

ORIGINAL ARTICLE

Blood fatty acid composition in relation to allergy in children aged 2–9 years: results from the European IDEFICS study

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BACKGROUND/OBJECTIVES: Blood polyunsaturated fatty acids (PUFA) are involved in allergy development, but the etiological role of n-6 and n-3 PUFA is still controversial. A European multicenter study of children (IDEFICS) provided the opportunity to explore the cross-sectional association between fatty acids (FA) and allergy.

SUBJECTS/METHODS: Blood FA levels were measured in 2600 children aged 2–9 years and were recorded as the percentage of weight of all FA detected. Logistic regression of allergy status on FA components was adjusted for age, sex, country, body mass index, family history of allergic disease, breast-feeding, and number of siblings. The results were given as odds ratios (OR) for current vs no allergy ever and an increase in FA by 1 s.d.

RESULTS: Overall, higher proportions of n-6 PUFA were associated with higher odds of allergy (OR = 1.21 (1.05, 1.40)). Monounsaturated FA (MUFA) were associated with reduced risk for allergy (OR = 0.75 (0.65, 0.87)), whereas saturated FA did not differ by allergy status. The strongest associations were observed in children < 4 years old, with ORs of allergy given as 1.62 (1.15, 2.29) for n-3 PUFA and 0.63 (0.42, 0.95) for MUFA. With regard to individual FA, these associations were independently observed for docosapentaenoic acid (22:5 n-3) and oleic acid (18:1 n-9).

CONCLUSIONS: Both PUFA subtypes were positively associated with allergy in an age-dependent manner, whereas MUFA was associated with less allergy. The observation of high proportions of n-3 PUFA in allergic children younger than 4 years might help to understand the nature of early onset of atopic disease.

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INTRODUCTION

During the past decade, a marked rise in the prevalence of atopic diseases has been observed in industrialized countries,¹ which is paralleled by changes in dietary habits.² Increased consumption of meat as well as vegetable oils and margarine has resulted in increased levels of n-6 polyunsaturated fatty acid (PUFA)—in particular, arachidonic acid (AA) and linoleic acid. Several studies associated consumption of margarine with an increased risk for allergy development, whereas consumption of butter appeared to be protective.^{3–5} In contrast to n-6 PUFA, the consumption of n-3 PUFA remained the same, resulting in the n-6 to n-3 PUFA ratio being much higher compared with the traditional value of 1–2.² It was suggested that increased consumption of n-3 PUFA should show a protective effect on atopy similar to cardiovascular disease, but the evidence is inconclusive.^{6–8}

The natural history of atopic disease begins often with eczema or food allergy in the neonatal period, and progresses with age to allergic rhinitis and asthma.⁹ The early presentation of disease suggests that early modulation of immune function is of key importance for development of tolerance or allergy.¹⁰ Several groups studied the association between blood fatty acids (FA) and

atopy in infants and children. A Finnish study reported higher proportions of n-6 PUFA in infants with atopic eczema compared with those in controls when measured in cheek cell phospholipids. They also found higher proportions of specific n-3 (eicosapentaenoic acid) and n-6 PUFA (γ-linolenic acid) when measured in serum phospholipids.¹¹ Differing associations between PUFA components and allergy were reported for 3-month-old infants depending on the lipid class studied.¹² Studies in children aged 2 years or older showed negative associations between n-3 PUFA and eczema, but no associations between PUFA and allergic rhinitis.^{13,14}

The aim of the present study was to compare the proportion of n-6 and n-3 PUFA and individual FA in children between 2 and 9 years of age with and without concomitant manifestation of atopic disease. As the children came from eight European countries, we expected to cover different lifestyle exposures that could affect individual blood FA levels.

SUBJECTS AND METHODS

A multicenter study for identification and prevention of dietary- and lifestyle-induced health effects in children and infants (IDEFICS study) was

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conducted between 2006 and 2012. A total of 16 228 children from eight countries aged 2 to 9 years were examined in 2007/08, providing data on anthropometry, lifestyle and biomarkers. Parents gave written informed consent for collection and storage of personal data and biological samples. Each survey center obtained ethical approval from the local or national ethics committee.¹⁵

One focus of the IDEFICS study lay on childhood obesity, which is a complex disease with many comorbidities. For this reason, a list of common childhood diseases was set up by a panel of pediatricians that included allergy but no subtypes of allergy. Researchers wanted to know about the phenotype of allergic disease, such as food allergy, eczema or allergic rhinitis, which was confirmed by a physician. This interpretation was conferred to the parents who completed a standardized questionnaire during an interview with a nurse. The questions about allergy read as follows: 'Has a physician ever diagnosed your child with an allergy? If yes, at which age was the first diagnosis established, and does your child have the disease currently?' The answers were categorized into 'current allergy', 'former allergy' or 'no allergy ever'. Parents also reported on diseases in the family, breast-feeding, and the number of siblings. About half of the children were exclusively breast-fed for 4 months or longer, and this value was used for dichotomization. Age- and sex-specific categories of body mass index (BMI)—that is, underweight, normal weight, overweight and obesity—were based on IOTF (International Obesity Task Force) cutoff values.¹⁶

Circulating blood FA were measured using capillary blood after finger prick collection on an antioxidant-treated card, directly methylated without extraction and subjected to a rapid gas chromatographic method.^{17,18} The procedure gives the contribution of individual FA as weight percentage (wt%) of global blood FA, and provides a validated approach to the assessment of the FA status in humans.¹⁸ FA with 16 to 24 C atoms were measured and are listed in Supplementary Table S1, separately for saturated FA (SFA), monounsaturated FA (MUFA) and PUFA. About 2/3 of n-6 PUFA consisted of linoleic acid and 1/3 of n-6 PUFA consisted of long-chain FA (LC-FA, with more than 18 C atoms). N-3 PUFA represented only 7% of total PUFA and consisted to 91% of LC-PUFA.

FA composition was measured in a subsample of 2600 children with oversampling of overweight children because of the study's focus on weight status.¹⁹ About 70% of the subsample was part of a case-control study for overweight and obesity,¹⁵ whereas the remaining 30% was randomly selected maintaining equal distributions of age and sex on group level. As this study investigates associations between biomarkers and disease, it is less dependent on the representativeness of the underlying sample. However, special attention was paid to confounding or effect modification of associations by weight status. Information on FA and allergy was complete in 2594 children, which defines the analytic sample.

Descriptive statistics comprised mean value and standard deviation (s.d.) for continuous variables and frequencies for categorical variables, separately for categories of current, former and no allergy ever. To account for the study design and because children with current or former allergy were older and more likely to be boys, comparisons of mean values of FA and of potential confounders by allergy status were adjusted for age, sex and country. For linear models, the normal distribution of residuals and their independence of predicted values were checked. Logistic models were tested using the Hosmer–Lemeshow test (P -value > 0.1). To illustrate the age variation of FA by allergy status, we used spline regression of FA components on age, allergy and their interaction (cubic splines with 5 knots). We performed logistic regression of current vs no allergy ever on the different FA components that were standardized to zero mean and unit s.d. Regression models were adjusted for age, age², sex, country, family history of allergic disease, BMI category, number of siblings and breast-feeding. Because of the small number of allergy cases, age-stratified models contained FA components only, followed by stepwise forward selection of variables. Quadratic terms for FA and interactions with covariates were tested. Results were given in terms of odds ratios (OR) with 95% confidence interval (CI) for current vs no allergy ever and a change in FA by 1 s.d. The performance of different logistic models was compared using the area under the receiver operating characteristic curve (AUROC). Statistical analyses were performed using SAS version 9.4 (SAS Institute Inc., Cary, NC, USA). Statistical significance was set at P -value < 0.05 (two-sided tests). To account at least partially for multiple testing, we used a P -value of 0.01 in age-stratified analyses or in analyses of individual FA.

RESULTS

Current allergy was reported in 330 out of 2594 (13%) children, of whom one-third ($n = 103$) were diagnosed before the age of two. About 23% of allergic children had outgrown their allergy ($n = 98$), of whom 40 were diagnosed before the age of two. Table 1 gives a description of variables for current or former allergy compared with no allergy ever. Children with allergy were on average half a year older than children who never had an allergy. Boys were more likely than girls to have an allergy. The proportion of children with allergy differed between countries, with higher values in Belgium, Estonia and Italy (not shown). Furthermore, current allergy was associated with family history of allergic disease, being an only child, and with shorter exclusive breast-feeding. Controlling for differences in age, sex and country, children with current allergy had higher proportions of total and n-6 PUFA, but lower MUFA, than did children with no allergy ever. N-3 PUFA was higher in allergy too, but significantly only for former vs no allergy ever. SFA and the ratio of n-6 to n-3 PUFA did not differ by allergy status.

To explore the age dependence of the association between allergy status and FA levels, we performed a spline regression of FA components on age and allergy (current vs no allergy ever). Figure 1 shows that children with current allergy had enhanced proportions of n-3 PUFA, n-6 PUFA and total PUFA (330 out of 2496 children). Particularly high proportions of n-3 PUFA were seen in allergic children below the age of 4 years (31 out of 381 children). Children with allergy had lower proportions of MUFA in the age intervals of 2–3.9 and 8–9.9 years (76 out of 460 children in the highest age interval).

The age-dependent trends observed in the raw data prompted us to analyze the association between FA and allergy status both in the total sample and by age category (Table 2). Overall, n-6

Table 1. FA variables and confounding factors by allergy status

	Allergy status		
	No allergy ever (ref.)	Former	Current
<i>N</i>	2166	98	330
	Mean (s.d.)	Mean (s.d.)	Mean (s.d.)
Age (years)	6.1 (1.8)	6.7 (1.7)***	6.5 (1.7)***
BMI z-score ¹⁶	1.00 (1.35)	1.16 (1.39)	1.02 (1.36)
	n (%)	n (%)	n (%)
Female sex	1067 (50%)	43 (44%)	146 (44%)*
Family history of allergic disease	26 (1%)	0 (0%)	10 (3%)*
Overweight and obesity	927 (43%)	44 (45%)	156 (47%)
Siblings (yes vs no)	1655 (79%)	80 (82%)	236 (74%)*
Breast-feeding ($\geq$ 4 months)	1041 (49%)	53 (55%)	136 (42%)*
<i>Fatty acids (wt%)</i>	Mean (s.d.)	Mean (s.d.)	Mean (s.d.)
PUFA	30.4 (2.9)	30.9 (3.2)	30.9 (2.9)*
n-6 PUFA	28.2 (2.8)	28.5 (3.0)	28.6 (2.7)*
n-3 PUFA	2.15 (0.63)	2.29 (0.64)*	2.21 (0.66)
MUFA	25.1 (2.5)	25.0 (2.5)	24.6 (2.3)***
SFA	44.6 (2.0)	44.1 (2.1)	44.6 (2.1)
n-6 PUFA/n-3 PUFA	14.3 (4.5)	13.4 (3.9)	14.1 (4.4)

Abbreviations: BMI, body mass index; FA, fatty acids; MUFA, monounsaturated FA; PUFA, polyunsaturated FA; SFA, saturated FA; wt%, weight percentage of total FA. Test of difference between children with former or current allergy compared with those with no allergy ever, adjusted for age, age², sex and country: * $P < 0.05$, ** $P < 0.01$ and *** $P < 0.001$.

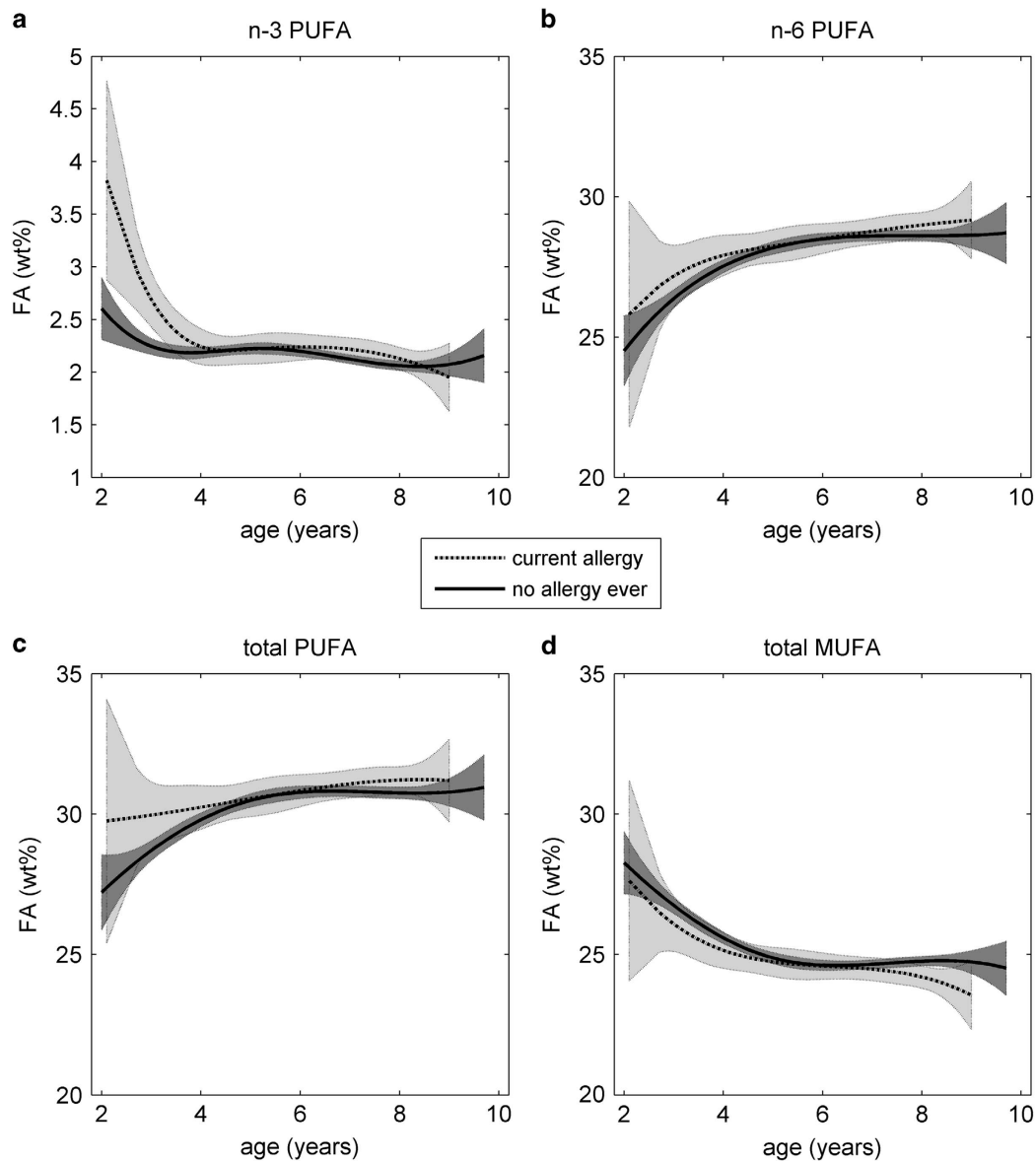


Figure 1. Regression of n-3 PUFA (a), n-6 PUFA (b), total PUFA (c) and total MUFA (d) on age, allergy and their mutual interaction (cubic splines with 5 knots, subjects with former allergy excluded).

Table 2. Prevalence of current allergy, and odds ratio of current allergy for an increase of FA by 1 s.d., overall and by age category (98 children with former allergies excluded)

	All ages ^a		Age categories ^b				
Age (years)	2–9.9	2–3.9	4–4.9	5–5.9	6–6.9	7–7.9	8–9.9
Total, n ^c	2414	370	365	287	392	560	440
Allergy, n (%) ^c	315 (13%)	30 (8%)	44 (12%)	34 (12%)	59 (15%)	80 (14%)	68 (15%)
n-6 PUFA	1.21** (1.05, 1.40)	1.54* (1.01, 2.34)	0.98 (0.71, 1.35)	0.97 (0.68, 1.37)	1.02 (0.77, 1.35)	1.16 (0.90, 1.49)	1.22 (0.91, 1.62)
n-3 PUFA	1.12 (0.98, 1.29)	1.62** (1.15, 2.29)	1.00 (0.73, 1.37)	1.08 (0.75, 1.56)	1.08 (0.82, 1.42)	1.09 (0.86, 1.39)	1.11 (0.86, 1.43)
MUFA	0.75*** (0.65, 0.87)	0.63* (0.42, 0.95)	1.04 (0.75, 1.43)	1.15 (0.80, 1.66)	0.87 (0.64, 1.18)	0.84 (0.65, 1.07)	0.60** (0.43, 0.84)
SFA	1.02 (0.90, 1.17)	0.80 (0.51, 1.24)	0.98 (0.69, 1.39)	0.86 (0.59, 1.26)	1.08 (0.85, 1.37)	0.99 (0.79, 1.25)	1.08 (0.83, 1.39)

Abbreviations: BMI, body mass index; FA, fatty acids; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; SFA, saturated FA. ^aLogistic regression of current allergy status on FA adjusted for age, age², sex, country, family history, BMI categories, number of siblings and breast-feeding: * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$. ^bLogistic regression of current allergy status on FA exposure followed by stepwise selection of potential confounders. ^cNumber of observations reduced because of missing values on specific covariates (breast-feeding, number of siblings).

Table 3. OR for current vs no allergy ever from logistic regression in different age intervals

Age (years)	Predictor	OR (95% CI)	P-value	AUROC
2–9.9	Age (years)	1.12 (1.04, 1.20)	0.003	0.63
	Number of siblings	0.85 (0.74, 0.98)	0.02	
	Breast-feeding	0.77 (0.61, 0.99)	0.04	
	Family history of allergic disease	2.65 (1.21, 5.80)	0.02	
	Oleic acid, 18:1 n-9	0.76 (0.65, 0.88)	0.0003	
	AA, 20:4 n-6	0.80 (0.68, 0.95)	0.01	
	DPA, 22:5 n-3	1.36 (1.19, 1.55)	< 0.0001	
2–3.9	Family history of allergic disease	8.12 (1.03, 64.3)	0.047	0.73
	Palmitoleic acid, 6:1 n-7	0.56 (0.34, 0.92)	0.02	
	DGLA, 20:3 n-6	1.54 (1.03, 2.28)	0.04	
	DPA, 22:5 n-3	1.62 (1.12, 2.35)	0.01	
4–4.9	Female sex	0.48 (0.25, 0.94)	0.03	0.59
5–5.9	—	—	—	—
6–6.9	Erucic acid, 22:1 n-9	1.31 (1.03, 1.67)	0.03	0.56
7–7.9	Mead acid, 20:3 n-9	1.36 (1.10, 1.69)	0.005	0.59
8–9.9	Age (years)	0.33 (0.13, 0.79)	0.01	0.63
	Oleic acid, 18:1 n-9	0.67 (0.50, 0.91)	0.009	

Abbreviations: AA, arachidonic acid; AUROC, area under the receiver operating characteristic curve; DGLA, dihomo γ -linolenic acid; DPA, docosapentaenoic acid; OR, odds ratio; 95% CI, confidence interval. Predictors were determined by stepwise forward selection from the set of 22 individual FA, age, sex, country, family history of allergic disease, BMI, breast-feeding and number of siblings.

PUFA was positively associated with allergy (OR=1.21 (1.05, 1.40) per increase by 1 s.d.) when adjusted for age, sex, country, BMI, breast-feeding, family history and number of siblings. In contrast, the odds of allergy were reduced by 25% for an increase in MUFA by 1 s.d. (OR= 0.75 (0.65, 0.87)). SFA was not associated with allergy status. There was no overall association between n-3 PUFA and current allergy. However, there was a strong positive association with n-3 PUFA in the youngest children (2–3.9 years) (OR=1.62 (1.15, 2.29)), and a weaker negative association with MUFA. MUFA was also negatively associated with allergy in the oldest age category (8–9.9 years). Stepwise selection of variables showed that lower MUFA rather than higher PUFA was associated with current allergy in the analysis combining all ages, and in the oldest children, whereas n-3 PUFA was the only FA associated with allergy in the youngest children. As n-3 PUFA consisted to 91% of LC-PUFA, regression results for the latter were similar to those of total n-3 PUFA (not shown).

Table 3 shows the result of regression models for current allergy using stepwise variable selection from 22 individual FA and potential confounders. In all ages combined, there were positive associations of allergy with age, family history and proportions of n-3 docosapentaenoic acid (n-3 DPA), whereas having more siblings, breast-feeding and higher proportions of oleic acid and AA were negatively associated with allergy. In the youngest age category, family history, n-3 DPA and dihomo γ -linolenic acid showed increased risk for allergy, whereas palmitoleic acid was associated with less allergy. In this age group, we obtained the best discrimination by allergy status (AUROC=0.73). Higher ages showed associations with n-9 FA only, both positively (erucic acid, mead acid) and negatively (oleic acid).

Post hoc analyses showed larger effect size for associations observed in the youngest age category when current allergy was restricted to diagnoses established before the age of two ($n=103$). N-3 PUFA was associated with higher odds of allergy (OR=2.27 (1.44, 3.56), AUROC=0.75). Stepwise selection of variables confirmed the association with n-3 DPA (OR=2.88 (1.73, 4.80)) and showed weak associations with vaccenic acid (18:1 n-7) (OR=0.52 (0.27, 0.99)) and family history (OR=15.3 (1.9, 124.9), AUROC=0.84) in the same age group.

Although the overall prevalence of allergy was higher in boys (Table 1), the associations between FA and allergy were stronger in girls, although not significantly so. Associations between PUFA and allergy were qualitatively similar in all countries, and higher than average associations were observed in countries with a high prevalence of allergies—that is, in Italy and Estonia. Overweight and allergy status were not mutually associated (Table 1), but the associations between current allergy and PUFA were stronger in overweight than in non-overweight subjects (P -value for interaction=0.01).

DISCUSSION

In this cross-sectional study of children aged between 2 and 9 years, the proportion of n-6 PUFA was higher in children with current allergy compared to children with no allergy ever. Lower proportions of MUFA were observed for current vs no allergy ever, whereas proportions of SFA, or the ratio of n-6 to n-3 PUFA did not differ by allergy status. The strongest association with allergy was seen in children between 2.0 and 3.9 years of age, where a difference in n-3 PUFA by 1 s.d. was associated with 62% higher odds of allergy. For 8–9.9 years of age, lower MUFA rather than higher PUFA was associated with current allergy. Among mutually adjusted FA, we observed positive associations between n-3 DPA and negative associations between oleic acid and allergy. The association with n-3 DPA was the main contribution to the positive association between total n-3 PUFA and allergy observed below the age of 4 years.

Studies investigating blood FA and atopic disease in childhood suggest a disturbed metabolism of essential FA,^{20–24} but there is no clear pattern or mechanism. PUFA has a range of effects on the immune system. Both n-3 and n-6 PUFA are strongly inhibitory regarding T-cell activation. AA serves as a substrate for eicosanoids, some of which are drivers of immunoglobulin E-mediated allergies.²³ N-3 and n-6 PUFA have also partly opposing properties. For example, high amounts of medium-chain n-3 PUFA can reduce the synthesis of AA through competition for enzymes for desaturation, or decrease the production of eicosanoids from AA.²⁴ Our findings of higher proportions of n-6 PUFA in allergic children, and no difference in

the n-6/n-3 ratio by allergy status, suggest that n-3 and n-6 PUFA have similar effects on allergy development in this age group. Although there was no overall effect of n-3 PUFA, we observed positive associations between n-3 PUFA and allergy in the youngest children. Similar associations were reported before,¹¹ and strengthened by observations of higher proportions of n-3 PUFA in cord serum of children with respiratory allergy at the age of 13 years compared to children without any allergy.²⁵ The authors suggested that the enhanced transfer of FA components with immune-dampening effects might hamper the maturation of the child's own immune system, and lack of early activation might increase the risk for allergy development. This interpretation is further supported by our observation that the association with n-3 PUFA was strongest in children with an early allergy diagnosis—that is, before the age of 2 years. Although the effect of enhanced maternal n-3 PUFA may have vanished after 2 years, it is possible that high levels were sustained through breast-feeding,²⁶ and that the decrease in n-3 PUFA levels observed here was paralleled with gradual weaning. It has been shown that the fat layer that is developed during the third trimester of gestation and that is particularly rich in docosahexaenoic acid (DHA) is maintained well into childhood.²⁷ It may also be that early differences by allergy status are maintained during infancy.

Although there were positive associations between n-6 PUFA and allergy in all ages combined, the association was not significant except in the youngest age group, as was the case with n-3 PUFA. It has been shown in animal models that PUFA is consumed during the allergic reaction.²⁸ This suggests that excess PUFA may not be observed in humans with acute allergy, except in young children as described above. Negative or missing associations with eicosanoid-related FA (AA, eicosapentaenoic acid and γ -linolenic acid) could be a sign of enhanced consumption of these FA during the allergic reaction, whereas some of their metabolites (n-3 DPA, dihomogamma-linolenic acid) show positive associations with allergy. Future analyses of seasonal allergies could shed more light on the association between specific PUFA and allergy in humans.

There is insufficient evidence showing that MUFA affects inflammatory processes.²⁴ In this study, palmitoleic and oleic acid were negatively associated with allergy, and these associations were also observed when adjusted for individual PUFA. Palmitoleic and oleic acids may be metabolized into n-7 and n-9 PUFA, respectively, competing with linoleic acid for the same enzymes,²⁹ which could lead to inverse associations with allergy. N-9 dietary FA have been suggested to have therapeutic effects on inflammation,²⁹ but we also observed a positive association between mead acid and allergy in children aged 7–7.9 years. The independence of effects of certain MUFA and PUFA in mutually adjusted models suggests genuine effects of MUFA that are not entirely due to opposite associations with PUFA, and all FA adding up to 100%.

This study was not specifically designed to study atopic disease, and the disease outcome was based on a physician diagnosis as reported by the parents. Physician-diagnosed allergy has been used for population cohorts of children before,³⁰ and the overall prevalence of 13% is comparable to epidemiological data for European countries.^{31,32} The outcome showed expected associations with variables such as sex, breast-feeding and family history of allergic disease.^{32,33} The lack of information on allergy subtypes was the main limitation of this study. The overrepresentation of overweight children in this study could lead to biased results as these children might have an altered FA profile as suggested by a positive association between PUFA and BMI. The associations reported here were indeed stronger in overweight than in non-overweight children, but the qualitative picture was similar in both groups. Because of the positive association between BMI and PUFA, one could assume that the association with atopy is strengthened owing to increased availability of PUFA in

overweight children in whom there might be an excess of substrate for synthesis of long-chain FA potentially promoting allergy development. Finally, the cross-sectional design did not allow the investigation of a causal relationship—that is, whether a disturbed FA profile precedes allergy development, or whether allergic disease entails changes in the FA profile. For instance, families with an allergic susceptibility might adapt their diet, which is reflected in the FA composition in blood.

CONCLUSIONS

Both PUFA subtypes were positively associated with allergy in an age-dependent manner, whereas MUFA was associated with less allergy. Regarding mutually adjusted individual FA, positive associations were found for n-3 DPA and negative associations for oleic acid. The observation of high proportions of n-3 PUFA in young allergic children might help to understand the nature of early onset of atopic disease.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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Supplementary Information accompanies this paper on European Journal of Clinical Nutrition website (<http://www.nature.com/ejcn>)

Mean values of FA (wt%)				
Nomenclature	Chemical nomenclature	No allergy ever (n = 2166)	Former allergy (n = 98)	Current allergy (n = 330)
Saturated fatty acids, SFA				
Palmitic acid, PAM	16:0	25.94 (1.53)	25.58 (1.54)	25.78 (1.50)
Stearic acid, STA	18:0	13.31 (1.00)	13.20 (1.00)	13.38 (1.01)
Arachidic acid	20:0	0.52 (0.08)	0.52 (0.07)	0.53 (0.08)
Behenic acid	22:0	1.78 (0.25)	1.78 (0.24)	1.82 (0.24)
Lignoceric acid	24:0	3.02 (0.43)	3.05 (0.44)	3.07 (0.43)
Mono-unsaturated fatty acids, MUFA				
Palmitoleic acid	16:1 n-7	1.31 (0.44)	1.33 (0.40)	1.27 (0.38)
Oleic acid, OA	18:1 n-9	18.84 (2.16)	18.70 (2.26)	18.33 (2.06)
Vaccenic acid	18:1 n-7	1.60 (0.34)	1.68 (0.37)	1.60 (0.32)
Paullinic acid	20:1 n-7	0.21 (0.05)	0.22 (0.06)	0.21 (0.05)
Erucic acid	22:1 n-9	0.08 (0.04)	0.09 (0.03)	0.09 (0.04)
Nervonic acid	24:1 n-9	3.00 (0.49)	3.01 (0.47)	3.05 (0.48)
Poly-unsaturated fatty acids, PUFA				
Mead acid	20:3 n-9	0.09 (0.04)	0.09 (0.05)	0.09 (0.04)
Linoleic acid, LA	18:2 n-6	18.01 (2.10)	17.73 (2.06)	18.26 (2.01)
γ-linolenic acid, GLA	18:3 n-6	0.23 (0.10)	0.24 (0.08)	0.24 (0.08)
Dihomo γ-linolenic acid, DGLA	20:3 n-6	1.22 (0.27)	1.28 (0.29)	1.24 (0.26)
Arachidonic acid, AA	20:4 n-6	7.38 (1.36)	7.77 (1.55)	7.48 (1.33)

Docosatetraenoic acid	22:4 n-6	1.04 (0.28)	1.14 (0.36)	1.07 (0.30)
Docosapentaenoic acid, n-6	22:5 n-6	0.28 (0.12)	0.31 (0.13)	0.28 (0.12)
DPA				
α -linolenic acid, ALA	18:3 n-3	0.20 (0.09)	0.20 (0.10)	0.21 (0.09)
Eicosapentaenoic acid, EPA	20:5 n-3	0.27 (0.11)	0.27 (0.11)	0.27 (0.11)
Docosapentaenoic acid, n-3	22:5 n-3	0.52 (0.16)	0.58 (0.18)	0.55 (0.17)
DPA				
Docosahexaenoic acid, DHA	22:6 n-3	1.17 (0.44)	1.23 (0.43)	1.18 (0.44)
