Understanding the concept of 'family history' in black asthmatic children

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Since 1992 the atopic status of asthmatic children attending the Chris Hani Baragwanath Hospital (CHBH) paediatric asthma clinic has been investigated.1,2 This is an almost entirely urban population (97.5%) who have been resident in the greater Soweto area their entire lives. We found that 71.6% of the children had one or more positive skin prick tests (SPTs) to common aero-allergens,3 confirming our belief that these children have a similar atopic diathesis to other groups reported.1 This is contrary to the existing perception that black asthmatic children in South Africa are not atopic.3

Additional findings were a 45.1% positivity rate to house dust mite (HDM) (Dermatophagoides pteronyssinus). This was an unexpected finding and far exceeded previous reports in patients living at high altitude.4 In addition these children had never lived or even visited the coast where sensitisation could have taken place. This study was followed by an analysis of mite antigen levels in the homes of the asthmatic children. Very low levels of mite antigen were found in mattress and floor dust.5 These levels were generally lower than the 2 µg/g required for sensitisation.6 Certainly in these children, it would appear that sensitisation to HDM had occurred on exposure to very small quantities of HDM antigen (Der p 1). No solid explanation for this finding has ever been proposed.

There has been a worldwide increase in the prevalence of atopic disease.7,8 Many environmental factors have been postulated to explain this trend, including urbanisation, dietary changes, changes in microbial burden and industrial pollution.9,10 On the other hand, atopic disease expression carries an inherited or genetic component, and the literature suggests that the most reliable way of determining this inherited tendency is to seek a positive family history of an atopic condition.11

Lyt et al. reported a low rate of positive family history for atopic conditions (22.2%).1 This has traditionally minimised the importance of family history in determining the nature of chest symptoms in children in this population group. In fact, some experts have gone as far as to suggest that some new environmental exposure was creating the allergic milieu in this group and that these children were expressing a de novo atopic condition. Among the suggested aetiologic factors, urban pollution has always been touted as promoting the development of allergy. However, most of the parents of the children studied felt that they did not live in a polluted environment, and this is supported by other South African (and international) studies which do not show an association between pollution and increased incidence of allergy.12-14 A change in the level of allergen exposure is also thought not to be the reason for the rising prevalence in this area.

Atopic conditions impact significantly on quality of life, and in a familial disease pattern this impact is felt by all members of the family. In children who have typical chest and nasal symptoms suggesting an allergic diathesis, a positive family history of an atopic condition is often the diagnostic clincher, especially in a country such as South Africa where medical resources are limited and special investigations are not available to the majority of the population. This study set out to determine if and how the question of 'family history' should be used in a practice setting.
In order to test the value of a family history in determining inheritance of atopy we explored the results of our previous studies.

**Objectives**

This study had two foci. Firstly, it sought to determine the predictive value of a family history of symptoms of atopic disease (and allergy) by seeking evidence for this condition in the parents of asthmatic children attending the CHBH Children’s Asthma Clinic. Secondly, it investigated the reason(s) why a positive family history was so seldom found in these children.

**Methods**

A random group of parents of the atopic asthmatic children attending the CHBH Children’s Asthma Clinic were identified and approached to complete a detailed questionnaire regarding their atopic status. In order to randomise the clinic patients adequately, every fourth patient file was selected from an alphabetical list of patients. These parents were contacted telephonically where possible. Only 40% of selected families were contactable, and even fewer responded to the study. The questionnaire included questions on birth history, medical history, symptomatology of atopy, occupation, habits, present environment, and family history. The parents were the biological parents of the children.

Skin-prick testing was done using the Hollister-Stier allergen extracts (Bayer Miles), and negative (0.5% phenol) and positive (1% histamine) controls were performed. The aero-allergen extracts used were Bermuda grass, corn pollen, 5-grass mix, tree mix, *Candida albicans*, *Aspergillus fumigatus*, cat-hair epithelium, dog-hair dander, feather mix, house-dust mix, and standardised *D. pteronyssinus*. Each extract was applied to the volar surface of the forearm with a sterile prick lancet and standardised prick test (SPT) POSITIVITY AS A SUBGROUP OF ALL SPT POSITIVITY IN MOTHERS

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<tr>
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<th>Positive SPT (%)</th>
<th>Negative SPT (%)</th>
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<tr>
<td>HDM-positive</td>
<td>28 (74)</td>
<td>41 (62)</td>
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<tr>
<td>HDM-negative</td>
<td>10 (26)</td>
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With regard to the clinical manifestations of an atopic status, analysis of asthma prevalence revealed that 7 of 69 mothers (10%) had a definitive diagnosis of asthma. Of these, 5 (71%) were SPT-positive. Only 3 of the fathers (5.5%) had asthma. All had a positive SPT. However, if all the symptoms suggestive of asthma, namely chronic cough or wheeze, were taken into account, the number of potential asthmatics increased to 18 mothers (26%) and 10 fathers (19%) (Table III). All parents were asked specifically about symptoms suggestive of asthma and rhinitis, including chronic cough and wheeze.

**Statistical analysis**

Frequencies of each skin test, odds ratios, chi-square tests (where appropriate), and Fisher’s exact tests were run using SAS.

**Results**

Fifty-four sets of parents and 15 single mothers were studied. Forty of the 54 parent sets (74%) had an atopic child, while 8 of the 15 single mothers (53%) had an atopic child. Of the 48 atopic children, 37 (77%) had at least 1 parent with a positive SPT ($p = 0.093$, significance at $\alpha = 0.1$) (Table I).

Fifty-five per cent of mothers had a positive SPT, while 48% of fathers had at least 1 positive SPT. Of special interest in this study was the high prevalence of HDM SPT positivity among parents. Twenty of 26 SPT-positive fathers (77%) had a positive HDM result, while 28 of 38 mothers (74%) had a positive HDM result (Table II).

With regard to rhinitis, 26% of mothers and 24% of fathers had symptoms of chronic rhinitis, but none were on therapy. Twelve of the 18 mothers (67%) had allergic rhinitis (SPT-positive), while 69% of fathers had allergic rhinitis (Table IV). However these data are often not reported in ‘family history’ questionnaires.

### Table I. Parents of Asthmatic Children ($N = 69$)

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<tr>
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<th>Atopic (either or both) parent/s (%)</th>
<th>Non-atopic parent (%)</th>
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<tr>
<td>Atopic children</td>
<td>37 (54)</td>
<td>11 (16)</td>
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<tr>
<td>Non-atopic children</td>
<td>12 (17)</td>
<td>9 (13)</td>
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### Table II. House Dust Mite (HDM) Skin-Prick Test (SPT) Positivity in Definitively Diagnosed Asthmatic Parents

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<tr>
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<th>SPT-positive (%)</th>
<th>SPT-negative (%)</th>
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<tr>
<td>Definitive diagnosis of asthma (mothers)</td>
<td>5 (71)</td>
<td>2 (29)</td>
</tr>
<tr>
<td>Definitive diagnosis of asthma (fathers)</td>
<td>3 (100)</td>
<td>0 (0)</td>
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Other South African (and international) studies do not show an association between pollution and increased incidence of allergy.
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All the diagnosed asthmatic parents were born in urban areas, and of those born in rural areas, none had asthma, providing support for the ‘hygiene hypothesis’ of allergy development.10

Discussion

There is a high prevalence of allergy (SPT-positivity) in the parents of atopic asthmatics, but because asthma and allergic rhinitis are seldom diagnosed in this population group, family history of an atopic disease is often absent. Simple questioning for family history of atopic disease is not therefore a good predictor of atopy in offspring in this cohort of patients. It seems obvious that this important aspect, viz. an initial history of a family history of an atopic disease is often absent. Simple questioning for family history of an atopic disease is not therefore a good predictor of atopy in offspring in this cohort of parents and their atopic asthmatic children.

This study found no association between sex of the atopic parent and a higher risk of atopy in children, as has been suggested for other races.11 Once again the prevalence of HDM SPT-positivity was found to be high and unexplained in this population living at high altitude, without previous travel to the coast. Some of the questions on origins of allergy and atopic disease may be unravelled through further study of this cohort of parents and their atopic asthmatic children.

References


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