

Prevalence and associated factors of atopic dermatitis symptoms in rural and urban Ethiopia

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Summary

Background Allergic diseases, including atopic dermatitis (AD), are increasingly becoming a clinical problem in developing countries.

Objective We investigated the prevalence of AD symptoms and the effects of potential environmental aetiologies in rural and urban areas of Jimma in southwestern Ethiopia.

Methods Information on allergic disease symptoms and lifestyle factors was gathered in an interviewer-led cross-sectional questionnaire-based population survey of 9844 urban and 3032 rural participants of all ages. A one-in-four subsample underwent skin prick testing for hypersensitivity to *Dermatophagoides pteronyssinus*, mixed threshings, and aspergillus.

Results Around 95% of those eligible took part in the survey. Lifetime cumulative prevalence of AD symptoms was generally low with an overall prevalence of 1.2%, but was higher in the urban (1.5%) than in the rural area (0.3%; odds ratio (OR) = 4.45 [95% CI 2.34–8.47]). AD symptoms were strongly associated with wheeze (adjusted OR = 22.03 [15.45–31.42]) and rhinitis symptoms (61.94 [42.66–89.95]). Of several environmental exposures assessed, residence in a house made of brick (rather than mud) walls with wooden (rather than clay) floor, exposure to cigarette smoke as a child, having lived outside of Jimma in the past, and being of the Tigrean ethnic group were associated with an increased risk of AD symptoms.

Conclusion Although the overall prevalence of AD symptoms was low in this Ethiopian population, a marked urban–rural gradient was evident. Lifestyle factors linked to urbanization were associated with an increased risk of AD symptoms.

Keywords allergic diseases, allergy, atopic dermatitis, atopy, hygiene hypothesis

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Introduction

Allergic diseases, including atopic dermatitis (AD), are becoming increasingly common worldwide [1]. What was initially thought to be an epidemic confined to western industrialized countries now appears to be extending to urban centres of less developed nations [2, 3]. Although a substantial body of research now exists on asthma symptoms in developing countries, the prevalence and determinants of AD symptom prevalence in developing countries has largely been ignored [4, 5].

Jimma is a town of around 80 000 inhabitants in southwestern Ethiopia. It has no major industry, people live in low-cost housing, eat predominantly local produce and often keep animals indoors. In the rural districts of Jimma, traditional subsistence farming prevails. We have previously reported

that although Jimma is at a relatively early stage of industrial and economic development, asthma has become a recognized clinical problem over the past two decades [6]. In the process of that survey, we also collected data on self-reported prevalence of allergic disease symptoms (including AD) and domestic kerosene use [7]. The present study documents the prevalence of AD symptoms in rural and urban settings in Ethiopia and their relationship to atopy and other risk factors in this population.

Methods

In 1996, we carried out a cross-sectional household survey in rural and urban Jimma in southwestern Ethiopia [6]. Details of this study have been published previously. In summary, participants were drawn in a one-in-eight systematic sample of households in Jimma town (urban sample) and in three rural communities more than 50 km by road from Jimma town and at least 15 km apart from each other (rural sample). Trained fieldworkers carried out the survey with assistance

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from local community leaders. An interviewer-administered questionnaire gathered data on household demography, housing and other lifestyle variables as well as symptoms of allergic disease. Parents responded on behalf of children under the age of 7. The questionnaire contained two questions to elicit AD symptoms: 'Have you ever had an itchy skin rash affecting the skin creases (of arms and legs)?', and if the answer was 'yes' to the first question, participants were asked 'Have you had this rash over the past 12 months?' In addition to the interviewer-administered questionnaire, all family members in every fourth household in the survey were skin tested for *Dermatophagoides pteronyssinus*, mixed threshings, and aspergillus allergen. Skin tests were defined as positive if the weal diameter, after subtraction of the saline-control response, was 3 mm or more. Ethics approval was gained from local ethics committees in Jimma and Nottingham.

The analysis of both questionnaire and skin test data was performed with SPSS-PC version 11.0 (LEAD Technologies Inc., Chicago, IL, USA). The two questions on AD symptoms were analysed with regard to known risk factors for AD, such as urban/rural prevalence differences, wheeze, rhinitis, atopy (skin test positivity to house dust mite (HDM), mixed threshings, or aspergillus), and demographic and lifestyle factors, such as socio-economic status (based on ownership of goods), number of older siblings, crowding, smoking, mattress and pillow material, type of bed, housing construction style (type of roof, walls and floor), animals kept in the house, insecticide use in the house, childhood vaccinations, ethnic group, and whether participants had lived somewhere else at some point in their life. Odds ratios (ORs) and corresponding 95% confidence intervals were

calculated for each potential risk factors with adjustment for age and sex, socio-economic status, place of residence, and domestic use of kerosene, a previously identified risk factor [7]. Factors identified as statistically significant during univariate analysis were then entered into a stepwise logistic regression model, in which age, sex, socio-economic status, urban residence and domestic kerosene use were controlled for.

Results

Questionnaire information was obtained from 12 876 (47.6% male) participants, of whom 9844 lived in urban and 3032 in rural areas. The mean participant age was 23.3 (SD 17.2) years (range 0–120 years) in the urban and 22.2 (SD 17.9) years (range 0–90) in the rural population, comprising around 95% of those eligible to take part [6]. A total of 153 (1.2%) participants reported they had AD symptoms at some point in their life. 87 (0.7%) participants had AD symptoms 'over the past year', and 143 out of 153 participants with AD symptoms lived in urban Jimma.

The following analysis only presents associations between risk factors and AD symptoms 'ever', since repeating the analysis for AD symptoms 'over the past 12 months' did not alter results significantly. The latter are, however, shown in all tables.

The relative odds of AD symptoms in relation to urban dwelling was 4.45 [95% CI 2.34–8.47]. The prevalence of AD symptoms increased with age and was higher in males than females (OR = 1.50 [1.08–2.06]). AD symptoms were also

Table 1. Atopic dermatitis (AD) symptom prevalences and their relation to demographic factors

	Complete sample population <i>N</i> (%)	AD symptoms ever			AD symptoms past year		
		<i>N</i> (%)	Unadjusted OR (95% CI)	<i>P</i>	<i>N</i> (%)	Unadjusted OR (95% CI)	<i>P</i>
Complete sample	12 876	153 (1.2)			87 (0.7)		
<i>Residence</i>							
Urban	9844 (76.5)	143 (1.5)	4.45 (2.34–8.47)	<0.001	81 (0.8)	4.18 (1.82–9.52)	<0.001
Rural	3032 (23.5)	10 (0.3)	1		6 (0.2)	1	
<i>Sex</i>							
Male	6134 (47.6)	88 (1.4)	1.50 (1.08–2.06)	0.02	49 (0.8)	1.42 (0.93–2.17)	0.11
Female	6742 (52.4)	65 (1.0)	1		38 (0.6)	1	
<i>Age</i>							
0–19	6886 (53.5)	49 (0.7)	1	<0.001	21 (0.3)	1	<0.001
20–39	3621 (28.1)	49 (1.4)	1.91 (1.29–2.85)	$P_{\text{trend}} < 0.001$	34 (0.9)	3.10 (1.80–5.34)	$P_{\text{trend}} < 0.001$
40–59	1702 (13.2)	33 (1.9)	2.76 (1.77–4.30)		20 (1.2)	3.88 (2.10–7.18)	
60+	667 (5.2)	22 (3.3)	4.76 (2.86–7.92)		12 (1.8)	5.98 (2.93–12.21)	
<i>Socio-economic status by ownership of goods</i>							
None of the below	5790 (45.0)	47 (0.8)	1	<0.001	30 (0.5)	1	0.22
Radio (but none of below)	4975 (38.6)	62 (1.2)	1.54 (1.05–2.26)	$P_{\text{trend}} < 0.001$	37 (0.7)	1.44 (0.89–2.33)	$P_{\text{trend}} < 0.02$
Telephone (but none of below)	947 (7.4)	21 (2.2)	2.77 (1.65–4.65)		8 (0.8)	1.64 (0.75–3.58)	
TV (but not car)	795 (6.2)	15 (1.9)	2.35 (1.31–4.22)		7 (0.9)	1.71 (0.75–3.90)	
Car	368 (2.9)	8 (2.2)	2.71 (1.27–5.79)		5 (1.4)	2.65 (1.02–6.86)	
<i>Kerosene use (for cooking/heating)</i>							
No	12 337 (95.8)	131 (1.1)	1	<0.001	73 (0.6)	1	<0.001
Yes	539 (4.2)	22 (4.1)	3.96 (2.50–6.28)		14 (2.6)	4.48 (2.51–7.99)	

OR, odds ratio; CI, confidence interval.

Table 2. Association between atopic dermatitis (AD) symptoms and wheeze, rhinitis, and skin hypersensitivity

Risk factor	Complete sample population	AD symptoms ever				AD symptoms past year				
		N (%)	Unadjusted OR (95% CI)	P-value	Adjusted* OR (95% CI)	P-value	N (%)	Unadjusted OR (95% CI)	Adjusted OR* (95% CI)	P-value
Wheeze										
No	12470 (96.8)	85 (0.7)	1		1		43 (0.3)	1	1	
Yes	406 (3.2)	68 (16.7)	29.31 (20.93–41.05)	<0.001	22.03 (15.45–31.42)	<0.001	44 (10.8)	35.11 (22.77–54.14)	25.59 (16.20–40.41)	<0.001
Rhinitis										
No	12548 (97.5)	62 (0.5)	1		1		32 (0.3)	1	1	
Yes	328 (2.5)	91 (27.7)	77.32 (54.63–109.44)	<0.001	61.94 (42.66–89.95)	<0.001	55 (16.8)	79.09 (50.33–124.29)	58.96 (36.23–95.94)	<0.001
Atopy†										
No	3089 (91.9)	46 (1.5)	1		1		28 (0.9)	1	1	
Yes	274 (8.1)	8 (2.9)	1.99 (0.93–4.26)	0.08	2.13 (0.96–4.73)	0.06	4 (1.5)	1.62 (0.56–4.65)	1.46 (0.48–4.42)	0.5
HDM‡										
No	3163 (94.1)	48 (1.5)	1		1		28 (0.9)	1	1	
Yes	199 (5.9)	6 (3.0)	2.02 (0.85–4.77)	0.11	2.27 (0.91–5.64)	0.078	4 (2.0)	2.30 (0.80–6.61)	2.07 (0.67–6.40)	0.27

*Odds ratios (ORs) adjusted for age, sex, socio-economic status (by ownership of goods), residence (urban/rural), and kerosene use. †Atopy was defined as at least one positive skin prick test to either *Dermatophagoides pteronyssinus*, mixed threshings, or aspergillus allergen. ‡HDM, house dust mite (*D. pteronyssinus*) skin hypersensitivity. CI, confidence interval.

generally associated with higher socio-economic status and kerosene use for cooking and heating (Table 1).

There was a strong association between AD symptoms and other symptoms of allergic disease (Table 2). The OR for AD symptoms in relation to wheeze in the past year after adjustment for age, sex, socio-economic status, urban residence, and domestic kerosene use was 22.03 [15.45–31.42] and in relation to rhinitis symptoms 61.94 [42.66–89.95]. AD symptoms were also more common in individuals sensitized to environmental allergens, including *D. pteronyssinus*, approaching conventional statistical significance (adjusted OR = 2.13 [0.96–4.73], $P = 0.06$).

Univariate analysis of environmental risk factors identified a significant association between AD symptoms and type of mattress, with the greatest risk in those sleeping on synthetic foam. Dwelling in houses with brick (rather than mud) walls, type of floor and having a roof made of corrugated iron all increased the risk of AD symptoms (Table 3). The effects of roof and mattress material were, however, reduced and no longer significant after controlling for age, sex, residence, socio-economic status, and domestic kerosene use. Parental smoking during childhood was significantly associated with AD symptoms after adjustment (OR = 2.13 [1.31–3.46]), and a personal history of smoking for at least one year was of borderline significance (adjusted OR = 1.70 [0.97–2.97], $P = 0.06$). Furthermore, those who had lived outside of Jimma town at some point in their life were at a significantly increased risk of developing AD symptoms (adjusted OR = 1.92 [1.29–2.84]), as did those belonging to the Tigre ethnic group (Table 4). In addition, living with animals increased the risk of AD symptoms with borderline statistical significance (OR = 1.35 [0.97–1.88], $P = 0.07$).

There was no significant association between AD symptoms and the number of older siblings (Table 3). Most vaccinations appeared to exercise a protective effect in univariate analysis (Table 4), but, after adjustment for confounders, only whooping cough vaccination remained statistically significant (adjusted OR = 0.60 [0.37–0.99]).

A reduction in AD symptom prevalence was observed in people using insecticides in their homes for cleaning and disinfecting purposes. This effect was particularly strong for DDT (adjusted OR = 0.33 [0.18–0.60], Table 4). Other potential risk factors explored included crowding (family size/number of rooms), pillow material and bed type, but no significant associations were identified. In the final logistic regression model the independent determinants of AD symptoms were age, sex, urban residence, domestic kerosene use, indoor DDT use, previous residence outside of Jimma, ethnic origin, floor material, and parental smoking during childhood (Table 5).

Discussion

Main findings

This cross-sectional population-based survey provides data on the prevalence and risk factors for AD symptoms in rural and urban areas of Jimma in southwestern Ethiopia. Overall, AD symptom lifetime prevalence was only 1.2% and, therefore, much lower than in most industrialized countries

Table 3. Associations between atopic dermatitis (AD) symptoms ever and lifestyle factors

Risk factor	Complete sample population <i>N</i> (%)	AD symptoms ever <i>N</i> (%)	Unadjusted OR (95% CI)	<i>P</i> -value	Adjusted OR* (95% CI)	<i>P</i> -value
<i>Material of mattress</i>						
Cotton	6045 (47.0)	80 (1.3)	1	<0.001	1	0.53
Synthetic foam	1553 (12.1)	35 (2.3)	1.72 (1.15–2.57)		1.26 (0.82–1.94)	
Grass	1937 (15.0)	22 (1.1)	0.86 (0.53–1.38)		1.25 (0.75–2.08)	
Kapoake	1342 (10.4)	3 (0.2)	0.17 (0.05–0.53)		0.49 (0.14–1.75)	
Mat	1872 (14.5)	13 (0.7)	0.52 (0.29–0.94)		1.28 (0.62–2.63)	
Other	125 (1.0)	0	–			
<i>Material of walls</i>						
Bricks	930 (7.2)	27 (2.9)	1	<0.001	1	0.02
Mud	11 675 (90.7)	121 (1.0)	0.35 (0.23–0.53)		0.52 (0.33–0.82)	
Other	270 (2.1)	5 (1.9)	0.63 (0.24–1.66)		0.77 (0.29–2.07)	
<i>Material of floor</i>						
Cement	4037 (31.4)	63 (1.6)	1	<0.001	1	0.003
Marble	705 (5.5)	15 (2.1)	1.37 (0.78–2.42)		1.53 (0.85–2.77)	
Wood	50 (0.4)	5 (10.0)	7.01 (2.69–18.25)		6.47 (2.41–17.37)	
Clay	8078 (62.7)	70 (0.9)	0.55 (0.39–0.78)		0.99 (0.64–1.54)	
Other	6 (0.0005)	0	–		–	
<i>Material of roof</i>						
Corrugated iron	10 365 (80.5)	140 (1.4)	1	0.004	1	0.95
Thatched	2428 (18.9)	12 (0.5)	0.36 (0.20–0.66)		1.12 (0.53–2.39)	
Other	83 (0.6)	1 (1.2)	0.89 (0.12–6.44)		1.16 (0.16–8.50)	
<i>Smoking</i>						
Parents smoked as child						
No	11 395 (88.5)	132 (1.2)	1	0.39	1	0.002
Yes	1481 (11.5)	21 (1.4)	1.23 (0.77–1.95)		2.13 (1.31–3.46)	
For at least 1 year						
No	12 258 (95.2)	136 (1.1)	1	<0.001	1	0.06
Yes	618 (4.8)	17 (2.8)	2.52 (1.51–4.20)		1.70 (0.97–2.97)	
<i>Number of older siblings</i>						
None	4256 (33.1)	62 (1.5)	1	0.12	1	0.29
One	2909 (22.5)	27 (0.9)	0.63 (0.40–1.00)		0.69 (0.44–1.10)	
Two	2093 (16.3)	28 (1.3)	0.92 (0.59–1.44)		1.11 (0.70–1.76)	
Three or more	3617 (28.1)	36 (1.0)	0.68 (0.45–1.03)		0.84 (0.54–1.29)	

*Odds ratios (ORs) adjusted for age, sex, socio-economic status (by ownership of goods), residence (urban/rural), and kerosene use. CI, confidence interval.

[3]. At the same time, AD symptoms were around four times more common in urban areas than in the countryside, suggesting that even a minor degree of urbanization and affluence, as seen in Jimma town, can have a significant impact on AD symptom prevalence.

Risk factor analysis

Housing We identified several risk factors that may explain part of this urban–rural gradient. One important aetiological factor could be an increase in HDM exposure secondary to the use of modern housing construction methods. For example, floor material was a significant risk factor. In comparison to people living in houses with traditional clay floor, wooden floor increased the risk of eczema symptoms more than sixfold. On the other hand, the increased risk seen with sleeping on synthetic foam mattresses, brick (rather than mud) walls and corrugated iron (rather than thatched) roofs in univariate analysis weakened and lost statistical significance

after adjustment. Additional evidence for the role of HDMs in AD symptom aetiology might be the reduced risk of developing AD symptoms associated with indoor DDT use for cleaning purposes, potentially secondary to insecticide-induced HDM eradication. However, as previously reported, DDT use had no direct effect on skin hypersensitivity to HDM allergen in our study population [6]. Another explanation would be a DDT-induced eradication of cockroaches, especially since we found cockroach allergen to be associated with allergic wheeze in a follow-on study in Jimma [8]. Cockroach skin hypersensitivity has also been implicated as an aetiological factor in AD in other geographical settings [9, 10]. In future studies, it would therefore be helpful to perform skin prick tests to a broader spectrum of environmental allergens.

Fewer infections? In industrialized countries, AD tends to be less common among children growing up with older siblings [11]. Based on this observation, it has been speculated that exposure to certain pathogens might protect against

Table 4. Exposure rates and odds ratios (ORs) for atopic dermatitis (AD) symptoms ever and risk factors

Risk factor	Complete sample population <i>N</i> (%)	AD symptoms ever <i>N</i> (%)	Unadjusted OR (95% CI)	<i>P</i> -value	Adjusted OR* (95% CI)	<i>P</i> -value
<i>Animals kept indoors</i>						
No	6953 (54.0)	83 (1.2)	1		1	
Yes	5923 (46.0)	70 (1.2)	0.99 (0.72–1.36)	0.95	1.35 (0.97–1.88)	0.07
<i>Lived elsewhere</i>						
No	8477 (65.8)	55 (0.6)	1		1	
Yes	4399 (34.2)	98 (2.2)	3.49 (2.50–4.86)	<0.001	1.92 (1.29–2.84)	0.001
<i>Ethnic group</i>						
Other	10 491 (81.5)	98 (0.9)	1	<0.001	1	<0.001
Amhara	2085 (16.2)	42 (2.0)	2.18 (1.51–3.14)		1.44 (0.98–2.12)	
Tigre	297 (2.3)	13 (4.4)	4.85 (2.69–8.76)		3.57 (1.95–6.53)	
<i>Vaccinations</i>						
None	4139 (32.1)	36 (0.9)				
Measles	4733 (37.5)	38 (0.8)	0.58 (0.40–0.85)	0.005	0.79 (0.49–1.28)	0.34
Whooping cough	4701 (37.2)	33 (0.7)	0.49 (0.33–0.72)	<0.001	0.60 (0.37–0.99)	0.045
Diphtheria	4711 (37.3)	36 (0.8)	0.55 (0.37–0.79)	0.002	0.72 (0.44–1.18)	0.19
BCG	4915 (38.9)	42 (0.9)	0.63 (0.44–0.90)	0.01	0.81 (0.51–1.28)	0.36
Small pox	1738 (13.8)	44 (2.5)	2.75 (1.92–3.92)	<0.001	1.36 (0.91–2.05)	0.14
Mumps	819 (6.5)	6 (0.7)	0.62 (0.27–1.40)	0.25	0.66 (0.29–1.53)	0.33
Tetanus	6682 (52.9)	66 (1.0)	0.73 (0.53–1.02)	0.06	0.98 (0.65–1.47)	0.92
Polio	4714 (37.3)	37 (0.8)	0.57 (0.39–0.83)	0.003	0.76 (0.47–1.25)	0.29
<i>Insecticide home use</i>						
None	7052 (54.8)	76 (0.6)	1		1	
Any insecticide	5824 (45.2)	77 (1.3)	1.23 (0.89–1.69)	0.20	0.70 (0.49–0.99)	0.04
DDT	2199 (17.1)	12 (0.5)	0.41 (0.23–0.74)	0.003	0.33 (0.18–0.60)	<0.001
Malathion	2186 (17.0)	26 (1.2)	1.00 (0.66–1.53)	0.996	0.76 (0.49–1.17)	0.21

*ORs adjusted for age, sex, socio-economic status (by ownership of goods), residence (urban/rural), and kerosene use.
CI, confidence interval.

Table 5. Final logistic regression model

Risk factor	Odds ratio (95 % CI)	<i>P</i> -value	Effect on atopic dermatitis risk
<i>Age</i>			
0–19	1	<0.001	↑
20–39	1.49 (0.97–2.30)		
40–59	2.03 (1.24–3.35)		
60+	3.69 (2.10–6.49)		
Male sex	1.56 (1.12–2.16)	0.008	↑
Urban residence	3.74 (1.82–7.75)	<0.001	↑
Domestic kerosene use	2.32 (1.40–3.83)	0.001	↑
Indoor DDT use	0.35 (0.19–0.64)	0.001	↓
Lived elsewhere	2.08 (1.39–3.12)	<0.001	↑
Tigrean ethnic group	2.77 (1.46–5.25)	0.001	↑
<i>Socio-economic status</i>			
None of the below	1	0.46	
Radio (but none of below)	1.84 (0.79–4.33)		
Telephone (but none of below)	1.46 (0.73–2.94)		
TV (but no car)	1.67 (0.88–3.16)		
Car	1.13 (0.74–1.73)		
Wooden floor	6.12 (2.19–17.13)	0.008	↑
Parental smoking as a child	2.22 (1.35–3.63)	0.002	↑

CI, confidence interval.

clinical expression of allergic disease [12]. This 'hygiene hypothesis' has also been extended to vaccinations and contact with farm animals. We found a weak reduction in

AD symptom prevalence with most childhood vaccines, but this association disappeared after adjustment for all types of vaccinations except whooping cough. We also found no evidence of an inverse relationship between number of older siblings, indoor keeping of animals and AD symptoms. The latter concords with findings from studies conducted among farm children in Europe [13, 14], where a protective effect was seen for allergen skin hypersensitivity, asthma, and allergic rhinitis but not for AD [15].

Pollution Outdoor pollution might be partially responsible for symptoms of allergic disease in industrialized countries. However, in view of the low levels of motorized transport and the absence of any major industry in Jimma town, this can be discounted as a likely explanation for the emergence of asthma and AD symptoms in Jimma. A stronger case can be made for indoor pollution, certainly in Jimma town, where we previously found a significant association between kerosene fuel use for heating and cooking, and AD symptoms [7]. A further source of indoor pollution is tobacco smoke. In Jimma, exposure to cigarette smoke as a child was a strong risk factor for AD symptoms. There is also some evidence from Germany that maternal smoking during pregnancy and lactation can increase the risk of developing AD [16]. However, a recent cross-sectional survey from Sweden has suggested an inverse relationship between exposure to tobacco smoke and AD [17].

Study limitations

A potential limitation of this questionnaire-based study was the use of only two questions relating to AD symptoms with an emphasis on flexural involvement, the body site typically affected in Caucasian patients. It is possible that the ratio of flexural to non-flexural AD is altered by geographical factors, such as exposure to sun and humidity. If this was the case, we would have underestimated the prevalence of AD symptoms in our study population.

Given that our survey was purely questionnaire based, some of the cases identified may have had a different skin condition. The two most common pruritic skin diseases found in rural southwestern Ethiopia are onchocerciasis and scabies [18]. However, had the itchy skin condition we detected in our study population been onchocerciasis, we would have expected to find a disease prevalence much higher than 1.2%. For example, Mengistu et al. [19] found prevalence rates of 23% for onchocerciasis among farmers in a cross-sectional study in rural southwestern Ethiopia. Rather than being common in the countryside and rare in urban areas, as is generally the case in onchocerciasis, AD symptoms showed the opposite prevalence distribution pattern in our study. Some participants with AD symptoms might possibly have had scabies rather than AD, but this would not account for the association with wheeze and rhinitis (adjusted ORs = 22.03 and 61.94, respectively, Table 2).

In European countries, both scabies and AD are more commonly seen in children than adults, and the increase in AD symptom prevalence with age in our study appears unusual. However, this is in keeping with our findings for 'wheeze in the past 12 months' in the same study population. Allergic wheeze seems to have emerged as a recognized clinical problem in Jimma town around 10 years prior to our study, suggesting new environmental exposures that mainly affect adults rather than children, at least initially [6] in this population.

Finally, recall bias is a concern in any epidemiological study that uses questionnaires to measure risk factors, such as parental smoking and childhood vaccinations. However, Jimma society has so far had very little contact with western medical concepts and pre-formed ideas about risk factors for allergic disease are therefore probably rare. Any error in the reporting of these exposures is likely to be similar for those with and without AD, resulting in a dilution of any true effect.

Much remains to be explained

The risk factors identified in this study can only explain some of the urban–rural gradient in AD prevalence in Jimma. Even in the final logistic regression model, the risk of AD symptoms in urban areas remained more than three times higher than in the countryside. The ethnic associations we found for AD symptom prevalence, especially in members of the Tigrean ethnic group, were independent of either migration into Jimma town or socio-economic status (measured by ownership of goods, education or profession) and could potentially be genetic in origin.

It is possible that another important factor in AD symptom aetiology in Jimma town is reduced exposure to endoparasites. As we have previously been able to demonstrate an inverse relationship between hookworm infection and allergic wheeze in the same population, part of the urban–rural gradient seen

in AD symptoms may be explained by reduced endoparasite exposure in Jimma town in comparison to rural areas [8]. This would also be a plausible explanation why migration into Jimma town was generally associated with an increased risk of AD symptoms. Further research into the relationship between endoparasites and AD is currently underway.

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Conflict of interest

None.

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