Original articles

Prevalence of exercise induced bronchospasm in Kenyan school children: an urban-rural comparison

L W Ng'ang'a, J A Odhiambo, M W Mungai, C M Gicheha, P Nderitu, B Maingi, P T Macklem, M R Becklake

Abstract

***Background*—Higher rates of exercise induced bronchospasm (EIB) have been reported for urban than for rural African schoolchildren. The change from a traditional to a westernised lifestyle has been implicated. This study was undertaken to examine the impact of various features of urban living on the prevalence of EIB in Kenyan school children.**

***Methods*—A total of 1226 children aged 8–17 years attending grade 4 at five randomly selected schools in Nairobi (urban) and five in Muranga district (rural) underwent an exercise challenge test. A respiratory health and home environment questionnaire was also administered to parents/guardians. This report is limited to 1071 children aged ≤12 years. Prevalence rates of EIB for the two areas were compared and the differences analysed to model the respective contributions of personal characteristics, host and environmental factors implicated in childhood asthma.**

***Results*—A fall in forced expiratory volume in one second (FEV_1) after exercise of ≥10% occurred in 22.9% of urban children and 13.2% of rural children (OR 1.96, 95% CI 1.41 to 2.71). The OR decreased to 1.65 (95% CI 1.10 to 2.47) after accounting for age, sex, and host factors (a family history of asthma and breast feeding for less than six months), and to 1.21 (95% CI 0.69 to 2.11) after further adjustment for environmental factors (parental education, use of biomass fuel and kerosene for cooking, and exposure to motor vehicle fumes).**

***Conclusions*—The EIB rates in this study are higher than any other reported for African children, even using more rigorous criteria for EIB. The study findings support a view which is gaining increasing credence that the increase in prevalence of childhood asthma associated with urbanisation is the consequence of various harmful environmental exposures acting on increasingly susceptible populations.**

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Respiratory Disease Research Unit, Kenya Medical Research Institute, P.O. Box 47855, Nairobi, Kenya
L W Ng'ang'a
J A Odhiambo
M W Mungai
C M Gicheha
P Nderitu
B Maingi

International Respiratory Disease Research Unit, International Union Against Tuberculosis and Lung Disease (IUATLD), Montréal, Québec, Canada
P T Macklem
M R Becklake

Respiratory Epidemiology Unit, Joint Departments of Epidemiology and Biostatistics and of Occupational Health, McGill University, Montréal, Québec, Canada
M R Becklake

Correspondence to: Dr M R Becklake, Respiratory Epidemiology Unit, Joint Departments of Epidemiology and Biostatistics, and of Occupational Health, McGill University, 1110 Pine Ave West, Montréal, Québec, Canada H3A 1A3.

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Airway hyperresponsiveness has been included as part of the working definition of asthma in epidemiological studies of childhood asthma,[1] although symptoms remain the cornerstone of most such surveys.[2] Exercise induced bronchospasm (EIB) is useful in this context because it is a common feature of asthma[3–5] and the exercise challenge test provides a non-invasive tool for determining airway hyperresponsiveness in epidemiological studies of both children and young people.[5] Another advantage is that it measures differences in response at a given "dose" (near maximal exercise) compared with the provocative dose (or concentration) of an agonist required to elicit a given response, and may therefore be more useful in detecting excessive airway narrowing. The exercise challenge test also circumvents difficulties arising from the use of questionnaires in communities with different language and cultural orientations.[6]

In Africa,[6] as elsewhere,[7] the prevalence of asthma has been shown to vary between countries, between regions, and between different geographical areas within a country. Studies in South Africa,[8] Zimbabwe,[9] and Ghana[10] have also shown the prevalence of EIB to be higher among children living in urban areas than in rural areas. The adoption of a westernised lifestyle concomitant with increasing urbanisation has been implicated. In addition, there is evidence to suggest that the prevalence of asthma, particularly childhood asthma in Africa, is increasing[6] in much the same way as has occurred in many industrially developed countries.[7] However, doubt has been expressed that such secular trends in asthma can be attributed entirely to the environmental changes imposed by urban living and/or westernised lifestyles, and the question has been raised as to whether urban populations are also becoming more susceptible to developing asthma.[11] In Kenya the only community based study on childhood asthma reported to date was conducted in an urban area, Nairobi, as the pilot phase of a research programme to investigate the prevalence and risk factors for the occurrence of markers of childhood asthma in Kenya and to examine the impact of urbanisation.[12] We report here on data gathered on the urban-rural differences in the prevalence of EIB among Kenyan

Table 1 Personal characteristics and lung function level of children studied

Personal characteristics	*Rural (n=485)*	*Urban (n=586)*
Percent boys	43.3%	49.7%*
Age (years)		
Mean (SD)	10.8 (1.1)	10.2 (0.7)*
Range	8–12	8–12
Height (cm)		
Mean (SD)	135.0 (6.7)	135.4 (7.0)*
Range	111–158	114–155
Weight (kg)		
Mean (SD)	28.2 (4.4)	28.1 (4.8)*
Range	20–54	17–50
Body mass (kg/m^2)		
Mean (SD)	15.3 (1.4)	15.2 (1.7)
Range	12.4–23.7	11.2–22.7
Lung function	*Rural (n=480)*	*Urban (n=577)*
FEV_1 (l)		
Mean (SD)	1.60 (0.29)	1.66 (0.28)*
Range	0.55–3.01	0.80–2.78
FVC (l)		
Mean (SD)	1.91 (0.35)	1.99 (0.34)*
Range	0.85–3.48	1.13–3.27
FEV_1/FVC (%)		
Mean (SD)	84.3 (8.0)	83.5 (6.9)
Range	44.9–155.2	57.6–100.0
Age, sex and size adjusted lung function level (FEV_1 and FVC)†		
FEV_1 (% predicted)		
Mean	100.7	99.4
95% CI	99.5 to 101.9	98.4 to 100.5
FVC (% predicted)		
Mean	100.5	99.6
95% CI	99.4 to 101.7	98.5 to 100.6

FEV_1 = forced expiratory volume in one second; FVC = forced vital capacity.
*Significant urban-rural differences ($p<0.05$).
†Internally adjusted using the study population's age, sex, height and body mass index.

school children and address the question of whether these differences can be explained by urban-rural differences in the distribution of known and suspected host and/or environmental determinants of childhood asthma.

Methods

STUDY DESIGN AND POPULATION

Details of the study design, target population, sampling procedures, and response rates for questionnaire information have been described elsewhere[13] but are briefly summarised here. A cross sectional survey design was used to target 10 year old (grade 4) school children attending five urban and five rural schools. The urban site selected was Nairobi, a city of over two million people with a high population growth arising both from migration from rural areas and from a high birth rate. English is the official language and Swahili the language of commerce. Several tribal languages are also spoken in the city. Nairobi supports both light and heavy industry and is a transport hub in the East and Central African region. There is also considerable motor vehicle pollution. The 136 public schools within the Nairobi urban area were classified by the school authorities into five neighbourhood social class levels as follows: upper middle, middle, low, very low (shanty), and the non-residential downtown Nairobi—that is, with no obvious neighbourhood social class. Stratified sampling was then used to select one school to represent each of five school neighbourhood social class levels. The rural site, Muranga district, approximately 80 km north of Nairobi, was selected because it enjoys a similar climate and is at the same altitude above sea level as Nairobi (approximately 6000 feet), altitude being a determinant of spirometric flow rates.[14] Three of the 68 schools were selected from an administrative sub-district comprising small rural holdings operated by peasant farmers, all from the same ethnic group (Kikuyu). The other two schools were selected from 56 schools within an administrative sub-district with large agricultural holdings operated by multinational companies. One of these schools was within a pineapple plantation and consisted of children from several Kenyan tribal groups, while the other school was in a coffee plantation and most of the children were from the Kikuyu tribe.

The 10 year old target age was achieved for the urban area (mean (SD) age 10.2 (0.9) years, range 8–15). However, in Muranga grade 4 children were older (mean (SD) age 11.4 (1.5) years, range 8–17). Because prevalence rates of EIB have been shown to decrease with age from childhood to adolescence,[5] the present analysis was limited to children aged 12 years or younger to focus on prepubertal children rather than those who had already undergone or were still undergoing puberty.

Table 2 Prevalence of exercise induced bronchospasm (EIB) by sex and area of residence

	Rural n (%)	*Urban n (%)*	*OR (95% CI)**
Boys and girls (n)	479	573	
EIB_{10}	63 (13.2)	131 (22.9)	1.96 (1.41 to 2.72)
EIB_{15}	29 (6.1)	56 (9.8)	1.68 (1.06 to 2.68)
Boys (n)	209	289	
EIB_{10}	24 (11.5)	68 (23.5)	2.37 (1.43 to 3.93)
EIB_{15}	13 (6.2)	30 (10.4)	1.75 (0.89 to 3.44)
Girls (n)	270	284	
EIB_{10}	39 (14.4)	63 (22.2)	1.69 (1.09 to 2.62)
EIB_{15}	16 (5.9)	26 (9.2)	1.60 (0.84 to 3.05)

*Prevalence odds ratio: urban versus rural.
EIB_{10}, EIB_{15} = 10% or more or 15% or more fall in FEV_1 after exercise.

QUESTIONNAIRE

A standardised English language questionnaire was administered at the respective school compounds by three interviewers who translated the questions freely into the language in which the parent or guardian of the participating child was most comfortable. Languages used included Kikuyu, English, Swahili, and Luo. This technique has been shown to give acceptable results in a study conducted in Johannesburg, another rapidly expanding urban centre in Africa in which up to seven tribal languages are in use in the urban workforce.[15] The research team actively followed up those parents who did not attend the school to complete the questionnaire. The questionnaire sought information on the child's respiratory health, personal and family history of asthma symptoms and allergy, as well as on the child's home environment.

SPIROMETRIC AND EXERCISE CHALLENGE TESTS

Spirometric tests were performed before exercise and at five and 10 minutes after exercise. The highest forced expiratory volume in one second (FEV_1) value from at least three acceptable trials (all within 100 ml of each other) before exercise was compared with the lowest from at least three acceptable trials (also all within 100 ml of each other) at five and 10 minutes after exercise in order to determine whether EIB had occurred. Spirometric tests were performed using a Vitalograph compact spirometer with the child in the standing posi-

Table 3 Host characteristics of children studied

	Rural	*Urban*
Family history*	n = 405	n = 508
Asthma symptoms n (%)	44 (10.9)	132 (26.0)†
Allergy n (%)	45 (11.1)	153 (30.1)†
Early life characteristics	n = 462	n = 553
Birth weight (kg)		
Mean (SD)	3.18 (0.56)	3.42 (0.66)†
Range	1.6–5.0	1.0–6.0
Prematurity n (%)	5 (1.1)	25 (4.5)†
Breast fed for less than 6 months n (%)	22 (4.8)	68 (12.3)†
Personal history of:	n = 461	n = 557
Asthma symptoms n (%)	13 (2.8)	53 (9.5)†
Allergy n (%)	19 (4.1)	97 (17.4)†
Eczema n (%)	17 (3.7)	38 (6.8)†
Allergic rhinitis n (%)	7 (1.5)	54 (9.7)†

*Family history of asthma symptoms was based on yes answers to the questions as to whether the child's natural father, mother or siblings had ever had wheezing or whistling in the chest with shortness of breath, asthma, or allergy.
†Significant urban-rural differences (p<0.05).

tion without any nose clip. Spirometer calibrations were done twice daily, morning and afternoon, according to ambient atmospheric pressure and temperature. Each child went through a vigorous six minute free running exercise in the school playground and the heart rate before and after exercise was recorded as a measure of the level of exercise stress achieved. The children ran in pairs and, after each circuit of the playing field, were urged by the staff responsible for the test to maintain their initial running pace. Standing height and weight without shoes were also recorded.

DATA MANAGEMENT AND ANALYSIS

All data collected were managed using a dBASE program. For the purposes of this study the outcome variable (EIB) was defined at two levels, EIB_{10} and EIB_{15}, representing a fall in FEV_1 after exercise of 10% and 15%, respectively, and was computed as follows:

$\{(\text{Pre-exercise } FEV_1 — \text{Post-exercise } FEV_1) / (\text{Pre-exercise } FEV_1)\} \times 100$

Table 4 Home environment characteristics of the children studied

	Rural n (%)	*Urban n (%)*
Smoking	n = 450	n = 537
Any smoker at home	241 (53.6)	178 (33.1)*
Shares bedroom with a smoker	27 (6.0)	31 (5.6)
Animals in the homestead	n = 461	n = 555
Cat	124 (26.9)	128 (23.1)
Dog	184 (39.8)	80 (14.4)*
Chicken	310 (67.1)	124 (22.3)*
Any animal	363 (78.6)	248 (44.7)*
Indoor animal†	10 (2.2)	67 (12.1)*
Animal sleeps indoor	52 (11.3)	66 (11.9)
Ventilation	n = 462	n = 555
Mean (SD) no. of windows	1.12 (0.35)	1.29 (0.46)*
Mean (SD) windows/room	4.27 (2.11)	4.67 (2.61)
Mean (SD) household crowding	1.97 (1.00)	2.34 (1.7)*
Sleeping area characteristics	n = 421	n = 586
Sleeps in kitchen/single room	40 (8.7)	125 (21.3)
Shares bed	337 (80.0)	331 (56.5)*
Domestic fuel‡	n = 462	n = 555
Wood	428 (92.6)	35 (6.3)*
Charcoal	144 (31.2)	437 (78.7)*
Kerosene	170 (36.8)	450 (81.1)*
Gas	17 (3.7)	271 (48.8)*
Electricity	6 (1.3)	194 (35.0)*
Socioenvironmental characteristics	n = 460	n = 554
Mean (SD) years of education§	7.38 (3.2)	11.3 (3.3)*
Exposed to motor vehicle fumes	n = 462	n = 555
Frequently	38 (8.2)	373 (67.2)
Sometimes	207 (44.8)	158 (28.5)
Rarely	215 (46.5)	24 (4.3)*

*Significant urban-rural differences ($p < 0.05$).
†Animal that spends most of the time indoors.
‡Domestic fuel ever used in the household.
§Years of education of parent/guardian (head of household).

The explanatory variable of major interest was area of residence (urban versus rural). Other determinants (also explanatory variables in this study) were grouped into three categories as follows: (1) personal characteristics (age, height, weight and sex); (2) host characteristics including a family history of allergy and/or of asthma symptoms as well as certain early life events (pre-term birth, birth weight, and duration of breast feeding); and (3) home environment characteristics including exposure to environmental tobacco smoke, animals in the homestead, characteristics of the child's sleeping area, kitchen characteristics as well as other socioenvironmental factors such as parental level of education and household crowding. Note that the term determinant is used here to indicate "any characteristic of an individual (whether constitutional, environmental or behavioural) that brings about a change in a health condition",[16] in this case EIB, regardless or not of whether it is causal. A determinant may increase or diminish risk—that is, it may be a risk or a protective factor.

Statistical analysis was performed using SAS statistical programs (version 6). Prevalence rates for EIB in rural and urban children were compared using χ^2 tests. Personal characteristics, host and home environment factors were also compared using χ^2 tests for categorical variables and *t* tests for continuous variables. A p value of ≤ 0.05 using a two tailed test was considered statistically significant. To examine the determinants of urban-rural differences in EIB, logistic regression models were constructed for each level of EIB (EIB_{10} and EIB_{15}). Explanatory variables, grouped under the categories mentioned above (personal, host and home environment characteristics), were serially included in these models to determine to what extent these factors explained the observed urban-rural differences. The decision to retain a variable in the final model was based on a priori judgment and/or change of the urban-rural odds ratio (OR) when the variable was added into the model. Effect measures were expressed as prevalence odds ratios with 95% confidence intervals (CI).

Results

PARTICIPATION RATES AND STUDY POPULATION CHARACTERISTICS

From a total of 1277 eligible children (603 urban, 674 rural), information on airway responsiveness to exercise was obtained for 98.7% and 93.6% of urban and rural children, respectively. The corresponding participation rates for the questionnaire were 94.2% and 89.6% for urban and rural areas, respectively.[13] Table 1 shows the characteristics of the two study populations. Boys made up a higher proportion of the study population in the urban area than in the rural area. Even with the restricted age range of up to 12 years, the urban children were significantly younger but taller than the rural children, though with comparable values for weight and body mass index. Compared with rural children, urban children also had higher baseline levels of FEV_1 and forced vital capacity (FVC), but not of the ratio

Table 5 Odds ratios (unadjusted and adjusted) with 95% confidence interval for urban versus rural residence for exercise induced bronchospasm (EIB_{10} and EIB_{15})

	Odds ratio* (95% confidence interval)	
	EIB_{10}	EIB_{15}
Unadjusted	1.96 (1.41 to 2.72) n= 1052	1.68 (1.06 to 2.68) n=1052
Adjusted for age and sex	1.80 (1.27 to 2.54) n=1052	1.43 (0.88 to 2.33) n=1052
Adjusted for age, sex, and host† characteristics	1.65 (1.10 to 2.47) n=792	1.04 (0.60 to 1.83) n=792
Adjusted for age, sex, host, and environmental‡ characteristics	1.21 (0.69 to 2.11) n=775	0.61 (0.28 to 1.33) n=775

*Odds ratios were calculated with rural children as the reference category.
†Host characteristics were breast feeding for less than six months and a family history of asthma symptoms.
‡Environmental characteristics were presence of animals in the homestead, ventilation (number of windows/room), parental education (years of education), use of biomass fuel and kerosene for cooking, and exposure to motor vehicle fumes on the way to school.

of FEV_1 to FVC. These differences were largely due to the size differences and, when internally adjusted for age, sex and size and expressed as percentage predicted, FEV_1 and FVC were actually slightly lower in the urban children than in the rural children.

PREVALENCE OF EIB

Table 2 shows the prevalence of EIB_{10} and EIB_{15} among children studied by sex and area of residence. For boys and girls combined, the prevalence rates for both EIB_{10} and EIB_{15} were significantly higher in the urban area than in the rural area, (OR 1.96, 95% CI 1.41 to 2.72 and 1.68, 95% CI 1.06 to 2.68, respectively). The heart rates after exercise did not differ significantly between the urban and rural children (mean (SD) 178 (17) versus 176 (17) beats per minute). Similar urban-rural differences in the prevalence of EIB_{10} and EIB_{15} were observed for boys and girls separately, but were more pronounced in boys than in girls.

URBAN-RURAL DISTRIBUTION OF HOST AND ENVIRONMENTAL DETERMINANTS

Table 3 shows the urban-rural distribution of the host characteristics of the children studied (a family history of asthma symptoms and/or allergy and certain early life characteristics). All are generally regarded as determinants of asthma and all were significantly more frequently reported in the urban children than in the rural children. Information on the child's personal history is also included in table 3 for descriptive purposes.

Table 4 shows the urban-rural distribution of the home environmental characteristics of the children studied. The presence of smokers and animals in the home was less frequently reported for urban than for rural children, but the urban children were more likely to have animals that spent most of the time indoors and to live in more crowded houses which, however, had more windows per room. They were also more likely to sleep in the kitchen but less likely to share a bed. Use of wood fuel was reported for the majority of rural homes while the use of all other fuels was reported more frequently for urban homes. Carpets were also exclusively reported for the urban area while urban homes were also less likely than rural homes to have a detached kitchen (not shown in table). Parents of urban children had, on average, more years of education than parents of rural children. Urban children were also more frequently exposed to motor vehicle fumes on their way to school than were rural children.

IMPACT OF URBANISATION ON EIB

Table 5 shows the results of modelling analysis directed at determining to what extent the urban-rural differences in the prevalence of EIB in this study could be explained by urban-rural differences in the determinants of EIB examined. Given urban versus rural residence, the unadjusted OR for EIB_{10} was 1.96 (95% CI 1.41 to 2.72). Adjusting for age and sex resulted in a reduction in the OR to 1.80 (95% CI 1.27 to 2.54). A further reduction in OR to 1.65 (95% CI 1.10 to 2.47) was observed after accounting for urban-rural differences in host factors (a more frequent family history of asthma symptoms) and early life events (breast feeding for less than six months), but the

Table 6 Relationship of exercise induced bronchospasm (EIB_{10}) to selected demographic, host and environmental characteristics

Characteristic	Odds ratio (95% confidence interval) Undjusted	Adjusted for area of residence	Adjusted for all other characteristics in the table
Sex*	1.00 (0.73 to 1.36)	1.04 (0.76 to 1.43)	1.13 (0.77 to 1.65)
Age	0.75 (0.63 to 0.89)	0.82 (0.68 to 0.99)	0.84 (0.67 to 1.06)
Breast fed for less than 6 months	1.27 (0.78 to 2.05)	1.09 (0.67 to 1.79)	1.07 (0.63 to 1.79)
Family history of asthma symptoms	1.61 (1.09 to 2.36)	1.40 (0.95 to 2.08)	1.50 (0.99 to 2.28)
Animal in the homestead	0.62 (0.45 to 0.85)	0.74 (0.52 to 1.04)	0.76 (0.51 to 1.14)
Ventilation (windows/room)	1.55 (1.08 to 2.22)	1.36 (0.94 to 1.97)	1.06 (0.68 to 1.65)
Cooking fuel			
Biomass	0.58 (0.37 to 0.90)	0.73 (0.46 to 1.15)	0.64 (0.37 to 1.11)
Kerosene	1.50 (1.07 to 2.10)	1.80 (1.27 to 2.55)	1.17 (0.74 to 1.84)
Parental education (years in school)	1.06 (1.01 to 1.11)	1.02 (0.97 to 1.07)	1.01 (0.95 to 1.08)
Exposure to motor vehicle fumes on the way to school			
Frequently	2.05 (1.28 to 3.26)	1.27 (0.72 to 2.27)	1.18 (0.62 to 2.25)
Sometimes	1.97 (1.22 to 3.17)	1.60 (0.97 to 2.64)	1.31 (0.74 to 2.31)
Rarely	Reference	Reference	Reference
Area of residence (urban vs rural)	1.96 (1.41 to 2.72)	—	1.21 (0.69 to 2.11)

*Odds ratios calculated using boys as reference.

differences remained significant. However, simultaneous adjustment for age, host and environmental factors resulted in a further reduction in the OR which was no longer significantly different from 1.0 (OR 1.21, 95% CI 0.69 to 2.11). Similar results were obtained for the smaller number of children with an EIB_{15} value (85 versus 194), though only the unadjusted OR was statistically significant. When the results were analysed separately by sex, similar trends were observed for both EIB_{10} and EIB_{15} for boys, and for EIB_{15} for girls (not shown). However, for EIB_{10} for girls the adjustment for host and environmental factors did not reduce the OR which, in the fully adjusted model, still remained above 1.0, though was non-significant.

Finally, we assessed the individual contribution of the various determinants of EIB to the urban-rural gradient for EIB observed in our study. The results of these analyses can be seen in table 6 which shows, for each determinant, the crude OR, the OR adjusted for area of residence, and the OR for the fully adjusted model. For sex there was an increase (non-significant) in the OR adjusted for area of residence and the fully adjusted OR. This was not unexpected because there were more girls among the rural than among the urban school children, and they were on average older and therefore more likely to have undergone puberty when male:female ratios for asthma reverse from above 1 to below 1. This is in keeping with the observation that, in childhood, boys exhibit the markers of asthma more frequently than girls whereas the reverse occurs in adolescence.[19] There was also an increase in the OR adjusted for area of residence and the fully adjusted OR for breast feeding for less than six months, another determinant more common among urban than rural school children (see table 3).

Discussion

The main findings in this study are the high rates for EIB in Nairobi school children, rates approximately 1.6 times greater than those recorded for children resident in rural areas. These rates appear to be largely, though not completely, explained by the urban-rural differences in recognised host and environmental determinants of childhood asthma. The prevalence rates for EIB among urban children in this study are similar to those reported for urban children in our pilot study carried out in 1990/91[12]: for EIB_{10}, 21.3% versus 22.9% and for EIB_{15}, 9.8% versus 10.5% in the pilot and present study, respectively. The comparability of rates recorded three years apart, together with the high participation rates achieved in the present study, strengthens our confidence in the validity of the estimates of prevalence levels of EIB in both rural and urban Kenyan school children, as well as the urban-rural differences reported here.

To set the findings in the context of other studies carried out in African children,[8–10 17 22] these are summarised in table 7. Two points are of interest: firstly, the prevalence of EIB in Kenyan children, both urban and rural, is considerably higher than that reported in any of the other studies of African children and, secondly, the overall urban-rural gradient in Kenya is comparable to that reported in a recent study in Ghana[10] but considerably lower than those reported in a study in South Africa in 1979[8] and in Zimbabwe in 1991.[9] Methodological differences which may have contributed to these between-study differences in EIB in African children include (1) population selection, in particular age and sex distribution,[5 17–19] (2) methods used to measure EIB (peak flow or FEV_1), (3) the time after effort that the forced expiratory flow was measured (from three to 15 minutes) and the number of trials carried out, and (4) the fall in flow rates after exercise taken to indicate EIB (10%, 12.5%, or 15%). While caution must obviously be exercised in interpreting these findings, the results suggest that the rates for EIB in urban Kenyan children are higher than any other

Table 7 Exercise induced bronchospasm (EIB) in African children

Reference	*Country*	*Region or group*	*n*	*Age range (in years)*	*Prev*	*EIB definition*	*Lung function index*
van Niekerk (1979)[8]	S Africa	Urban	695	6–9	3.17%	15% fall in PEFR or FEV_1 after 6 min running exercise	PEFR and FEV_1 before and at 3 and 5 minutes after exercise
		Rural	671		0.14%		
Terblanche (1990)[17]	S Africa	White (urban)	698	6–20	5.87%	10% fall in FEV_1 after 6 min running exercise	FEV_1 before and at 10 minutes after exercise
		Coloured (urban)	694		4.05%		
Keeley (1991)[9]	Zimbabwe	Urban high SES	726	7–9	5.8%	15% fall in PEFR after 6 min running exercise or 15% increase in PEFR after bronchodilator	PEFR before and at 5 and 10 minutes after exercise
		Urban low SES	642		3.1%		
		Rural	687		0.1%		
Ng'ang'a (1992)[12]	Kenya	Urban	408	9–12	10.5%	15% fall in FEV_1 after 6 min running exercise	FEV_1 before and at 5 and 10 minutes after exercise
					21.3%	10% fall in FEV_1 after 6 min running exercise	
Addo Yobo (1997)[10]	Ghana	Urban rich	599	9–16	4.7%	12.5% fall in PEFR after 6 min running exercise	PEFR at baseline, 5 and 8 minutes after exercise
		Urban poor	220		2.2%		
		Rural	270		2.7%		
Present study	Kenya	Urban middle class	331	8–12	10.3%	15% fall in FEV_1 after 6 min running exercise	FEV_1 at baseline, 5 and 10 minutes after exercise
		Urban poor	242		9.1%		
		Rural plantation*	140		12.9%		
		Rural peasant	339		3.2%		
Present study	Kenya	Urban middle class	331	As above	22.1%	10% fall in FEV_1 after 6 min running exercise	As above
		Urban poor	242		24.0%		
		Rural plantation*	140		21.4%		
		Rural peasant	339		9.7%		

SES = socioeconomic status; FEV_1 = forced expiratory volume in one second; PEFR = peak expiratory flow rate.
*Two schools within large scale farming estates operated by multinational agro-based companies.

reported from Africa and, had we used a less rigorous definition such as EIB_{10} as used in the Cape Town study,[17] rates in the Kenyan children would have been almost doubled.

The reasons for the higher rates in Kenyan children are not obvious. Methodological differences (in the age group targeted and/or the definition of EIB used) are unlikely to be the full explanation. A factor which may be important is whether the parents and/or the children were raised in the urban environment and therefore subjected since childhood to urban pollution by industrial and motor vehicle emissions, both of which are features of the city of Nairobi. Urban-rural differences in diet and in the frequency and nature of childhood infections, neither of which were examined in this study, have also been implicated.[18] In other parts of the world residence at high altitudes such as those of the Kenya study sites (higher than that of any of the other study sites cited in table 7) is usually associated with lower rather than higher rates of asthma, and this is attributed to the fact that house dust mites do not survive the low temperatures and relative humidity of cold winters.[18] In tropical Africa, however, this does not appear to be so. For instance, house dust mite allergen was isolated from mattress dust of asthmatic patients in a report from the Nigerian Savanna in 1975.[20] More recently, in 1993, rates of 40% for skin positivity to mite allergen were reported among asthmatic patients attending an allergy clinic in Nairobi,[21] rates not all that much lower than the 58% reported among asthma patients in Ibadan, Nigeria, situated on a coastal plain.[22]

In this study the overall gradient for urban-rural differences in EIB was between 1.6 and 1.7, comparable to that found in the 1997 study in Ghana,[10] but considerably less than the gradient of more than 20 reported in the Zimbabwe study,[9] whether the comparison was with urban rich or poor, and the South African study[8] which compared Xhosa children living in a Cape Town suburb (shanty town) with Xhosa children living in rural Transkei. The high urban-rural gradients in the latter two studies were essentially driven by very low rural rates. For the purposes of comparison we stratified the data in our study along similar lines (see table 7). The prevalence rate for EIB_{15} among children attending the three rural schools located in peasant villages was 3.2%, comparable to the rate of 2.7% found in rural Ghana,[10] but considerably higher than the rates of under 0.2% reported from rural South Africa[8] and Zimbabwe.[9] Certainly in the South African study carried out in 1979,[8] and probably in the Zimbabwe study carried out in 1991,[9] the children from the rural areas studied are described as following a much more traditional lifestyle than was the case for the children from the rural peasant villages in our study. The prevalence rate for EIB_{15} was 12.9% for the children from the schools located within the large agro-industrial plantations, comparable to the rate of 10.3% among the Nairobi children studied. The children from these agro-industrial plantation areas follow an even less traditional lifestyle than that of children attending the peasant area schools, and are also exposed to agricultural aeroallergens—for example, from coffee plants in bloom—and/or to pesticides and fungicides found in other studies to be significantly associated with higher asthma rates—for example, among Canadian farmers.[23] The role of outdoor agricultural allergens and irritants, not directly examined in the present study, clearly needs further investigation.

In the Zimbabwe study the urban sample was also stratified into high and low socioeconomic status (rich and poor) so, for the purpose of comparison, we stratified the urban schools into urban middle class (three schools) and urban poor (two schools). Not only were the rates of EIB_{10} much higher in our study, but the rates for the urban poor and urban middle class (10.3% versus 9.1% for EIB_{15}) were similar, in contrast to the almost two fold differences between urban rich and urban poor reported in Zimbabwe[9] (5.8% versus 3.1%) and in Ghana[10] (4.7% versus 2.2%). One reason may be the nature of urban pollution in Nairobi, a combination of heavy industrial and vehicle pollution.

In their discussion the authors of the three studies that have found urban-rural differences in EIB in Africa all invoke the differences in living environment, in lifestyle, and in outdoor pollution as potential explanations for the observed differences.[8–10] The statistical model used in the present study confirmed the importance of environmental factors, but also implicated host factors such as a family history of asthma symptoms and allergy. In other words, our results imply an increased susceptibility for asthma in urban compared with rural Kenyan children. One can only speculate as to why this might have occurred. Given the importance currently being attached to early life events when patterning of the infant's immune system is thought to take place,[24] and to the role of infections, some of which appear to increase and others to decrease the risk of asthma,[24] the age of migration to an urban area may be important. On the one hand, it is likely that most of the parents of the urban children in our study were not born in the Nairobi area but had moved there in search of employment. Thus, the higher reported rates of parental history of asthma symptoms and/or allergies, if due to urban factors, must be attributed to exposures encountered in early adulthood. On the other hand, it is likely that most of their children were born and raised in the Nairobi area and were thus exposed to indoor and outdoor urban allergens, pollutants, and irritants from birth and in the first year of life. In turn, these exposures, singly or in combination, may have tipped the balance of their immune system from a Th1 (non-asthmatic) to a Th2 (asthmatic) phenotype response.[24] In addition, birth in the Nairobi area might have resulted in a change in phenotype in the urban children in the present study who exhibited EIB but for whom a family history was not reported.

Evidence of the importance of the time of migration from a rural to an urban area was found in a recent study carried out in Ethiopia[25] in which self-reported rates for asthma among 9844 residents of all ages of Jimma (a non-industrialised town of approximately 80 000 inhabitants with no major industry and little motorised transport) were three times higher than those among 3032 residents of three rural subsistence communities in the area (3.6% versus 1.3%). There was also an urban-rural gradient for children under 10 years of age (2.4% versus 1.1%). However, the age of onset of asthma for all ages appeared to be around 10 years before the study was conducted, suggesting that changes in the urban community environment had occurred at that time. Although the authors were unable to identify a single key determinant, they pointed to several factors meriting further study, including changes in housing style, immunisation, and the introduction of insecticides into common use. Data on whether the children or adults resident in Jimma were born there or, if not, when they migrated, were not given in the report, nor were they collected for Nairobi residents in the present study, but such information should clearly be gathered in any future study.

Even though in the present study the susceptibility to allergic conditions, as reflected in the reported family history, was higher in urban children than in rural children, rural children were more frequently exposed to sources of allergens and irritants that may exacerbate or maintain the airway inflammation, thought to be responsible for asthma symptoms and airway hyperresponsiveness. However, closer examination of our data suggests that circumstances that concentrate indoor aeroallergens and irritants were in fact more likely to be found in the urban than in the rural area. These include indoor pets and carpets, more crowding, and sleeping in the kitchen. In other words, the urban environment, in addition to increasing the susceptibility of children to develop EIB—that is, increasing the incidence of childhood asthma—may also have increased its severity due to sustained and heavier exposure to allergens and irritants, indoor and outdoor, in the urban environment.

When the present study was planned, environmental factors, particularly those related to the indoor environment, were considered the key and probably the most important determinants of the increasing rates of childhood asthma in westernised countries[26 27] and, plausibly, in African societies recently urbanised.[28] However, opinion is now shifting to the view that a change in population susceptibility may also be important[11] and that this is attributable to prenatal and/or early life events which prime the infant's immune system, including certain infections,[18] parasitosis,[29] vaccination and/or immunisation.[30] Parasitosis was thought to be responsible for the low rates of childhood asthma reported in studies carried out in Africa in the 1960s and 1970s,[6] the postulated mechanism being a potentiated IgE response to parasite allergens and a significant non-specific IgE component which acts as a blocking antibody to modulate allergic reactivity.[29] The potentially protective role of certain infections in infancy, including parasitosis, was not investigated in the present study but should be a component of any future studies.

In conclusion, a speculative but not implausible scenario to explain the findings of the current study might be as follows. Early life events such as exposure to certain infections occurring in the first year of life (not studied here) as well as exposure to indoor aeroallergens and environmental tobacco smoke are likely to have increased the incidence of asthma among urban infants who also appear to be genetically more susceptible to developing asthma and allergies than rural children, particularly if premature and breast fed for less than six months. In addition, sustained exposure to indoor aeroallergens and irritants is likely to have increased the severity of EIB exhibited by urban children, thus increasing the prevalence of its symptom markers. In other words, an increase in both prevalence and incidence of EIB is likely to explain the higher rates in Kenyan school children resident in Nairobi compared with those resident in rural areas. Extrapolation of this scenario to asthma symptoms and diagnosis should, however, be made with more than a little caution since EIB, though a well accepted marker of asthma, particularly in children, is not diagnostic. Future studies on the determinants of childhood asthma in Kenya, if not in Africa in general, should focus on clarifying not only the role of environmental factors (such as the use of agricultural pesticides and fungicides in rural areas and of vehicle pollution in rural and urban areas), but also the role of early life events including infections (bacterial, viral or parasitic), vaccination, and immunisation, none of which were addressed in this study.

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