk of asthma (7, 8). In addition, other studies have suggested that the increased intake of butter, which is rich in saturated fats, may reduce the risk of asthma (9, 10), hay fever and atopic sensitizations in children (11). There are, however, no relevant intervention studies, and thus no definitive conclusion can be drawn regarding these last two issues.

It seems likely that above all prenatal or very early life environmental factors may influence the development of allergic disease (12, 13). While such studies have addressed only the possible effect of dietary fats on allergic diseases in adults or children, studies, which explore dietary habits during pregnancy or in early life, are scanty.

In addition, it has been suggested that there may be preferential transmission of atopic disease through the mother (14) and that risk factors for atopy may vary according to the presence or absence of maternal atopic disease (15).

The aim of this study was to investigate whether the consumption of fish, butter and margarine during pregnancy has different effects on the development of allergic disease and sensitizations in the offspring of atopic or non-atopic mothers. We chose allergic sensitizations as the end-point because it is well known that these develop in the first years of life and often precede clinical allergy.

Materials and methods

Study design and population

The study was conducted in Italy between September 2001 and March 2002, and enrolled 1044 children consecutively attending outpatients’ allergic clinics in six different General Hospitals in Rome. It was designed to investigate known prenatal/perinatal risk factors and possible new risk factors for asthma and atopy in the offspring.

In the present study, the occurrence of allergic sensitizations to foods and to inhalants in the offspring were the main outcomes to be evaluated in relation to the consumption of fish, butter and margarine during pregnancy, in relation to the presence and absence of maternal atopy.

Data for maternal atopy were available for 988 mothers, so the present study involved 988 children, subdivided into a group of allergic mothers’ offspring (n = 295) and a non-allergic group (n = 693). About 744 children were affected by atopic diseases (asthma or rhinitis or eczema). The remaining 244 were attending allergy clinics mainly owing to respiratory symptoms (cough, rhinitis, etc.), gastrointestinal symptoms (diarrhoea, failure to thrive, etc) or skin disease (urticaria, cutaneous rashes, etc.). Exclusion criteria were immunodeficiency, connective tissue disease or chronic respiratory tract disease other than asthma.

Definition of variables

A detailed description of the methods of the study has been reported elsewhere (16). Briefly, the parents had to complete a standardized, self-administered questionnaire before completing the medical history and having a structured interview with a doctor. They then underwent skin-prick tests (SPT).

The questionnaire included several questions on a number of known or possible prenatal and perinatal risk factors for childhood asthma and atopy, such as fever episodes, flu episodes, use of antibiotics, use of salicylate, threatened abortion, use of isoxsuprine, hyperemesis and mode of delivery.

Socio-demographic variables were collected, such as maternal age, education (number of years) and occupation (housewives, office workers, retail trade workers, self-employed professionals), maternal and paternal smoking (no, <10 cigarettes per day, >10 cigarettes per day), number of pregnancies, mode of delivery, age of gestation (<36 wk or over), birth weight and
also paternal asthma or allergic rhinitis or eczema.

Information regarding maternal intake of fish, butter and margarine during pregnancy was obtained using a 5-level scale: never, 1 time/month, 1 time/wk, 2–3 times/wk, almost daily.

Mothers reporting at least one of the following: (a history of) asthma, hay fever or atopic eczema were defined as allergic and mothers reporting that they had none of these complaints were defined as non-allergic.

Skin-prick tests were performed by trained doctors on the volar aspects of the forearm with standardized allergen extracts, using a lancet with a 1 mm tip. A positive (histamine 10 mg/ml) and negative (diluent) control was added. A weal reaction ≥3 mm of the mean weal diameter to one or more of the allergens tested 15 min after the application of the allergen extracts, after subtraction of their reaction to the negative control, were considered positive.

We tested for the most common allergens in our country. Of the inhalants, we tested Dermatophagoides pteronissinus, Alternaria tenuis, Aspergillus fumi-gatus, mixed grass pollen, Artemisia vulgaris, Parietaria officinalis, Olea europaea, cat dander. Of the dietary allergens, we tested raw cow’s milk and egg white and also other foods when indicated by the clinical history.

Statistical analysis

The spss software package version 9.0 was used for all calculations. The association between possible prenatal risk factors and other known risk factors such as age, gender, number of older siblings, education, occupation and atopic sensitization for foods and inhalants were preliminarily analysed with Pearson chi-squared tests.

Logistic regression was performed to analyse the association between fish, butter or margarine consumption (using ‘never’ as the reference) and the two outcome variables, after adjusting for variables that, in the groups, were associated with the two outcomes in the univariate analysis at a p-value of <0.10. Adjusted odds ratio (aOR) and 95% confidence interval (CI) were computed to determine the degree of association. Moreover, food consumption was included as a categorical variable to test for linear trend. Spearman’s correlation coefficients were computed to evaluate the degree of correlation among dietary variables. The p-values of <0.01 were considered statistically significant because of multiple comparisons.

Results

Table 1 shows the background characteristics of the study population, after subdivision into the two study groups, i.e. children with allergic and non-allergic mothers. Among allergic mothers, 206 were affected by rhinitis, 72 by eczema and 94 by asthma. The mean age of children and mothers was about 5 and 36 yr respectively and the groups were comparable for the main background variables. Prevalence of skin sensitization for foods was slightly greater in the allergic mothers’ group (10.8%) vs. the non-allergic mothers’ group (7.1%; Pearson chi-square: p = 0.048) whilst the number of children free of any atopic diseases (asthma, oculorhinitis and eczema) was greater in the non-allergic mother’s group (26.7%) vs. the allergic mothers’ group (20%; p = 0.02). As expected, sensitizations in the...
children for inhalants significantly increased with age, while those for food decreased, both in the allergic mothers' and non-allergic mothers' groups. Skin sensitizations for inhalants were similar in the two groups (Fig. 1a,b).

Table 2 shows maternal dietary intake of fish, butter and margarine during pregnancy. Fish intake was more frequent than that of butter or margarine. In particular, in both groups about 75% reported eating fish 1 time/wk or more, while only about 40% and 20% reported a similar intake for butter or margarine respectively. Furthermore, 3.7% of the mothers reported never having eaten fish against 27.3% and 68.6% for the same intake of butter and margarine. In particular, in both groups about 75% reported eating fish 1 time/wk or more, while only about 40% and 20% reported a similar intake for butter or margarine respectively. The scant intake of butter and margarine is in line with the traditional diet in central Italy (17).

There is no significant difference in the fish and butter intake between the allergic mothers' and the non-allergic mothers' groups. In contrast, margarine intake is significantly more frequent (Pearson chi-square: p = 0.004) in the allergic mothers' group: there is also a correlation between the increasing intake of margarine and the risk of allergy in the mothers, so that daily intake of margarine increases the risk almost threefold. However, after adjusting for maternal smoking, maternal education and maternal occupation, the association between maternal asthma and maternal intake of margarine is not statistically significant.

There were several significant correlations between the intake of fish, butter and margarine, particularly between butter and margarine (Table 3).

Dietary intake and food sensitizations

Table 4 shows the OR, aOR and 95% CI of the association of maternal food intake during pregnancy and food sensitizations in the offspring.

To reduce the effects of small represented groups (<5%) in the analysis and to simplify the tables, we reclassified reported intake from five into three subgroups, i.e. ‘1 time/month or less’, ‘1 time/wk’, ‘2–3 times/wk or more’. The first comprises ‘never’ and ‘1 time/month’, the second ‘1 time/wk’, the third ‘2–3 times/wk’ or ‘almost daily’. The responses ‘never’ and ‘1 time/month’ represent quite a similar level of intake as do ‘2–3 times/wk’ and ‘almost daily’.

In the allergic mother group there is no clear correlation between maternal intake of fish, butter and margarine and food sensitizations. Adjusting for confounders does not substantially change the results.

In the non-allergic mothers' group there is no correlation between butter and margarine intake and food sensitizations. On the contrary, a protective effect of fish intake on SPT positivity for foods was observed. In particular, frequent maternal intake of fish reduced the prevalence of food sensitizations by over a third. The protective effect was evident even in the logistic regression adjusted for confounders (aOR 0.22; 95% CI: 0.08–0.55 for intake ‘1 time/wk’ and 0.23; 95% CI: 0.08–0.69 for intake ‘2–3 times/wk or more’). Adjusting also for butter and margarine intake slightly increased the protective effect of fish intake (aOR 0.20; 95% CI: 0.07–0.55 and 0.16; 95% CI: 0.04–0.58 for intake ‘1 time/wk’ and ‘2–3 times/wk or more’, respectively). Adjusting also for other common confounding factors (such as gender, maternal smoking, paternal atopy), did not substantially change the results.

Adjusting also for hyperemesis, which seems related to a high daily intake of saturated fats before pregnancy (18) and with higher serum
prostaglandin E2 (PGE2) levels during pregnancy (19), further increased the aOR (0.13; 95% CI: 0.04–0.38 and 0.14; 95% CI: 0.04–0.47 for intake /C212 1 time/wk/C213 and /C212 2–3 times/wk or more/C213, respectively).

A similar protective effect results also when the association between food sensitizations and fish intake was carried out with the original five subgroups of foods intake (p-value for trend 0.006). Given these data, we decided to explore whether the protective effect were evident for individual foods. To this end, we sought the association of fish intake with the two main food allergens in our study population, i.e. cow’s milk and eggs white.

Intake of fish ‘1 time/wk’ and ‘2–3 times/wk or more’ reduced milk sensitization respectively about sixfold (aOR 0.15; 95% CI: 0.04–0.59) and more than 10-fold (0.05; 95% CI: 0.00–0.54), and a similar protective effect resulted also for egg sensitization (aOR 0.26; 95% CI: 0.09–0.76 for intake of fish ‘1 time/wk’ and 0.33; 95% CI: 0.10–1.07 for intake of fish ‘2–3 times/wk or more’).

Finally, even in the whole population, i.e. allergic group plus non-allergic group, there was a significant trend between increased consumption of fish and decreased prevalence of SPT positivity for foods (p-value for trend: 0.02 and 0.008 with fish intake classified in the original five and subsequent three subgroups, respectively). A significant protective effect of fish intake resulted in the logistic regression only for intake /C212 1 time/wk/C213 (1 time/wk: aOR 0.34; 95% CI: 0.15–0.75; p = 0.007, 2–3 times/wk or more: 0.42; 95% CI: 0.17–1.02; p = 0.05; adjusted for variables that, in the whole study population, were associated with SPT positivity for foods in the univariate analysis at a p-value of <0.10, e.g. age, number of older siblings, allergic clinics, maternal age, age of gestation, gender, maternal education, paternal atopy and also maternal atopy).

Dietary intake and inhalant sensitizations

There was no clear relationship between maternal food intake during pregnancy and inhalant sensitizations in the offspring. (Table 5).

In the allergic mothers’ group butter intake ‘1 time/wk’ seemed to have a significant protective effect, also after adjusting for confounders; however, this was not confirmed with the increase of butter intake. Adjusting also for fish and margarine did not substantially modify the results.

In the non-allergic mothers’ group, fish intake ‘2–3 time/wk or more’ seemed to have a slight

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**Table 2. Intake of fish, butter and margarine in the allergic mothers’ and non-allergic mothers’ groups**

<table>
<thead>
<tr>
<th></th>
<th>Allergic mothers (n)</th>
<th>%</th>
<th>Non-allergic mothers (n)</th>
<th>%</th>
<th>OR (95% CI)</th>
<th>p-value for trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fish</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never (referent category)</td>
<td>13</td>
<td>4.6</td>
<td>22</td>
<td>3.3</td>
<td>1.0</td>
<td>0.627</td>
</tr>
<tr>
<td>1 time/month</td>
<td>49</td>
<td>17.3</td>
<td>114</td>
<td>17.2</td>
<td>0.72 (0.33–1.56)</td>
<td></td>
</tr>
<tr>
<td>1 time/wk</td>
<td>138</td>
<td>48.8</td>
<td>330</td>
<td>49.8</td>
<td>0.70 (0.34–1.44)</td>
<td></td>
</tr>
<tr>
<td>2–3 times/wk</td>
<td>76</td>
<td>26.9</td>
<td>182</td>
<td>27.5</td>
<td>0.70 (0.33–1.47)</td>
<td></td>
</tr>
<tr>
<td>Almost daily</td>
<td>7</td>
<td>2.5</td>
<td>15</td>
<td>2.3</td>
<td>0.78 (0.25–2.44)</td>
<td></td>
</tr>
<tr>
<td>Butter</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never (referent category)</td>
<td>81</td>
<td>29.2</td>
<td>165</td>
<td>26.3</td>
<td>1.0</td>
<td>0.446</td>
</tr>
<tr>
<td>1 time/month</td>
<td>75</td>
<td>27.1</td>
<td>208</td>
<td>33.2</td>
<td>0.73 (0.50–1.06)</td>
<td></td>
</tr>
<tr>
<td>1 time/wk</td>
<td>72</td>
<td>26.0</td>
<td>168</td>
<td>26.8</td>
<td>0.87 (0.59–1.28)</td>
<td></td>
</tr>
<tr>
<td>2–3 times/wk</td>
<td>38</td>
<td>13.7</td>
<td>72</td>
<td>11.5</td>
<td>1.07 (0.66–1.72)</td>
<td></td>
</tr>
<tr>
<td>Almost daily</td>
<td>11</td>
<td>4.0</td>
<td>14</td>
<td>2.2</td>
<td>1.60 (0.69–3.68)</td>
<td></td>
</tr>
<tr>
<td>Margarine</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never (referent category)</td>
<td>173</td>
<td>64.1</td>
<td>436</td>
<td>70.6</td>
<td>1.0</td>
<td>0.032</td>
</tr>
<tr>
<td>1 time/month</td>
<td>41</td>
<td>15.2</td>
<td>92</td>
<td>14.9</td>
<td>1.12 (0.74–1.68)</td>
<td></td>
</tr>
<tr>
<td>1 time/wk</td>
<td>34</td>
<td>12.6</td>
<td>43</td>
<td>7.0</td>
<td>1.98 (1.22–2.23)</td>
<td></td>
</tr>
<tr>
<td>2–3 times/wk</td>
<td>11</td>
<td>4.1</td>
<td>38</td>
<td>6.1</td>
<td>0.72 (0.36–1.46)</td>
<td></td>
</tr>
<tr>
<td>Almost daily</td>
<td>11</td>
<td>4.1</td>
<td>9</td>
<td>1.5</td>
<td>3.08 (1.25–7.56)</td>
<td></td>
</tr>
</tbody>
</table>

Data on fish intake, butter intake and margarine intake were available in 283, 277 and 270 in the allergic mother group and in 683, 627 and 618 controls respectively.

**Table 3. Correlations between the intake of fish, butter and margarine**

<table>
<thead>
<tr>
<th></th>
<th>Fish</th>
<th>Butter</th>
<th>Margarine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fish</td>
<td>1.0</td>
<td>0.084*</td>
<td>0.119**</td>
</tr>
<tr>
<td>Butter</td>
<td>0.084*</td>
<td>1.00</td>
<td>0.305**</td>
</tr>
<tr>
<td>Margarine</td>
<td>0.119**</td>
<td>0.305**</td>
<td>1.00</td>
</tr>
</tbody>
</table>

*Correlation is significant at the 0.05 level (two-tailed).
**Correlation is significant at the 0.01 level (two-tailed).
protective effect, even though not significant, which increased after adjusting for confounders in the logistic regression (aOR 0.55; 95% CI: 0.28–1.08; p = 0.08). Adjusting also for butter and/or margarine intake did not substantially change the effect (aOR 0.54; 95% CI: 0.27–1.10; p = 0.09).

Butter intake ‘1 time/wk’ seemed to be a significant risk factor: also in this case; however, this was not confirmed with the increase of butter intake.

Finally, a trend towards a protective effect, even if not significant, resulted in the whole study population (allergic mothers’ group plus non-allergic mothers’ group) given that the highest fish intake reduced the risk for inhalant sensitizations by about half (aOR 0.57; 95% CI: 0.32–1.03, p = 0.06; adjusted for variables that, in the whole study population, were associated with SPT positivity for inhalants in the univariate analysis at a p-value of <0.10, e.g. age, gender, number of older siblings, allergy clinics, maternal age, number of pregnancies, maternal education, preterm labour, maternal atopy). In this group, butter intake did not appear to have any significant effect.

Discussion

To our knowledge, studies which have specifically addressed the possible effect of maternal diet...
during pregnancy on the development of atopic sensitizations in children are scarce.

We found that the higher the intake of fish during pregnancy, the lower the development of allergic sensitizations to foods in the offspring of mothers who do not suffer from allergic disease. A similar trend was found in the whole study population, even though statistically significant in the logistic regression only for intake ‘1 time/wk’. Neither maternal butter nor margarine intake seemed related to SPT positivity in the offspring of either group. However, it should be mentioned that the intake of margarine and butter is much lower in our study population than in other studies: margarine intake as a spread was reported by 45% of children in Bolte et al.’s study, while only about 7% in this study reported eating margarine more than 2–3 times/wk (7) and daily consumption of butter in this study is only about one-third of that reported by Woods et al. (about 3% vs. about 10%; 9). This low level of consumption limits the possibility of identifying any potential effect in the offspring.

In agreement with others studies (7, 8), the intake of margarine was significantly more frequent in the mothers suffering from allergic disease.

Methodological issues must be discussed before the findings can be interpreted.

Our study population was selected from allergy clinics, thus caution is required before extrapolating the results of our study to the general population. Because of the cross-sectional study design, the frequency of maternal food intake during pregnancy was retrospectively assessed, and therefore subject to a certain level of recall bias. However, pregnancy is generally lived as a very particular period in life: this fact may lessen the possibility of forgetting even trivial events. Moreover, the retrospective design of this study limits the possibility of affirming a causal relationship between fish consumption and reduced skin test positivity.

Another issue could be that maternal dietary habits may influence the offspring’s dietary habits and, as we did not investigate the latter, we cannot exclude the possibility that the children’s diet also influenced the development of skin test sensitizations. However, as fish intake was quite similar in the allergic mothers’ and in the non-allergic mothers’ groups, while the protective effect was shown only in the non-allergic mothers’ group, it seems unlikely that maternal dietary habits influencing child dietary intakes may have played a role.

Finally, we took into account many known prenatal and perinatal risk factors and socio-economic variables, and also hyperemesis, which is a common cause of modifications in dietary habits during pregnancy (20). However, the effect of other relevant factors not considered in this study, such as breast feeding, or perhaps other unknown factors, should be kept in mind.

This study investigated the development of atopy using objective criteria, such as SPT, and demonstrated that this protective effect of fish consumption on food sensitizations is consistent also regarding specific foods, such as cow’s milk and egg white.

It also showed that maternal atopy clearly interferes with this protective effect. We believe that this is a very important point, given that the majority of studies concerning the prevention of allergic disease are carried out, above all, in families genetically predisposed to developing allergy.

The hypothesis that consumption of dietary fatty acid can influence the development of atopic disease relies on several mechanisms, among which the fact that fatty acid intake may have effects on the immune system. Increased consumption of ω-6 PUFA, such as linoleic acid, may promote the formation of PGE2 and alter the T-helper (Th)1/Th2 cell balance thus enhancing the formation of immunoglobulin E (IgE) from T cells and promoting allergic sensitizations. In contrast, ω-3 PUFA, such as eicosapentaenoic (EPA) and docosahexaenoic (DHA) acid, found in fish oil, competitively inhibit the formation of prostaglandins and leukotrienes derived from arachidonic acid (21).

Therefore, there is growing interest in the role of fatty acid intake during pregnancy and the development of allergic disease.

A population-based birth cohort study recently had ‘not found convincing evidence that higher exposure to ω-6 vs. ω-3 fatty acids in utero promotes the development of eczema or wheezing in early childhood, although the cord blood findings for the AA:EPA ratio and eczema and for the LA:ALA ratio and later onset wheeze were in keeping with this hypothesis’. In the above study, however, only 1% of mothers ate fish more than three times a week and the possible effect of fatty acid exposure on atopic sensitizations was not investigated (22).

Recently, in a double-blind placebo-controlled trial, Dunstan et al. explored whether supplementation with fish oil capsules from 20 wk gestation until delivery could modify the immune response in infants at high risk of atopy. She showed that all neonatal cytokine [interleukin (IL)-5, IL-13, IL-10 and interferon (IFN)-γ] responses to all allergens tended to be lower in
the fish oil-treated group (23). Moreover, supplementation with fish oil reduced IL-13 levels in cord blood, and was positively associated with IgA and sCD14 levels in breast milk (24). Interestingly, although the study was not designed to examine clinical effects, she also noted that infants in the fish oil group were three times less likely to have a positive SPT to egg white at 1 yr of age (OR = 0.34; 95% CI: 0.11–1.02) an OR almost equal to that observed in this study in the non-atopic mothers’ group (0.33; 95% CI: 0.10–1.07; 24).

Recent data show that in allergic pregnant mothers there is a disturbed fatty acid metabolism that could affect the fatty acid composition in their babies. It was shown that there is a correlation between maternal and neonatal levels of linoleic acid, arachidonic acid, docosapentaenoic acid and DHA only in the non-allergic mothers, while none of these relationships were observed between allergic mothers and their babies (25). Therefore, it may be that allergic mothers need a greater intake of ω-3 PUFA to contrast allergic development in children.

In the Dunstan et al. study (24), fish oil capsules provided a supplementation of 3.7 g of ω-3 PUFA, which is approximately equivalent to one fatty fish meal/day while in this study only 2.5% of the mothers reported a similar intake. Thus, it is tempting to speculate that our lower intake could account for the inability of this study to demonstrate a protective effect of fish consumption on atopic sensitizations in the allergic mothers’ group.

Owing to the retrospective design of this study, we are not able to affirm that the effect of fish consumption on skin test sensitizations is actually due to the higher intake of ω-3 PUFA; however as recently suggested, it is possible that general diet and natural foods play a role in the development of allergic diseases rather than individual nutrients (26).

It is well known that the first IgE responses develop to food proteins, particularly those to egg white and cow’s milk, and then towards inhalants (27). This could explain why the protective effect of fish intake during pregnancy seems, in our study, greater towards foods than inhalants.

It has been shown that the majority of atopic manifestations and allergic sensitizations occur in infants without demonstrable risk at birth, thus suggesting the search for measures applicable to the population at large, independent of individual risk assessment (28). Our study demonstrated that maternal consumption of fish is inversely related with SPT positivity in the offspring of mothers who do not suffer from allergic disease. If the results of our work are confirmed by other well-designed research, such as prospective population-based studies, enrolling also non-allergic mothers, it could be beneficial to advise mothers to increase fish intake during pregnancy. This could constitute a simple yet effective means to contrast the increase of allergic sensitizations and perhaps also combat the allergic epidemic.

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