

Intake of unsaturated fatty acids and HDL cholesterol levels are associated with manifestations of atopy in adults

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Summary

Background The increase in allergic diseases is still unexplained. It was hypothesized that the intake of unsaturated fatty acids is a contributing cause of this development. We investigated the relationship between serum cholesterol levels, intake of polyunsaturated fatty acids (PUFA) and manifestations of atopy in a population-based setting.

Methods A nested case-control study was performed within the population of the 3rd MONICA survey in Augsburg (Germany). The serum levels of total, high-density lipoprotein (HDL) and low-density lipoprotein (LDL) cholesterol of 1537 adults (aged 28–78 years, response 61.4%) and the estimated intake of PUFA in a subset of 139 men were compared with the frequency of a doctor's diagnosis of asthma, allergic rhinoconjunctivitis (AR), atopic eczema (AE) and allergic sensitization as measured by skin prick and Radio Allergo Sorbent Test.

Findings In bivariate analyses, we obtained a negative linear association between total and LDL cholesterol levels and the frequency of AR and sensitization, which was no longer significant after adjustment for important confounders. In contrast, positive linear associations were found between HDL cholesterol levels and AR and AE and, furthermore, between the intake of PUFA and allergic sensitization in men ($P < 0.01$). After adjustment, an increasing risk for atopic diseases with increasing levels of HDL cholesterol and an increasing risk for allergic sensitization with increasing intakes of PUFA remained statistically significant.

Interpretation There is indication that HDL cholesterol also plays a role in the complex interaction of fat intake, metabolism and the manifestation of atopy in adults. These findings may contribute to the understanding of time trends and regional differences of allergies.

Keywords adults, allergic sensitization, allergy, cholesterol, epidemiology, PUFA

Submitted 25 January 2002; revised 24 January 2003; accepted 26 June 2003

Introduction

There is no doubt that atopic diseases (allergic rhinoconjunctivitis (AR), allergic asthma, atopic eczema (AE)) have increased significantly in industrialized countries over the past decades [1, 2]. The origin of this increase, however, is not known [3]. The rapid change in frequency cannot be explained by genetic factors and led to the study of environmental and ('western') lifestyle factors as possible contributing causes [4–7]. In this context, special attention was paid to nutritional factors, including fatty acids. The increase in allergies seems to coincide with an increased consumption of polyunsaturated fatty acids (PUFA) and a decreased intake of saturated fatty acids. PUFA and especially n-6 PUFA as precursors of pro-inflammatory

mediators and effectors on the microbial gut flora are discussed as promoters of allergic sensitization [8–10]. An imbalance of the PUFA composition with higher levels of linoleic acid (18:2 n-6) in atopic individuals was found by several clinical studies [11]. However, there is no convincing evidence of a δ -6-desaturase defect as a cause of the altered PUFA balance [12, 13].

Epidemiological studies in this area were mostly performed in children. In this context, the consumption of PUFA was discussed as a contributing cause of the increasing prevalence of hayfever in East Germany after the reunification [14, 15]. Population-based data on this association in adults are scarce [16] and have not systematically analysed various outcomes of the clinical manifestations of atopy.

If the association of PUFA intake and atopy held true in adults, we further speculated that cholesterol as a marker of a traditional diet should be inversely correlated with manifestations of atopy. With respect to cholesterol fractions, one could further hypothesize that high-density lipoprotein (HDL) cholesterol would be positively and low-density

Conflicts of interest: none.

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lipoprotein (LDL) cholesterol would be negatively associated with atopy. To our knowledge, population-based data on this issue are not available so far.

For that purpose, in a large population-based case-control study in adults, we investigated the association of blood lipid concentrations (total, HDL, LDL cholesterol), PUFA intake, and the presence of atopic diseases and allergic sensitization.

Methods

Study design

Here, we report on a nested case-control study, which was performed between September 1997 and December 1998 in Augsburg, Germany. Cases were defined according to the history of a doctor's diagnosis of atopic disease (AR, AE, asthma) or allergic sensitization as measured by the skin prick test (SPT) or RAST. Details of the sampling frame and study design are given in Fig. 1. The subjects were recruited from the 3rd MONICA (MONItoring of trends and determinants of CARDiovascular disease) survey, which was performed at

this place during the years 1994 and 1995. The objective and protocol of the MONICA study, which had started there in 1984, have been published earlier [17, 18].

The study base of the MONICA surveys consisted of all registered residents aged 25–74 years of the City of Augsburg and two adjacent counties. A random sample stratified for age and gender was studied in 1994/1995 ($n = 4356$). In the sera of these subjects, allergen-specific IgE antibodies to common aeroallergens (grass and birch pollen, house dust mite, cat, cladosporium) were determined by fluorescence-enzyme-immuno-assay technique (CAP-FEIA, Pharmacia, Uppsala, Sweden). Subjects were defined as being sensitized when they exhibited at least one positive finding (RAST class ≥ 1) to one of the allergens tested.

For the allergy study, we aimed to recruit an enriched sample of 1600 subjects out of the 4178 individuals with valid RAST results. Subjects were selected, stratified by age and sex, so as to provide 50% with and 50% without a positive RAST and furthermore so that, within these groups, 50% had given a positive answer to one of the following questions of the MONICA questionnaire: 'Have you had asthma attacks within the last 12 months?', 'Do you have allergic

Study design and sampling frame

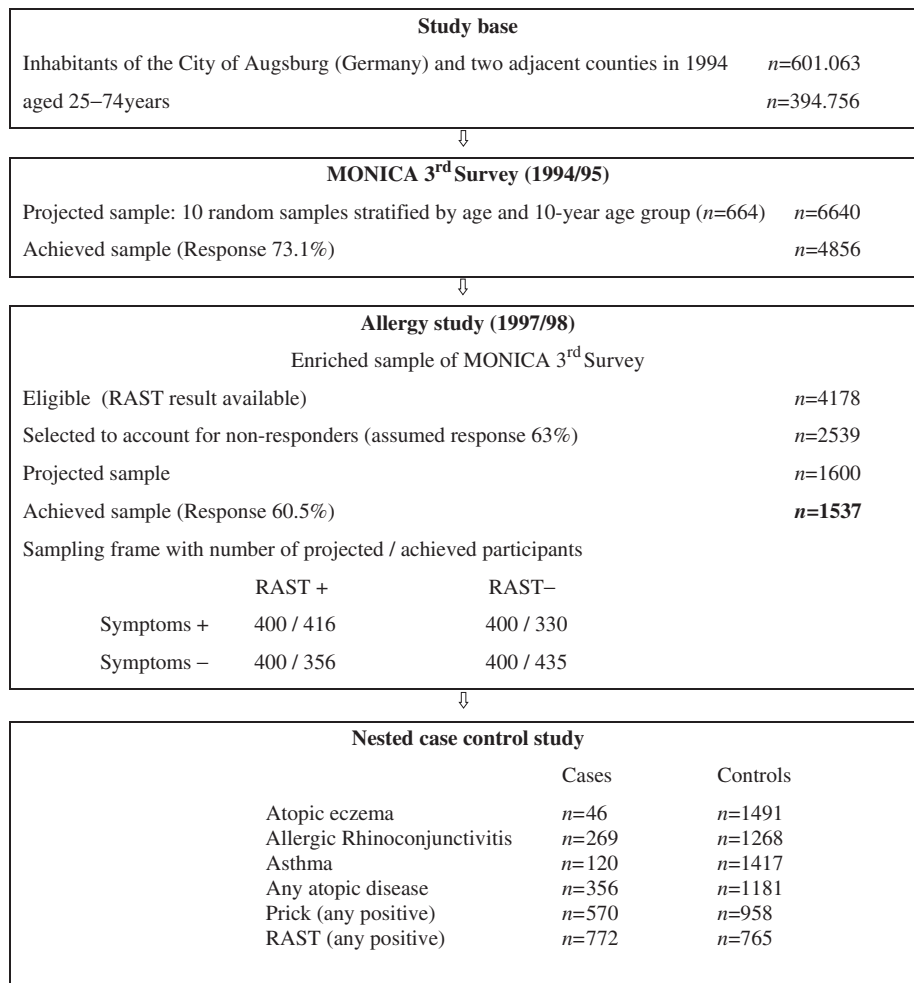


Fig. 1. Study design and sampling frame.

rhinitis, e.g. hayfever?', 'How much do you suffer from allergies/skin irritation?' (positive = moderate/severe). To account for an estimated response of 63%, a gross sample of 2539 subjects was approached. Finally, 1537 subjects participated (60.5%), of which 50.2% exhibited a positive RAST result and 53.9% of this group and 43.1% of those with a negative RAST result reported symptoms of atopy.

From all participants, a written and informed consent was obtained prior to the beginning of the study.

Outcome assessment

Laboratory analysis and food diary

Blood test results as well as food diary data were available from the previous MONICA survey.

Total cholesterol was analysed in non-fasting venous blood samples by the autoanalyser Hitachi 917 using the CHOD-PAP method (Boehringer, Mannheim, Germany). HDL cholesterol was measured in the same way after precipitation with Phosphotungstate/Mg²⁺ and LDL cholesterol, subsequent to precipitation with dextran sulphate (Quantolip, Fa. Immuno, Heidelberg, Germany). The total as well as HDL and LDL serum cholesterol levels were expressed in mg/dL. The continuous outcomes were transformed into categorical variables according to quartiles.

Data on a 7-day food diary were available from a subgroup of 139 men over 45 years of age. This selection followed the protocol of the 'MONICA optional Study on Nutrition' in order to study a high-risk population for cardiovascular diseases. The amounts of food were determined by weighing or by the use of models or measuring cups, or by the estimation in household measures. The coding was carried out on the basis of the 'Bundeslebensmittelschlüssel' (BLS version 2.1) and estimates of the mean daily intake of unsaturated fatty acids were given in g/day. Methodological details on the food diary have been published earlier [19].

Outcome assessment, sociodemographic parameters and parental history

As part of the case-control study, a computer-assisted standardized interview was carried out. We asked whether the person had ever been suffering from AR, asthma or AE; in case this question was answered positively, it was asked whether a doctor had diagnosed the disease. A doctor's diagnosis of allergic rhinitis, asthma and AE was taken as primary clinical outcomes. Information regarding the highest degree of school education as proxy for the socioeconomic status as well as information on the parental history of atopic diseases (allergic rhinitis, asthma, AE) were obtained.

Skin prick test

A conventional lancette SPT was performed on the volar aspect of the forearms. A sample of common aeroallergens (grass, birch, rye, mugwort pollen, alternaria, cladosporium, cat, dog, house dust mite) was tested, and saline and histamine were used as controls (ALK-Scherax, Hamburg, Germany). Results were read after 15 min and considered to

be positive when the weal diameter exceeded at least 2 mm. Results showing a positive reaction to saline or no reaction at all (including histamine) were excluded from further analysis. Subjects were defined as being sensitized when they exhibited at least one positive finding to one of the allergens tested.

Statistical analyses

Common descriptive parameters were given for continuous outcomes. Odds ratios (ORs) and 95% confidence intervals (CIs) were used as parameters of association and stability in 2 × 2 table comparisons. χ^2 trend tests were performed in 2 × N tables and the corresponding P-values are given. Group comparisons of continuous outcome parameters were carried out by non-parametric techniques (Mann-Whitney U-test). Multiple logistic regression models were used to test the impact of HDL cholesterol and PUFA intake on various dichotomous outcomes of atopy. Age, sex, parental history of atopy and school education were included as confounding variables in each model and, additionally, the total energy intake was included in the model for PUFA intake.

For logistic regression, incomplete data on parental history of atopy ($n = 475$) were coded as negative in order not to lose substantial statistical power. As part of the quality assessment of the study, about 90% of the interviews were tape recorded. A random evaluation of 20% of these recordings showed that negative answers were systematically labelled as 'missing' by some interviewers. Therefore, it seems justified to correct these data.

Adjusted ORs and 95% CIs are given. The calculations were carried out using the Statistical Package for the Social Sciences SPSS, release 10.0 and the Statistical Analysis System SAS, release 6.12.

Results

Description

Complete data were available from 1537 subjects, which is equivalent to a response of 61.4% with respect to the gross sample. According to the study design, half of the study group was female (50.4%) and also half of the study population was sensitized to at least one aeroallergen as measured by RAST (50.2%). The mean age was 50.5 years (median 50) and ranged between 28 and 78 years in the allergy study. A positive history of atopic diseases was given by 356 persons (23.2%) and with respect to single disorders, 46 (3.0%) reported AE, 269 (17.5%) allergic rhinitis and 120 (7.8%) asthma. The SPT was positive to at least one aeroallergen in 37.3% and a positive parental history of atopic diseases was obtained from 21.2% of the study population.

The mean values, standard deviations and ranges for the blood cholesterol parameters and the PUFA intake were as follows: total cholesterol (227.5, 41.9, 87.1–403.8 mg/dL), HDL cholesterol (55.1, 16.1, 20.0–139.4 mg/dL), LDL cholesterol (138.1, 41.1, 10.6–325.8 mg/dL), PUFA (15.2, 6.3, 6–58 g/day).

Mean values of cholesterol and PUFA intake in different outcome parameters of atopy

The mean values for cholesterol (total, HDL, LDL) and PUFA intake were compared with the history of atopic diseases (AE, allergic rhinitis, asthma) and allergic sensitization (SPT, RAST). Total cholesterol was found to be higher in non-atopic subjects. This finding was consistent in all signs of atopy, with the exception of asthma. Differences were found in the groups of 'allergic rhinitis' and 'allergic sensitization' (SPT and RAST). A similar pattern was observed when analysing LDL cholesterol levels. Non-atopic persons showed higher levels of LDL cholesterol than atopic individuals. This finding was also consistent for all groups of 'atopy', with the exception of asthma, and statistically significant for the groups of 'allergic rhinitis' and 'allergic sensitization' (SPT and RAST). An inverse association was observed for HDL cholesterol, which was found to be consistently higher in all groups representing manifestations of atopy, except for allergic sensitization measured by RAST. The differences were pronounced for subjects with AE, allergic rhinitis and allergic sensitization in the prick test. Subjects with manifestations of atopy also showed a higher intake of PUFA. This was true for all outcomes of atopy, except for AE. The differences were pronounced for allergic rhinitis and allergic sensitization (SPT and RAST) (Table 1). An analysis of these findings stratified by gender makes it clear that the results are mainly due to effects attributable to the group of women (Table 2).

Frequency of parameters of atopy in categories of cholesterol and PUFA intake

To test an unadjusted dose-response relation, the frequency of outcomes of atopy was determined within quantitative categories of cholesterol levels and PUFA intake. The frequency of atopic outcomes decreased in a linear manner with increasing total cholesterol levels. The corresponding trend tests were significant for allergic rhinitis and sensitization in SPT and RAST (Table 3). Similar trends were observed in the analysis of LDL cholesterol categories, with results being significant for AE, allergic rhinitis and sensitization in SPT (Table 4). In contrast, the frequency of atopy increased linearly with increasing categories of HDL cholesterol. These findings were significant for AE, allergic rhinitis and SPT reactivity (Table 5). With respect to the consumption of unsaturated fatty acids, the frequency of manifestations of atopy showed a positive association with increasing daily intake. Significant trends were observed for allergic rhinitis and sensitization, measured either by SPT or RAST (Table 6).

Regression analysis

After adjusting for important confounders (sex, age, parental history of atopic disease, school education), higher levels of HDL cholesterol were associated with an increased risk of atopy. This association was consistent for all atopic diseases and significant for AE, allergic rhinitis and any atopic disease

Table 1. Mean concentrations of total cholesterol, HDL and LDL cholesterol (mg/dL) and estimated daily intake of unsaturated fatty acids (g/day) by different outcomes of atopy

Outcome	Total cholesterol	HDL cholesterol	LDL cholesterol	Intake of unsaturated fatty acids, <i>n</i> = 139 men
Atopic eczema				
Yes (<i>n</i> = 46)	216.2	61.0	125.1	14.2
No (<i>n</i> = 1491)	227.9	54.9	138.5	15.3
<i>P</i>	0.09	<0.01	0.03	0.80
Allergic rhinoconjunctivitis				
Yes (<i>n</i> = 269)	222.5	57.8	132.7	17.6
No (<i>n</i> = 1268)	228.6	54.5	139.3	14.7
<i>P</i>	0.03	<0.01	0.02	0.01
Asthma				
Yes (<i>n</i> = 120)	230.1	55.7	142.2	16.4
No (<i>n</i> = 1417)	227.3	55.0	137.8	15.2
<i>P</i>	0.54	0.27	0.29	0.31
Any atopic disease				
Yes (<i>n</i> = 356)	224.5	57.5	134.8	16.6
No (<i>n</i> = 1181)	228.5	54.3	139.1	14.9
<i>P</i>	0.13	<0.01	0.09	0.07
Prick (any positive)				
Yes (<i>n</i> = 570)	223.8	56.2	134.4	16.2
No (<i>n</i> = 958)	229.8	54.3	140.5	14.5
<i>P</i>	<0.01	0.04	<0.01	<0.01
RAST (any positive)				
Yes (<i>n</i> = 772)	225.2	55.0	136.1	16.1
No (<i>n</i> = 765)	229.9	55.1	140.1	14.2
<i>P</i>	0.03	0.98	0.07	<0.01

HDL, high-density lipoprotein; LDL, low-density lipoprotein.

Table 2. Mean concentrations of total cholesterol, HDL and LDL cholesterol (mg/dL) by different outcomes of atopy and gender

Outcome	Total cholesterol		HDL cholesterol		LDL cholesterol	
	Women	Men	Women	Men	Women	Men
Atopic eczema						
Yes	210.8	228.4	63.9	54.4	118.9	139.3
No	226.4	229.4	61.4	48.4	134.2	142.9
P	0.06	0.92	0.29	0.045	0.06	0.67
Allergic rhinoconjunctivitis						
Yes	218.4	228.0	65.0	48.2	124.2	144.0
No	227.6	229.6	60.7	48.5	135.9	142.6
P	0.04	0.45	<0.01	0.97	<0.01	0.79
Asthma						
Yes	228.6	232.1	58.6	52.1	141.1	143.6
No	225.5	229.2	61.8	48.2	132.8	142.7
P	0.54	0.74	0.20	0.06	0.07	0.94
Any atopic disease						
Yes	220.9	229.2	63.6	49.3	128.4	143.5
No	227.5	229.4	60.8	48.3	135.4	142.6
P	0.13	0.73	0.04	0.36	0.07	0.94
Prick (any positive)						
Yes	219.6	228.6	62.9	48.6	126.7	143.2
No	229.9	229.8	60.6	48.5	138.2	142.6
P	<0.01	0.61	0.09	0.79	<0.001	0.75
RAST (any positive)						
Yes	221.8	228.5	62.1	48.2	129.2	142.8
No	229.5	230.5	61.0	48.7	137.7	142.8
P	0.01	0.58	0.37	0.95	<0.01	1.0

HDL, high-density lipoprotein; LDL, low-density lipoprotein.

Table 3. Frequency of outcomes of atopy by quartiles of total cholesterol (mg/dL)

Outcome	Total cholesterol (mg/dL)				P (trend test)
	I <200.3	II 200.3–224.2	III 224.3–254.5	IV >254.5	
Atopic eczema	4.4	2.6	2.9	2.1	0.083
Allergic rhinitis	19.8	19.4	15.4	15.4	0.047
Asthma	7.3	8.0	7.0	8.9	0.540
Any atopic disease	25.8	24.4	20.6	21.9	0.111
Prick (any positive)	41.2	39.3	36.0	32.6	0.009
RAST (any positive)	54.4	51.6	48.2	46.7	0.021

Table 4. Frequency of outcomes of atopy by quartiles of LDL cholesterol (mg/dL)

Outcome	LDL cholesterol (mg/dL)				P (trend test)
	I <109.65	II 109.65–136.3	III 136.4–165.2	IV >165.2	
Atopic eczema	4.4	3.4	2.1	2.1	0.034
Allergic rhinitis	19.8	18.6	16.9	14.7	0.048
Asthma	5.5	9.3	7.7	8.9	0.161
Any atopic disease	24.8	24.5	22.0	21.5	0.192
Prick (any positive)	42.6	37.1	35.6	34.2	0.016
RAST (any positive)	53.0	51.4	49.7	46.6	0.068

LDL, low-density lipoprotein.

(Table 7). Stratified analyses for gender, using gender-specific quartile limits of HDL cholesterol, revealed significant positive associations of HDL levels and AE in men and between HDL levels and allergic rhinitis in women.

Adjusting for the same confounders (except for sex and total energy intake in addition), the intake of PUFA showed a significant linear association with manifestations of atopy. Because diary data were available only for a subgroup, the corresponding number of cases with clinical manifestations of atopy was limited and it was not meaningful to test models

with single atopic diseases as a dependent variable. Significant results were obtained for allergic sensitization as measured by RAST (Table 8).

Discussion

Here, we report positive associations of HDL cholesterol levels and the intake of PUFA (men only) with various manifestations of atopy in adults.

Table 5. Frequency of outcomes of atopy by quartiles of HDL cholesterol

Outcome	HDL cholesterol (mg/dL)				P (trend test)
	I <43.6	II 43.6–52.2	III 52.3–64.0	IV >64.0	
Atopic eczema	1.0	2.6	3.1	5.2	0.001
Allergic rhinitis	12.9	18.9	16.1	22.3	0.004
Asthma	5.8	8.5	8.0	8.9	0.150
Any atopic disease	17.8	23.8	21.0	30.1	<0.001
Prick (any positive)	32.5	39.6	36.4	40.9	0.050
RAST (any positive)	48.6	53.1	49.7	49.2	0.900

HDL, high-density lipoprotein.

Table 6. Frequency of outcomes of atopy in men by quartiles of estimates daily food intake of PUFA

Outcome	PUFA (g/day)				P (trend test)
	I <12	II 12–14	III 15–17	IV >17	
Atopic eczema	5.1	3.2	0	5.4	0.872
Allergic rhinitis	5.1	19.4	12.5	32.4	0.005
Asthma	5.1	0	0	13.5	0.123
Any atopic disease	15.4	19.4	12.5	37.8	0.041
Prick (any positive)	28.2	41.9	46.9	56.8	0.012
RAST (any positive)	35.9	58.1	62.5	67.6	0.006

PUFA, polyunsaturated fatty acids.

We consider the following aspects of the design as strengths of the study. The case-control study was nested in a representative random sample of the general population. This allows a broader generalization of the results than previous hospital-based studies. The statistical power of the study is enhanced by the rather large sample size of 1537 subjects. This study was performed in adults (mean age 50.5 years), which offered the opportunity to test hypotheses (mainly studied in children to date) and to study outcomes that are not as common in young children (allergic rhinitis)

under conditions of a higher exposure variability (cholesterol levels). Multiple outcome manifestations of atopy in terms of disease and sensitization were obtained, which allowed one to study the internal consistency of these findings.

The data on cholesterol, PUFA intake and allergen-specific IgE antibodies were obtained about 3 years prior to the case-control study. The personal history of atopic diseases refers to a diagnosis ever made by a physician. Therefore, we cannot prove that changes in cholesterol levels and PUFA intake preceded the manifestations of atopy. We assume, however, that parameters of the fat metabolism have an impact on the occurrence of allergies and not vice versa. Assuming a true association, it is unlikely that misclassifications other than random and non-differential ones have occurred. This, however, would have biased the parameter of association towards the null.

The sampling procedure of the study included an enrichment of potential cases mainly by selecting half of the subjects as exhibiting allergen-specific IgE antibodies. This design may raise concerns as to whether the selection status should have been included in further analyses on other outcomes of atopy. We believed, however, that it is not justified to control in regression models for the presence of allergen-specific IgE antibodies when analysing highly correlated outcomes (sensitization in the prick test, atopic diseases) as a dependent variable. To further clarify this issue, the same logistic regression models as presented in Tables 7 and 8 were calculated with additional adjustment for selection status (i.e. presence of allergen-specific IgE antibodies). All results that turned out to be significant in the previous models remained significant in these analyses. The effect estimates were almost identical to moderate changes only in the model analysing PUFA intake and SPT reactivity. We therefore concluded that selection status did not modify the results and that the data are presented adequately.

There is evidence that the level and intake of PUFA is associated with atopic disorders. It is more than 60 years since Hansen [20] reported reduced plasma levels of essential fatty acids in patients with eczema and proposed an abnormal metabolism. Based on epidemiological studies, a correlation between the intake of unsaturated fatty acids or margarine and the prevalence of atopic diseases in children was reported [14, 15, 21]. A significant association between margarine consumption and allergic sensitization and rhinitis symptoms

Table 7. Multiple logistic regression models for different outcomes of atopy and HDL cholesterol (adjusted* odds ratios and 95% confidence intervals)

Outcome	HDL cholesterol (mg/dL)			
	I <43.6 (ref.)	II 43.6–52.2	III 52.3–64.0	IV >64.0
Atopic eczema	1.00	2.06 (0.63–6.73)	2.23 (0.68–7.32)	3.51 (1.10–11.18)
Allergic rhinitis	1.00	1.45 (0.97–2.19)	1.12 (0.73–1.73)	1.59 (1.03–2.44)
Asthma	1.00	1.47 (0.83–2.59)	1.27 (0.70–2.29)	1.35 (0.74–2.47)
Any atopic disease	1.00	1.33 (0.93–1.92)	1.05 (0.72–1.53)	1.62 (1.11–2.36)
Prick (any positive)	1.00	1.28 (0.94–1.74)	1.05 (0.76–1.44)	1.21 (0.87–1.68)
RAST (any positive)	1.00	1.21 (0.90–1.61)	1.06 (0.79–1.43)	1.05 (0.77–1.43)

*Adjusted for sex, age, parental history of atopic disease, school education.
HDL, high-density lipoprotein.

Table 8. Multiple logistic regression models for different outcomes of atopy and estimated daily intake of PUFA fatty acids (adjusted* odds ratios and 95% confidence intervals)

Outcome	PUFA (g/day)			
	I <12	II 12–14	III 15–17	IV >17
Total cholesterol (mg/dL)	239.1	246.7	230.1	246.1
Any atopic disease	1.00	1.06 (0.28–4.04)	0.67 (0.16–2.84)	2.90 (0.87–9.73)
Prick (any positive)	1.00	1.52 (0.54–4.44)	1.99 (0.70–5.65)	2.67 (0.94–7.57)
RAST (any positive)	1.00	3.05 (1.07–8.65)	3.81 (1.34–10.88)	4.84 (1.65–14.22)

*Adjusted for age, parental history of atopic disease, school education, energy intake (kcal).
PUFA, polyunsaturated fatty acids.

was observed in a population-based group of boys [22]. Recently, a European study reported a positive association of the intake of monounsaturated fatty acids and allergic sensitization in adults [16]. The intake of n-6 PUFA was also found to be positively associated with reported symptoms of AR in Japanese women [23].

Detailed analyses of PUFA composition in the sera of atopic children or their breast milk diet revealed a tendency towards higher levels of n-6 PUFA (linoleic acid) and metabolites (arachidonic acid, PGE2) and corresponding lower levels of n-3 PUFA (linolenic acid) and metabolites (eicosapentaenoic acid, LTB5) as compared with non-atopic children [11, 13, 24, 25]. It is speculated that disturbances in the composition of PUFA and their long-chain metabolites may modify the metabolic pathway of pro-inflammatory substances (prostaglandins, leukotriens) and also affect cell membrane functions [9, 11, 26]. According to a double-blind controlled trial, eicosapentaenoic acid seems, however, not to be clinically effective in the treatment of AE [27]. In the context of these findings, an impairment of the δ -6-desaturase activity was not confirmed, and the effect of dietary supplementation of PUFA in order to treat atopic disorders remains controversial [9].

Our findings are limited by the fact that data on the consumption of unsaturated fatty acid were available only for a subgroup of men and that data of the composition of the fatty acids are not available. The positive association of PUFA consumption and atopy, however, is consistent with previous reports and extends this observation to the group of adults. We were furthermore able to demonstrate a dose-response relationship that held true for allergic sensitization in multiple logistic regression analysis and points to a possible causal relationship.

Little is known on the possible associations between allergic diseases and cholesterol levels. We found no stable association of total and LDL cholesterol levels with manifestations of atopy. HDL cholesterol levels were dose dependently positively associated with various outcomes of atopy. The associations with respect to atopic diseases remained significant in multiple logistic regression analysis. The association with asthma was also positive and more pronounced in men, but did not reach statistical significance. These associations have, to our knowledge, not been tested in a population-based study in adults so far. The findings on HDL cholesterol, however, are consistent with a previous report from India [28], where total cholesterol was found to be significantly lower and

HDL cholesterol significantly higher in children with respiratory allergy as compared with other paediatric patients.

Following the previous considerations, these findings support the hypothesis that fat consumption and metabolism play a role in the manifestation of allergies. Thereby, PUFA reflects a rather modern diet as part of the western lifestyle, which possibly contributes to the increase in allergies in these regions. Total and LDL cholesterol, in contrast to HDL cholesterol, indicates a traditional diet that may contribute to the explanation of the rather low frequency of allergies in the former GDR.

The underlying metabolic mechanisms, however, remain to be elucidated.

Acknowledgements

We would like to thank all participants of the study. We are indebted to the survey team, the interviewers and technicians, and K. Papke, B. Filipiak, Dr B. Giesecke and Dr R. Holle from the GSF-National Research Centre for Environment and Health. We would also like to acknowledge the contribution in logistics of the B. Schwertner Company. This study was supported by a governmental funding source: German Ministry of Education, Science, Research, and Technology (Bundesministerium für Bildung, Wissenschaft, Forschung und Technologie, 01ER9502).

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