

Inhalant Allergies in Zimbabwe: A Common Problem

Elopy Nimele Sibanda

Department of Immunology, University of Zimbabwe College of Health Sciences, Harare, Zimbabwe

Key Words

Rhinitis · Inhalant allergy · Allergen sources · Zimbabwe

Abstract

Allergic rhinitis, allergic conjunctivitis, allergic asthma and atopic dermatitis affect a quarter of the population of the industrialised countries and are the most common symptoms of type I hypersensitivity reactions. Their prevalence has been documented in many communities, yet data from Africa are limited. In the 5-year period from September 1997 to September 2002, approximately 14,000 patients of all ages were referred to the only specialist allergy clinic in Harare, Zimbabwe, for allergy investigation, diagnosis and therapeutic management. In approximately one tenth of these patients, food allergies were diagnosed; less frequent presentations included allergic pharyngitis, bee- or wasp-induced reactions and latex allergy. An allergologic basis for the clinical conditions was established following a careful clinical and physical examination, evaluation of family history and the detection of in vivo (skin prick test) or in vitro (radio-allergo-sorbent test; RAST) allergen-specific IgE antibodies to known sources of inhalant allergens, using commercial allergen extracts and a RAST assay kit. Asthma diagnosis was supported by the clinical and laboratory findings of salbutamol-reversible airways obstruction. Case files of the first 1,046 patients are reviewed in order to define the clinical presentation of allergic patients in

this region. It is highlighted that allergy in general and inhalant allergy in particular are very common, if not widely acknowledged, clinical problems in this Central African region.

Copyright © 2003 S. Karger AG, Basel

Introduction

The extent of and triggers for allergic diseases within Africa are unknown. This may be a consequence of a combination of two or more interrelated factors. First, the diagnosis of allergy requires clinical assessment and appropriate laboratory testing undertaken by suitably trained and experienced personnel. Unfortunately, in Africa there are only a few physicians who are specifically trained in the diagnosis and management of allergic diseases and the laboratory infrastructure is also inadequate. The relevant specialties of allergology and clinical immunology are not well developed, and as a consequence, allergy has not been studied systematically [1]. Even where personnel and diagnostic facilities are available, the diagnostic reagents used are expensive and often not tailored for the peculiar fauna and flora of Africa. Reliance on elevated total serum IgE levels as surrogate indicators of allergy is complicated by the high prevalence of helminth infections, which may be associated with elevated antibody levels in the absence of atopy. Apart from the diagnostic constraints, differences in the environment,

KARGER

Fax + 41 61 306 12 34
E-Mail karger@karger.ch
www.karger.com

© 2003 S. Karger AG, Basel
1018–2438/03/1301–0002\$19.50/0

Accessible online at:
www.karger.com/iaa

Correspondence to: Dr. Elopy Nimele Sibanda
Department of Immunology
University of Zimbabwe College of Health Sciences
PO Box A178, Avondale, Harare (Zimbabwe)
Tel. +263 4 791631, Fax +263 4 303345, E-Mail esibanda@africaonline.co.zw

ethnicity and in particular allergen sources contribute to variations in the prevalence of allergy. These factors contribute to a paucity of data on atopic diseases in Africa.

Recently, the International Study of Asthma and Allergy in Childhood [2] was completed, followed by a number of epidemiological, mainly paediatric studies [3–5] which show that the prevalence of asthma in particular, and other allergies in general, is high in many African communities, and that the incidence tends to be higher in urban than in rural environments [6, 7]. In the light of the currently topical hygiene hypothesis [8], it has been argued that the African tropical environment with the attendant high prevalence of infectious diseases therein somehow makes atopic reactions less common. While these arguments, which are supported by sound immunological explanations, may be true for the populations studied, they could still be unrepresentative of the yet to be studied reality in the large and heterogeneous African continent.

This report, based on a 1,046-person sample out of 14,000 patients whose allergy symptoms were severe enough to warrant referral to a Specialist Allergy Clinic, summarises the current impact/incidence of allergy in Zimbabwe and highlights the high prevalence of allergic diseases and locally relevant allergen sources. The findings may reflect the pattern and prevalence of allergen sensitisation in other African countries.

Patients and Investigations

The case records of the first 1,046 of a total of 14,000 patients referred to a Specialist Allergy and Clinical Immunology practice in the Zimbabwean capital, Harare, were analysed. All patients had symptoms of allergic diseases and were referred for confirmation of allergen sensitisation, identification of possible allergen sources and requisite management. Eight hundred and three of the patients were atopic, as defined by the demonstration of immediate type I IgE antibody-mediated reactivity to allergen extracts in genetically predisposed patients. The patients' age, predominant presenting complaints, total and allergen-specific IgE antibody levels and skin prick test (SPT) results were analysed.

Clinical Assessment

An in-house organ- and symptom-specific questionnaire was routinely administered during the course of a clinical evaluation. Data relating to the type, frequency and severity of symptoms referable to the upper or lower respiratory tract, the eyes, the skin or gastro-intestinal

tract, triggering factors and a history of the type and effectiveness of medicines taken were systematically captured. Personal and/or family history of atopy was also recorded. Based on the questionnaire and on clinical examination, the patients were offered the clinically relevant and available diagnostic tests. SPTs, epicutaneous patch tests and the identification of allergen-specific IgE antibodies by radio-allergo-sorbent tests (RAST) were routinely offered. Lung function was assessed by spirometry before and/or after nebulisation with salbutamol. Nasal or respiratory challenge tests were only infrequently offered.

In vivo Tests

Alpyral (Bayer Allergy, Spokane, Wash., USA) extracts were used for the identification of inhalant allergen sources by SPT. The extracts used were the following: house dust mites (*Dermatophagoides pteronyssinus*), Bermuda grass (*Cynodon dactylon*), grass mix, tree mix, mould mix (*Alternaria*, *Aspergillus*, *Penicillium*), feather mix (chicken, goose, duck) and animal dander (cat, dog). Allergopharma (Allergopharma, Reinbek, Germany) extracts were used to investigate sensitisation to individual mould and food allergen sources, while ALK (ALK-Abelló, Hørsholm, Denmark) extracts were used to demonstrate bee or wasp venom allergy. Patients with minimum wheal diameters of 4 mm after 20 min of SPT were classified as reactive. The Standard European 23 epicutaneous patch test panel (Trolab, Germany) was used in the investigation of dermatitis or occupational allergy.

In vitro Tests

Sensitisation to allergen sources was demonstrated using commercial allergen extracts. Positive (10 mg/ml histamine) and negative (glycerinated saline) controls were included in the battery of tests.

An enzyme immunoassay (Abbott Matrix Analyser, Abbott Diagnostics, USA) and the Pharmacia CAP system (Pharmacia, Uppsala, Sweden) are routinely used to determine levels of allergen-specific IgE antibodies. Total IgE is measured by serological methods.

Social and Demographic Characteristics of the Patients

The patients were born between the years 1937 and 1999, and ages at the time of investigation ranged from 1 to 62 years. Approximately two thirds were female. The age distribution by decade is shown in table 1, while the age by year of birth is shown in figure 1. The majority of the patients were from Harare, with only a small percent-

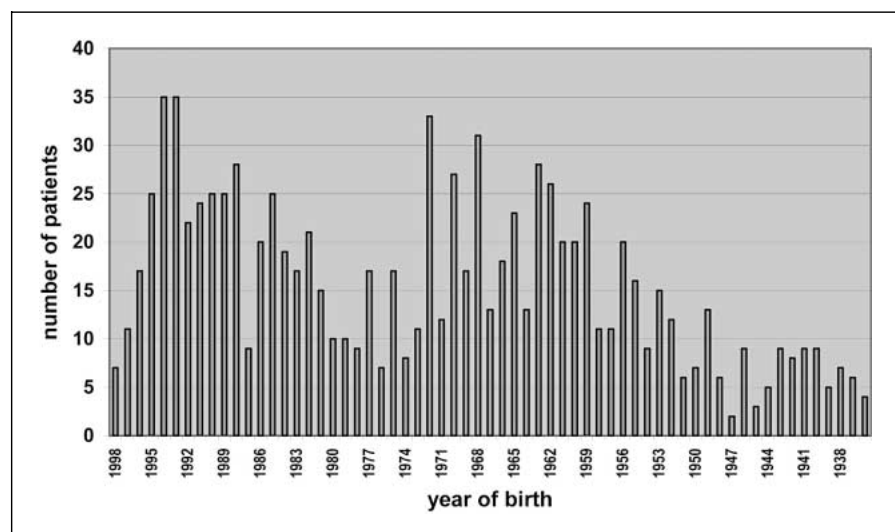


Fig. 1. Demographic spread of patients with allergic conditions. The bars show the absolute number of patients and their respective year of birth.

Table 1. Age distribution of the patients referred to the Allergy Clinic

Age group	Total	Peak year	Lowest year
0–9 years	231	1993 (15%)	1998 (3.3%)
10–19 years	189	1988 (14.8%)	1987 (4.8%)
20–29 years	151	1972 (21.8%)	1976 (4.6%)
30–39 years	208	1968 (14.9%)	1964/67 (6.3%)
40–49 years	143	1959 (16.8%)	1950 (4.9%)
50–59 years	73	1949 (17.8%)	1947 (2.7%)
60–63 years	22	1938 (31.8%)	1936 (18%)

The ages are grouped into decades. The birth years with the highest and lowest numbers of referred patients per decade are indicated. Dates of birth were not available in 7 of the 1,024 patients.

age being referred from elsewhere in the country. The population of Harare, the capital of Zimbabwe, has doubled in the past 25 years. The city's adult population in particular represents a cross-section of the inhabitants of this tropical country with their varying background exposure to infectious and parasitic organisms.

The costs of investigation and treatment were covered by medical insurance schemes in all but 2% of the patients. Medical insurance is optionally available to people in any formal employment regardless of socio-economic background, and therefore the patients were a fair representation of the background population. Considering that approximately 400,000 of the 1.4 million

inhabitants of Greater Harare have medical insurance cover, the 14,000 first-time secondary referrals would account for just under 3% of the people that could access specialist medical care in the private sector.

Atopic Conditions

In all age groups, the commonest presenting condition was allergic rhinitis (33%). The numbers of patients whose primary reasons for referral were other atopic conditions were similar, i.e. asthma in 10%, conjunctivitis in 9%, dermatitis in 10% and urticaria in 10%. Allergic pharyngitis was a rare and exclusively adult presentation. Although allergic rhinitis frequently co-existed with either or both asthma and conjunctivitis, allergen sensitisation was analysed separately.

Admittedly, the figures presented here are likely to underestimate the true extent of inhalant allergies in Zimbabwe. Firstly, the patients were secondary referrals with severe or intractable disease. Secondly, the patients were insured and could pay for private health care. Patients who were indigent or had milder diseases would not have been referred, and these are certainly in the majority. Thirdly, the management of patients with atopic diseases in Zimbabwe is not the preserve of clinical immunologists (allergologists), and therefore data from one allergy clinic may under-represent the disease spectrum. The age-defined specialties of paediatrics, internal medicine and organ-based specialists (otorhinolaryngologists, ophthalmologists and dermatologists) manage patients with asth-

Table 2. Table showing mean wheal diameters as indicators of the degree of sensitisation with a given allergen source

Allergen extract	Number	Mean mm	SD	Median mm	95% CI
House dust mite (<i>D. pteronyssinus</i>)	428 (53.3)	11.3	5.1	11	10.7–12
Bermuda grass	284 (35.3)	11.2	5.7	10	10.5–12
Maize pollen	233 (29)	9.2	4.1	9	5.7–9.8
Temperate grass mixture	206 (25.6)	8.6	3.9	8	8.9–9.1
Temperate tree mixture	138 (17.2)	7.7	6.1	6	6.7–8.7
Mould mixture	120 (14.9)	6.1	1.9	5	5.7–6.4
Feather mixture	127 (15.8)	5.8	1.6	5	5.5–6.1
Dog hair and dander	122 (15.2)	6.4	2.7	6	5.9–6.9
Cat hair and dander	145 (18.0)	6.9	3.5	6	6.3–7.4

Figures in parentheses represent percentages. Temperate grass mixture: Timothy, sweet vernal, redtop, Kentucky blue and Orchard pollen. Temperate tree mixture: maple (*Rulac negundo*), plane (*Platanus occidentalis*), false acacia (*Robinia*), horse chestnut and lime pollen. Mould mixture: *Alternaria*, *Aspergillus* and *Penicillium*. Feather mixture: goose, duck and chicken.

ma, rhino-sinusitis, conjunctivitis and dermatitis. Importantly, the latter specialties are abundantly represented and well established in Harare. The numbers are most likely skewed by referral patterns and should be regarded as representing the tip of a large iceberg.

Year of Birth, Annual Rainfall and Atopy

The numbers of patients referred for investigation of suspected allergy varied widely with the year of birth. Patients born in certain years (1938, 1949, 1959, 1968, 1972, 1988 and 1993) accounted for between 15 and 30% of all persons with allergy in a given 10-year age bracket, whereas patients born in other years (1936, 1947, 1950, 1964/67, 1976, 1987 and 1998) made up only 3–6% of referred patients. A review of meteorological data [9] shows that these years correspond with seasons with above- and below-average annual rainfall levels, respectively. The data suggest that there is a correlation between annual rainfall figures and allergen sensitisation. With abundant rainfall, plants, grasses and weeds, which are potential allergen sources, increase. The variation in the prevalence of inhalant allergies in Zimbabwe and perhaps Africa may thus be explained in part by different rainfall patterns and vegetation. A correlation between the numbers of patients with rhino-conjunctivitis and the annual rainfall pattern was observed.

Allergen Sources

Reactivity to aeroallergens in all the patients included in this paper was demonstrated by SPT. In addition, 70% of them were tested for total and antigen-specific IgE using the RAST technique. The results obtained using the two methods were concordant. Patients who were not reactive with SPT were unlikely to be reactive with RAST, although a number who were RAST negative were SPT positive. The SPT technique is cheaper and is a more specific indicator of sensitisation than the serological test. SPT or RAST reactivity to inhalant allergen sources was confirmed in 803 of the referred patients (76%). The remainder were not atopic or had other diagnoses.

The extent of sensitisation to the allergen extract was estimated by measuring wheal diameters, which reflect the type I (IgE-mediated) hypersensitivity response (table 2). All positive responses (wheal diameter greater than 3 mm and a saline control of 3 mm or less) were computed. The majority of those tested were sensitised to either house dust mites (*D. pteronyssinus* and *Dermatophagoides farinae*) (53%) or Bermuda grass (*C. dactylon*) (35%). Other common allergen sources were maize pollen (*Zea mays*), grass mixtures, tree mixtures, animal hair or dander, feathers and moulds, in that approximate order of frequency (table 3). When mono-sensitised, the patients were reactive to the house dust mites or Bermuda grass pollen, making these two the commonest single allergen sources. This predominance of reactivity to house dust mites and grass allergen extracts is in agreement with the findings of others in Europe [10, 11], North America [12]

Table 3. Allergen sources associated with the common presenting allergic conditions amongst atopic patients

	<i>D. pteronyssinus</i>	Bermuda	Maize	Grass	Tree	Dog	Cat	Moulds	Feathers
Allergic rhinitis (n = 341)	56.8	50.4	39	30.8	16.4	19.9	19.5	16.4	17.6
Conjunctivitis (n = 90)	58.3	50	30	35.4	12.5	16.7	20.8	13.5	13.5
Asthma (n = 104)	58.6	48.1	28.8	27.9	22.1	17.3	30.8	12.5	11.5
Dermatitis (n = 100)	64	30	16	26	12	14	12	14	10
Urticaria (n = 102)	35.3	25.5	11.8	13.7	24.5	13.7	13.7	14.7	21.6

The figures denote the percentages of patients sensitised to the listed allergen sources. In all cases, the house dust mites and Bermuda grass were the predominant allergen sources.

Table 4. Botanical grouping of the panel of grass pollen extracts used to determine sensitisation to inhalant allergen sources in Zimbabwe

Grass pollen allergen sources			
Bermuda grass (<i>Cynodon dactylon</i>)	<i>Chlorideae</i>		<i>Panicoideae</i>
Maize pollen (<i>Zea mays</i>)	<i>Maydeae</i>		<i>Panicoideae</i>
Five-grass pollen mixture			
Timothy (<i>Phleum pratense</i>)	<i>Festucae</i>	<i>Festuciformes</i>	<i>Pooideae</i>
Sweet vernal (<i>Anthoxanthum odoratum</i>)	<i>Festucae</i>	<i>Festuciformes</i>	<i>Pooideae</i>
Redtop (<i>Agrostis alba/stolonifera</i>)	<i>Festucae</i>	<i>Festuciformes</i>	<i>Pooideae</i>
Kentucky blue (<i>Poa pratensis/angustifolia</i>)	<i>Festucae</i>	<i>Festuciformes</i>	<i>Pooideae</i>
<i>Dactylis glomerata</i> (Scop)	<i>Festucae</i>	<i>Festuciformes</i>	<i>Pooideae</i>
Orchard (<i>Bromus glomerata</i>)	<i>Hordae</i>	<i>Festuciformes</i>	<i>Pooideae</i>

and the Southern African region [13]. Recently, Westritschnig et al. [14] demonstrated significant differences in the identity of disease-eliciting proteins in grass pollen and house dust mite extracts in sensitised patients.

House Dust Mites

The majority of the patients (53%) had SPT and RAST reactivity to house dust mites. An earlier sub-analysis had shown that all but 2% of the *D. farinae*-reactive patients were also reactive to *D. pteronyssinus*; therefore, in the interest of costs, only the *D. pteronyssinus* allergen extracts were used in the tests. *D. pteronyssinus*-sensitised patients were frequently co-sensitised with other allergen sources. The prevalence of house dust mite sensitisation reported in this selected population corroborates data from earlier Zimbabwean reports [7, 15] and is in agreement with reports from Ethiopia [16], Kenya [17], Tanzania [5], Nigeria [4, 18], Gabon [19], South Africa [20] and Ghana [21]. While rates of IgE antibody reactivity to the major Der p 2 allergen, for example, are similar in European and African sera, reactivity to Der p 5, Der p 7 and Der p 10 has been shown to be much higher in the

latter population [14]. The implications of these observations for disease pathogenesis or therapy remain to be elucidated.

Pollen Allergen Sources

Pollen allergens were the second most common source of sensitisation. Reactivity to Bermuda grass extracts was documented in 35.3% of the patients. In general, Bermuda grass-sensitive patients were also reactive to maize pollen and the 5-grass temperate grass mixture. The 5-grass pollen mixture was very infrequently the only or predominant allergen source; however, skin wheal diameters elicited with the extract were large (mean 8.6 mm compared to 11 mm with Bermuda grass, table 2). This suggests that although the grasses are exotic, allergenic proteins from these extracts contribute significantly to sensitisation.

Detailed SPT, serological and immunoblot analysis of serum reactivity to Timothy grass (*Phleum pratense*), which is part of the 5-grass extract reported by Westritschnig et al. [14], has shown that unlike in European patients, the Zimbabwean sera were reactive to Phl p 4 and Phl p 1,

but not to Phl p 2 and infrequently to Phl p 5, Phl p 6, Phl p 7 and Phl p 12, suggesting primary sensitisation to the *Chloridoideae* family of grasses (table 4), which does not contain group 2, 5 and 6 allergenic proteins [22]. When viewed together with lower-level reactivity to the 5-grass mixture, these data show differences in disease-eliciting allergens and suggest that while pollen extracts are good surrogate indicators of allergen sources, they still fall short of precisely defining the allergens. This has profound implications for diagnosis and, more importantly, the allergen-specific immunotherapy of grass-sensitised patients. Alternatively, the observed reactivity to temperate grasses may indicate cross-reactivity to homologues of antigens that are present, for example in *C. dactylon*, *Z. mays* or other, untested grasses from the same family.

Tree Pollen

Not a single patient was primarily sensitised to the tree pollen sources that were tested. This is hardly surprising since the tree pollen mixture used in SPT contained maple (*Rulac negundo*), horse chestnut, plane (*Planatus occidentalis*), false acacia (*Robinia*) and lime pollen, all of which are exotic and not represented in the local environment. Nevertheless, like the temperate grass mix, the degree of sensitisation, as measured by wheal diameters, was high. This reactivity is likely to be a result of cross-reactivity with other tree, grass or weed pollen allergens.

Animal Dander and Feathers

Patients who were reactive to cat hair or dander frequently had concomitant reactivity to dog hair. Patients who were sensitised to feathers were commonly reactive to egg or chicken allergens (data not shown). The pattern of reactivity to dog or cat hair and dander as well as to feathers has not been extensively reported in Africa. In a comparative study between Ghanaian and British cohorts, it was observed that sensitisation to domestic pet allergens was lower in African than British patients despite comparable numbers of pets kept in the two localities. Although the study populations cannot be compared, the Zimbabwean findings, like the reports from Nigeria [4] and Ghana [23], show that allergy to cat hair and dander is prevalent in Africa. The extent of sensitisation with these allergens will only be determined after systematic inquiry.

Mould Allergens

The allergen extract that was used to determine reactivity to moulds included *Alternaria*, *Aspergillus* and *Penicillium*. The identification of the specific reaction-elic-

iting mould was performed using the Allergopharma extracts. Sensitisation to the mould allergen extract was rare, but was usually high when present. Cross-sensitisation was infrequent, and sensitisation to *Alternaria* was the most frequent.

Wheal Diameter and Total IgE Levels

Mean wheal diameters of the patients with the most prevalent allergen sources (*D. pteronyssinus* and *C. dactylon*) were correlated with the total IgE levels. The mean or median wheal diameters for 192 patients with house dust mite sensitisation (mean = 11.34 mm) were found to correlate with total IgE levels (630.5, SD 757; median 297.5) as analysed using the Pearson linear correlation ($r = 0.3941$; 95% confidence intervals (CIs) 0.2674, 0.5074), linear regression ($r = 0.3941$) and Spearman rank correlation ($r = 0.3830$). A similar correlation was observed for the Bermuda grass-sensitised patients, whose mean wheal diameter (11.24 mm) correlated with total IgE levels (mean 706, SD 1,010; median 279.9; 95% CIs 494, 918) by the same statistical instruments. The linear regression coefficient was $R(r) = 0.3431$, the Pearson linear correlation coefficient was $r = 0.3431$ (95% CIs 0.1452, 0.5147) and the Spearman rank correlation was $r = 0.4607$ (95% CIs 0.2735, 0.6144). Although open to other interpretations, these observations suggest that, in the population studied, allergen-specific antibodies contribute significantly to the total IgE antibody levels.

Concluding Remarks

The sequence of events that leads to the development of allergic diseases in genetically predisposed (atopic) individuals includes (1) the exposure to environmental allergens, (2) the development of specific IgE antibodies to allergenic proteins, (3) the cross-linkage of allergens with mast cell-bound IgE antibodies, resulting in the release of mediators of inflammation and, finally, (4) the manifestation of disease [24]. The many variables that dictate whether or not an individual develops an IgE-mediated allergic disease include an atopic genetic background, long-term exposure to an allergen source and environmental factors. Inhalant allergies are by definition closely associated with the presence of respirable allergen sources and will almost inevitably occur if individuals satisfy the requirement of atopy, a genetically determined innate ability to mount IgE antibody-mediated responses

to otherwise innocuous and common, ubiquitous substances, such as pollen, dust or animal dander. The coming together of an allergen source and an atopic individual is a random event which is unlikely to respect geographical boundaries. It is therefore not surprising that investigations proved that allergic conditions are found to be as common in Zimbabwe as in most parts of the world, notwithstanding variable exposure to other environmental triggers.

There is worldwide variability in environmental allergies, but not in atopy and in the capacity to produce specific IgE antibodies. The expression of the atopic constitution has, for example, been found to be similar in Australian and Nigerian children although disease prevalence was different [4]. One of the factors that define differences is the flora and fauna of different continents and countries. While some allergen sources, such as *D. pteronyssinus* and *D. farinae*, animal dander, feathers and mould spores, are ubiquitous, others are environment specific (weeds, tree and grass pollen). The prevalence of symptoms of aeroallergen-mediated type 1 hypersensitivity diseases is therefore more likely to be determined by the distribution and types of environmental allergen sources, particularly pollen allergen sources. The major allergens of the ubiquitous allergen sources are known and the sensitisation of atopic individuals with these sources has been reported in many countries in the world. Recently, it has become apparent that antibody reactivity to house dust mite allergen extracts does not automatically translate to reactivity to the same allergenic proteins within that extract, thus emphasising the need for component-resolved, patient-tailored diagnostic approaches. Much less has been reported about grass and tree pollen allergen sources, which are unique to the continent.

Grass and Tree Pollen

The types of grasses and trees that are present in any one area vary with geographic and climatic conditions. Indeed, as reported here, the number of patients presented with atopic diseases varies with the rainfall intensity during their birth year, suggesting (1) that childhood exposure to aeroallergens influences the manifestation of atopic diseases and (2) that variations in the climate impact on atopic symptoms. The observed predominance of reactivity to natural extracts of *C. dactylon* and *Z. mays*, which belong to the *Panicoideae* family of grasses (table 4), is consistent with the predominance of these allergen sources in the local sub-tropical environment. The *Panicoideae* (*Cynodon* and *Z. mays*), in contrast to the *Festuciformes* grasses of the *Pooideae* families, are

appropriate and locally relevant allergen sources in this population. These two grasses are likely to be examples rather than representing a totality of a vast number of potential grass allergen sources which, in Southern Africa, are estimated at 947 indigenous and 115 naturalised grass species [25]. By the same token, it is probable that more grasses from this group may be responsible for allergen sensitisation, and until they are identified, data on the type and prevalence of type I diseases will remain underestimates and incomplete.

A large percentage of the patients were reactive to a mixture of natural grass extracts belonging to the *Festuciformes* family and to a variety of temperate tree extracts. This reactivity is incongruous with the local fauna and flora. These pollens are exotic in Zimbabwe. Although sensitisation to non-indigenous tree and grass extracts was relatively high, mono-sensitisation was infrequent and the respective wheal diameters were smaller and allergen-specific IgE antibody levels lower than those with Bermuda grass or maize. This could result from cross-reactivity between the exotic and indigenous pollens or cross-sensitisation with imported ornamental grasses/trees or non-species-specific antibody reactivity to common pollen allergens, which include two EF-hand allergens [26] and profilin [27]. The findings also emphasise that natural extracts are not the most appropriate sources of disease-associated pollen allergens. Timothy grass alone has at least 8 allergenic molecules [28], and it is conceivable that a similar order of magnitude can be found in other extracts leading to cross-reactivity. In the face of possible sensitisation with ubiquitous plant allergens, the identification of individual-specific allergy-eliciting molecules is virtually impossible if diagnosis is based on natural extracts. The identification of the primary sensitiser becomes difficult and the selection of appropriate preparations for allergen-specific immunotherapy challenging. The use of natural extracts in allergen-specific immunotherapy also carries the danger of iatrogenic sensitisation of patients with new components of the allergen extracts and increases the risk of life-threatening anaphylaxis. These concerns are likely to be addressed by the increased use of component-resolved allergens in both the diagnosis and treatment of pollen-sensitive patients [29]. These observations underscore the need to identify and characterise regionally relevant allergen sources. The recent study of grass pollen allergen sensitisation in Central Africa [14], which identifies significant differences in the types of allergens recognised by African sera, if pursued, should enable the identification of allergen-inducing proteins in the African context.

Comparisons with Other African Countries

Aeroallergen sensitisation patterns in atopic Zimbabwean children have previously been studied [15]. In that study, the presence of house dust mites and *C. dactylon* allergen-specific IgE antibodies in a third of patients with atopic dermatitis, a quarter with asthma or conjunctivitis and a sixth of those who presented with allergic rhinitis was demonstrated. The current cross-sectional analysis of patients of all ages, while confirming the earlier Zimbabwean observations, is not unique for this population. Relatively high rates of aeroallergen sensitisation have been

reported in South Africa [30], which has the largest diagnostic and clinical allergy referral service in sub-Saharan Africa, and in Morocco [31], which also has well-developed allergy services.

The experiences in this region and the limited published data should not lead to the conclusion that allergies are – for whatever reason – infrequent in Africa. It is more plausible that the apparent low prevalence of data on allergic diseases is a result of a lack of systematic investigation and the employment of inappropriate or locally inapplicable diagnostic reagents.

References

- 1 Sibanda EN: Research and clinical aspects of immunology in Africa. *Curr Opin Immunol* 2001;13:528–532.
- 2 Beasley R, Keil U, von Mutius E, Pearce N: Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis and atopic eczema: ISAAC. The International Study of Asthma and Allergies in Childhood (ISAAC) Steering Committee. *Lancet* 1998;351:1225–1232.
- 3 Yamanberhan H, Bekele Z, Venn A, Lewis S, Parry E, Britton J: Prevalence of wheeze and asthma and relation to atopy in urban and rural Ethiopia. *Lancet* 1997;350:85–90.
- 4 Farinan OA, Peat JK, Woolcock AJ: Prevalence of atopy, asthma symptoms and diagnosis, and the management of asthma: Comparison of an affluent and non-affluent country. *Thorax* 1999;54:606–610.
- 5 Sunyer J, Terregrosa J, Anto JM, Menendez C, Acosta C, Schellenberg D, Kahigwa E: The association between atopy and asthma in a semi-rural area of Tanzania (East Africa). *Allergy* 2000;55:762–766.
- 6 Van Nierkerk CH, Weinberg EG, Shore SC, Hesse HDV, Van Schalkwyk DJ: Prevalence of asthma: A comparative study of urban and rural Xhosa children. *Clin Allergy* 1979;9:319–324.
- 7 Keeley DJ, Neill P, Gallivan S: Comparison of the prevalence of reversible airways obstruction in rural and urban Zimbabwean children. *Thorax* 1991;46:549–553.
- 8 Strachan DP: Hay fever, hygiene and household size. *BMJ* 1989;299:1259–1260.
- 9 Department of Meteorological Services, Rainfall Unit, Ministry of Agriculture, Zimbabwe.
- 10 Brown CW, Hawkins L: Allergy prevalence and causal factors in the domestic environment: Results of a random population survey in the United Kingdom. *Ann Allergy Asthma Immunol* 1999;83:240–244.
- 11 Schutz-Kiss D, Popp W, Wagner C, Reiser K, Havelec L, Zwick K: Sensitisation to inhaled allergens in the Vienna population (in German). *Wien Klin Wochenschr* 1995;107:331–335.
- 12 Galant S, Berger W, Gillman S, Goldsobel A, Incaudo G, Kanter L, Machtinger S, McLean A, Prenner B, Sokol W, Spector S, Welch M, Ziering W: Prevalence of sensitization to aeroallergens in California patients with respiratory allergy. Allergy Skin Test Project Team. *Ann Allergy Asthma Immunol* 1998;82:203–210.
- 13 Morris A: Pattern of aeroallergen sensitisation amongst Cape Town adults with allergic rhinitis (abstract). *Curr Allergy Clin Immunol* 1998;11:18.
- 14 Westritschnig K, Sibanda E, Thomas W, Auer H, Aspöck H, Pittner G, Vrtala S, Spitzauer S, Kraft D, Valenta R: Analysis of the sensitisation profile towards allergens in Central Africa. *Clin Exp Allergy*, in press.
- 15 Kambarami RA, Marechera F, Sibanda EN, Chitiyo ME: Aero-allergen sensitisation patterns amongst atopic Zimbabwean children. *Cent Afr J Med* 1999;45:144–147.
- 16 Selassie FG, Stevens RH, Cullinan P, Pritchard D, Jones M, Harris J, Ayes JG, Newman Taylor AJ: Total and specific IgE (house dust mite and intestinal helminths) in asthmatics and controls from Gondar, Ethiopia. *Clin Exp Allergy* 2000;30:356–358.
- 17 Perzanowski MS, Ng'ang'a LW, Carter MC, Odhiambo J, Ngari P, Vaughan JW, Chapman MD, Kennedy MW, Platts-Mills TA: Atopy, asthma and antibodies to *Ascaris* among rural and urban children in Kenya. *J Pediatr* 2002;140:582–588.
- 18 Warrell DA, Fawcett IW, Harrison BDW, Agamah AJ, Ibu JO, Pope HM, Maberly DJ: Bronchial asthma in the Nigerian savanna region. A clinical and laboratory study of 106 patients with a review of the literature on asthma in the tropics. *Q J Med* 1975;174:325–347.
- 19 Van den Bigelaar AHJ, Lopuhua C, van Ree R, van der Zee S, Jans J, Hoek A, Migombet B, Borrmann S, Luckner D, Kremsner PG, Yazdankhsh M: The prevalence of parasite infestation and house dust mite sensitization in Gabonese schoolchildren. *Int Arch Allergy Immunol* 2001;126:231–238.
- 20 Prescott RA, Potter PC: Allergenicity and cross-reactivity of buffalo grass (*Stenotaphrum secundatum*). *S Afr Med J* 2001;91:237–243.
- 21 Addo-Yobo EO, Custovic A, Taggart SC, Craven M, Bonnie B, Woodcock A: Risk factors for asthma in urban Ghana. *J Allergy Clin Immunol* 2001;108:363–368.
- 22 Niederberger V, Laffer S, Froschl R, Kraft D, Rumpold H, Kapiotis S, Valenta R, Spitzauer S: IgE antibodies to recombinant pollen allergens (Phl p1, Phl p2, Phl p5 and Bet v 2) account for a high percentage of grass pollen specific IgE. *J Allergy Clin Immunol* 1998;101:258–264.
- 23 Woodcock A, Addo-Yobo EO, Taggart SC, Craven M, Custovic A: Pet allergens in homes in Ghana and the United Kingdom. *J Allergy Clin Immunol* 2001;108:463–465.
- 24 Kay AB: Allergy and Allergic Disease. Oxford, Blackwell Science, 1997.
- 25 Health on the Net: Allergy Glossary (www.hon.ch/Library/Theme/Allergy/Glossary/allergy.html).
- 26 Valenta R, Hayek B, Seiberler S, Bugajska-Schetter A, Niederberger V, Twardosz A, Natter S, Vangelista L, Pastore A, Spitzauer S, Kraft D: Calcium binding allergens: From plants to man. *Int Arch Allergy Immunol* 1998;117:160–166.
- 27 Kazemi-Shirazi L, Niederberger V, Linhart B, Lidholm J, Kraft D, Valenta R: Recombinant marker allergens: Diagnostic gatekeepers for the treatment of allergy. *Int Arch Allergy Immunol* 2002;127:259–268.
- 28 DeWitt AM, Niederberger V, Lehtonen P, Spitzauer S, Sperr WR, Valent P, Valenta R, Lidholm J: Molecular and immunological characterization of a novel timothy grass (*Phleum pratense*) pollen allergen, Phl p 11. *Clin Exp Allergy* 2002;32:1329–1340.
- 29 Valenta R: Recombinant allergen-based concepts for diagnosis and therapy of type I allergy. *Allergy* 2002;57(suppl 71):66–67.
- 30 Potter PC, Berman D, Toerien A, Mahlerbe D, Weinberg EG: Clinical significance of aeroallergen identification in the Western Cape. *S Afr Med J* 1991;19:80–84.
- 31 Yazidi AA, Nejari C, Bartal M: Skin sensitization to pollens in Morocco. Multicenter study (in French). *Rev Mal Respir* 2001;18:465–467.