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Associations among eczema, asthma, serum immunoglobulin E and depression in adults: a population-based study

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Keywords: Asthma; atopy; depression; eczema; immunoglobulin E.

Atopy or IgE-mediated immune reaction has been suggested to be related to the link between allergic diseases and depression (1). Previous studies, however, have rarely addressed the issue of whether serum total IgE levels could explain the associations between allergic diseases and depression. In addition, little is known about the associations of depression with atopic and nonatopic phenotypes of eczema and asthma.

Serum total IgE level and IgE sensitization do not explain the associations of depression with eczema and asthma.

To answer these questions, we conducted a cross-sectional, population-based study. Subjects (n = 4052) were from the National Health and Nutrition Examination Survey 2005–2006, aged ≥20. The presence of depression was assessed by the Patient Health Questionnaire. Serum total and allergen-specific IgE levels were measured, and atopy was defined as having a positive specific IgE test to at least one of 19 inhalant and food allergens. The presence of eczema and asthma was ascertained by questionnaires. Atopic eczema was defined as the presence of both eczema and a positive specific IgE test to at least one of 19 inhalant and food allergens; cases of nonatopic eczema were people with eczema but with no IgE sensitization to any of the inhalant or food allergens tested. Atopic asthma was defined as the presence of both asthma and a positive specific IgE test to at least one of the 15 inhalant allergens, while cases of nonatopic asthma were people with asthma but with no IgE sensitization to inhalant allergens. Potential confounders such as body mass index, poverty income ratio, marital status, educational level, alcohol assumption, and chronic medical illnesses were obtained by questionnaires. Serum cotinine level was measured as an indicator of smoking status. Logistic regressions were used to estimate the associations. Because the distribution of serum total IgE level in the population was right skewed, natural log-transformed values were used to provide the best-fitting model for the analysis.

Of the study population (n = 4052, mean age 46.8 years), 44% of the participants had prevalent atopy. There was no association between atopy and depression, and between serum total IgE levels and depression (Table 1). Both atopic eczema (OR, 2.06; 95% CI, 1.12–3.78) and nonatopic eczema (OR, 2.30; 95% CI, 1.29–4.09) were significantly associated with an increased likelihood of depressive disorders after controlling for potential confounders, asthma and total IgE levels. Only atopic asthma was significantly associated with depression (OR, 1.81; 95% CI, 1.03–3.18) after controlling for potential confounders, eczema and total IgE levels.

Our data suggested that the association between eczema and depression was not explained by the IgE-mediated immune reaction. The comorbidity of depression with eczema might be related to the chronic pruritic dermatosis, which leads to sleep disturbances, anxiety and depression (2, 3).

In contrast to eczema, the association between asthma and depression was significant only in the atopic asthma phenotype; however, this difference might not be attributable to IgE sensitization *per se*, because there was no association between aeroallergen sensitization and depression (Table 1). Besides, the association between atopic asthma and depression was independent of total IgE levels. One possible explanation for the association with depression only being evident in patients with atopic asthma might be the earlier age of onset of atopic asthma compared to that of nonatopic asthma (4). There is evidence that early onset of asthma and hospitalization in childhood is associated with liability to depression in later life (5, 6).

In conclusion, our study suggested that IgE-mediated mechanisms do not explain the relationship between eczema (or asthma) and depression. The causal pathways between allergic diseases and depression, as well as the mechanisms of differential association of depression with atopic and nonatopic asthma require further investigation.

Table 1 Associations of depression with atopy, eczema, asthma, and serum total IgE level

Variables	Value	Univariate analysis OR (95% CI)	Multivariate analysis OR (95% CI) Model 1*	Model 2†
Atopy‡, (%)				
No	56.5	1.0 [Reference]	1.0 [Reference]	1.0 [Reference]
Yes	43.5	1.01 (0.83–1.24)	1.08 (0.85–1.37)	0.99 (0.74–1.31)
Any IgE sensitization to inhalant allergens, (%)				
No	58.2	1.0 [Reference]	1.0 [Reference]	1.0 [Reference]
Yes	41.8	0.92 (0.73–1.16)	0.98 (0.75–1.27)	0.89 (0.67–1.19)
Eczema phenotype, (%)				
Never	89.6	1.0 [Reference]	1.0 [Reference]	1.0 [Reference]
Nonatopic	5.1	2.09 (1.35–3.25)	2.26 (1.31–3.89)	2.30 (1.29–4.09)
Atopic	5.3	1.81 (1.10–2.97)	2.17 (1.21–3.81)	2.06 (1.12–3.78)
Asthma phenotype, (%)				
Never	85.9	1.0 [Reference]	1.0 [Reference]	1.0 [Reference]
Nonatopic	5.3	1.88 (1.13–3.13)	1.45 (0.87–3.25)	1.45 (0.87–2.28)
Atopic	8.8	1.82 (1.07–3.10)	1.81 (1.04–3.14)	1.81 (1.03–3.18)
Serum total IgE (kU/l), median (IQR)	46.0 (111.9)	1.07 (0.99–1.16)§	1.01 (0.91–1.12)§	0.98 (0.89–1.09)§

CI, confidence interval; IQR, inter-quartile range; OR, odds ratio.
*Adjusted for age, gender, race, educational level, marital status, poverty income ratio, body mass index, alcohol consumption, serum cotinine level, and chronic medical illnesses (arthritis, diabetes mellitus, heart disease, hypertension and emphysema).
†Adjusted for all covariates in model 1, plus asthma, eczema, and serum total IgE levels.
‡Atopy was defined as having any IgE sensitization to inhalant or food allergens.
§ORs for depression for each unit increase in the natural log-scaled serum total IgE level.

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Atopy susceptibility and chromosome 19q13

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Keywords: 19q13; atopy; IgE; microsatellite; susceptibility.

Atopy and atopic disease such as asthma show strongly familial characteristics, with heritability estimates varying between 36% and 79% (reviewed in Ref. (1)). Chromosomal region 19q13.1–13.3 has been identified as containing genes predisposing to atopy or asthma-related phenotypes (reviewed in Ref. (2)). A region at ~58 Mbps has shown linkage to atopy and IgE phenotypes in a study of 111 Italian Caucasian families (3); however, the linkage peak was broad spanning several megabases. The aim of the current study was to replicate and further define this potential atopy locus using two independent UK cohorts.

We have replicated and further defined an atopy susceptibility locus on chromosome 19q13.

In the first analyses, we utilized the Southampton asthma sibling pair cohort (n = 341 families) which has been extensively described (2) using total IgE, elevated specific IgE (0.35 kU/l) and positive skin prick test (> 3 mm diameter, SPT) phenotypes. Five microsatellite markers spanning the region D19S402 (56.9 Mbp), D19S601 (57.3 Mbp), D19S571 (57.9 Mbp), D19S180 (58.4 Mbp) and D19S572 (58.8 Mbp) were genotyped as described (2). Errors in genotyping were detected using the inheritance check in Family-Based Association Test (FBAT) software (4). Global FBAT analyses (additive model) identified association between D19S571 and number of specific IgE responses [spIgE(no.)] with borderline significance for total IgE levels (tIgE) and positive specific IgE (spIgE+) (Fig. 1A). Marker