Asthma in African cities

P Cullinan

Does African city life provoke asthma? In this issue of Thorax Ng’ang’a and colleagues present the findings from a meticulous cross sectional survey of 10 year old schoolchildren in Kenya.1 One quarter of 573 children living in the capital, Nairobi, developed a 10% fall in baseline forced expiratory volume in one second (FEV₁) after exercise—approximately twice the prevalence among a similar number of children living in “rural” locations. The difference was more pronounced in boys but when both sexes were combined it disappeared after statistical adjustment for a variety of “host” and “environmental” characteristics. In both rural and urban children the prevalences of exercise provoked bronchial reactivity were considerably higher than in earlier studies in Africa; these are helpfully summarised by the authors of the present survey.

Bronchial hyperresponsiveness measured using an exercise stimulus is, not, of course, necessarily synonymous with “asthma”. Although a reasonable specificity (and somewhat lower sensitivity) in children has been demonstrated against conventional indices of asthma,2 it is worth remembering that these comparisons have been made in communities with very different disease patterns and cultural experiences from rural or newly urban Africa. Atmospheric conditions too, particularly air humidity and temperature,3 are important determinants of test results and, where geographical differences are being claimed, it is comforting to know that the measurements were made under similar ambient conditions. On the assumption, however, that the test has equivalent validity in rural and urban Kenyan children, these results indicate a doubling of asthma risk among the latter and, in part, circumvent the seemingly intractable linguistic and conceptual problems associated with symptom based definitions of the disease—problems by no means confined to the developing world.

At face value, and in the context of similar findings elsewhere on the continent, they suggest that there is something asthmagenic about living in an African city. Fitting an explanation to the pattern is fraught with difficulty and tends to end in vague generalisations about “Westernisation”, a catch-all phrase which covers the host of theories familiar to anyone who has attended a gathering of asthma epidemiologists. Westernisation, it is argued, encourages an “increase in susceptibility”, a process whose pattern (if not determinants) appears to have been mirrored in European populations over the past 50 years. In circumstances where a large proportion of the population is at risk, quite small shifts in one or more important exposures—in this case presumably environmental—can result in large changes in disease frequency. Viewed in this way, distinctions between case severity and prevalence or incidence are not particularly helpful and will be difficult to sustain if we ever learn to perceive asthma as a continuous rather than a dichotomous state.

“Westernisation”, it seems, can be surprisingly rapid; in a survey of Ethiopian town dwellers, admittedly based on recall, asthma seems to have emerged only some 10 years earlier.4 Confusingly, the higher urban prevalence of wheeze was accompanied by a lower rate of IgE sensitisation to a house dust mite allergen. In former eastern Germany, rates of atopy in Leipzig have approached those in more “western” Munich over an even shorter period.5 In this Kenyan population, however, the rate of exercise induced bronchospasm was no higher in the Nairobi schools than five years previously, perhaps indicating that much of the process has already taken place.

Ng’ang’a and colleagues suggest that the “urban” risk is largely accounted for by a cocktail of personal (age and sex), host (a family history of asthma and a short period of breast feeding), and environmental (animal contact, home ventilation, parental education, domestic and outdoor exposure to respiratory irritants) factors. From a descriptive study with several potential risk factors under consideration it is not easy to sort out which is important or which are “determinant” as opposed to “confounding” factors—particularly where there has been a partially subjective approach to variable inclusion in the final logistic model. However, at least three possible explanations are worth consideration. The first is that children brought up in traditional villages have a systematically greater rate of early infection than those from the city, thus deriving an immunological milieu inimical to the development of Th2 associated allergic responses. (The role of infection in IgE associated allergy is, of course, considerably complicated where there is endemic parasitosis.) Supporting data seem few but it is unlikely that poorer urban children, at least, endure less domestic crowding than those in villages. Rates of other contagious conditions are at least as high in African villages as in those from the countryside.6 Notably, in the present study there was no difference in the rates of bronchial hyperresponsiveness across the social gradient of children in Nairobi. The second possible explanation concerns the issue of diet and here differences between urban and rural Africans have been long established, with the latter enjoying a diet which is broadly higher in carbohydrate and fibre and lower in animal protein and fat. Other dietary differences, perhaps more pertinent to asthma, deserve further consideration; salt intake, for example, has been reported to be higher in asthmatic than in non-asthmatic children in Nairobi.7 Although the present study provides no information on diet, anthropometric differences between the urban and rural children were very small. The third explanation is the issue of respiratory irritation. Exposure to the combustion products of indoor fires is notoriously high in many African village homes but exposure to traffic generated pollutants is largely confined to those living in the cities. In western Europe the importance of the latter in childhood asthma


has been doubted but it is worth noting that vehicle pollut-
ant levels in less developed cities can be staggering-ly high. In the current study there was a suggestion of an exposure-
response relationship with a trend in risk across three cat-
ergories of “motor vehicle fume” exposure.

To what extent is the distinction between “urban” and “rural” useful in the study of asthma? In British populations it appears to have little significance, presumably because there is relatively little heterogeneity in exposure. In Germany it seems to depend on which city is being referred to. Where each speaks a different language,1 it is salutary to remember that the distinction has not served others well. Large bowel disorders, appendicitis,10 hypertension,11 and diabetes12 have all been reported to be more common among urban than rural Africans but, with the possible exception of the first, none of these differences has been explained satisfactorily. Conceiving a single aspect of urbanisation which accounts for all is not easy and generally proceeds no further than comments about “unhealthy lifestyles”.13 Intriguingly, in the study by Ng’ang’a et al bronchial hyperresponsiveness among the children living in plantation villages appeared to be as common as in the city. A useful further analysis might include comparisons of groups defined by their frequency of hyperresponsiveness rather than place of residence.

Do these findings provide useful lessons for the determin-
ants of asthma in other parts of the world and are further studies of asthma in Africa justified? Unhappily, in a contin-
tent with more than its share of other health problems, a condition that is now affecting one quarter of urban schoolchildren seems also to warrant attention. To “western” epidemiologists of asthma who may have exhausted much of the remaining variation in disease and exposure rates, there is in Africa the very real attraction of clear differences in disease frequency. Although “urban-rural” variability is a reasonable place to start, it has prob-
able outlived its usefulness. Future surveys might address specific hypotheses incorporating more elaborate, but not necessarily more expensive, measurements of relevant exposure and include issues of migration and survival using populations with wider age spans than those captive in the classroom. Perhaps some anthropological insight would also be helpful; do, for example, questions concerning a personal or family history of wheeze have the same mean-
ing to a city dweller and one living in a village, particularly where each speaks a different language?

P CULLINAN

Department of Occupational and Environmental Medicine, National Heart & Lung Institute, Maresfield Road, London SW3 6LY, UK

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