URBANISATION AND ADOLESCENT RISK BEHAVIOUR

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Objective. To investigate whether there is an association between the length of time lived in an urban area and selected adolescent risk behaviours.

Design. Cross-sectional survey in which students completed an anonymous, confidential questionnaire.

Setting. Four high schools in black communities in the Cape Peninsula, South Africa.

Participants. A sample of 1 296 students obtained by multistage cluster sampling.

Main outcome measures. Selected risk behaviours.

Results. There is a relationship between urbanisation and certain risk behaviours. The following risk behaviours were associated with urbanisation: use in the previous month of alcohol, cannabis, and cannabis mixed with Mandrax; being a victim of violence; perpetration of an act of violence; and suicidality. Conversely, participation in sexual intercourse and solvent sniffing in the previous month were not associated with urbanisation.

Conclusion. Urbanisation is associated with an increase in the prevalence rates of some risk behaviours. Mental health promotion efforts may be informed by further research aimed at the identification of: (*i*) the characteristics of risk behaviour that determine whether it is associated with urbanisation; and (*ii*) where applicable, the specific aspects of the urbanisation process that contribute to an increase in risk.

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Widespread migration from the countryside to the city is a key social characteristic of the developing world. In 1975, about one-quarter of the global population lived in urban areas.[1] This proportion was expected to increase to about 40% by the year 2000, an increase of 60%.[1,2]

When compared with other age groups, young people are disproportionately likely to have migrated from rural to urban areas. The proportions of migrants aged between 15 and 29 years from rural areas of Punjab, Sudan and Ecuador have been

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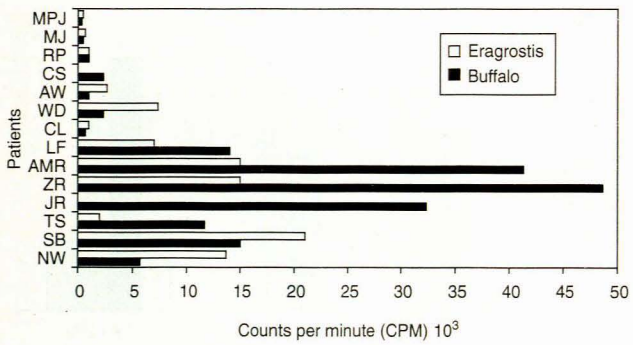

Fig. 8. T-cell proliferation to pollen extracts of buffalo and Eragrostis *grasses. Values shown are mean cpm of triplicate wells as determined by ^{3}H-Th incorporation. The top 7 individuals are asymptomatic, SPT +ve, while the rest are grass-sensitive individuals.*

and many also react to Kikuyu grass. The major buffalo allergen is a 34 kD periodate-sensitive glycoprotein, pI 5-7, which is consistent with the principal allergenic component of most grasses, the group 1 (Gp 1) allergen.[9-12] None of the sera exhibited a binding pattern unique to a single species, although 4:32 volunteers, with negative Bermuda SPTs, exhibited negligible binding to a Bermuda grass extract on immunoblotting.

Cross-reactivity among the four species studied has been clearly demonstrated by inhibition of ELISA, RAST, and immunoblotting. *Eragrostis* was able to abrogate IgE binding by most allergic sera, and to abolish binding in some patient sera. It is therefore an important allergenic grass, considering its year-long pollination period and widespread distribution in southern Africa. *Eragrostis* is a logical candidate for a desensitising vaccine for the region.

It was interesting to find that Belgian grass-sensitised individuals had cross-reactive IgE that binds to components on immunoblots of the African indigenous buffalo, Kikuyu and *Eragrostis* grasses. These individuals had not been exposed to the subtropical grasses. It has been found that 80% of the South African patients sensitive to buffalo have concordant sensitivity to rye. Local subjects also have specific IgE to timothy grass pollen in CAP-RASTs, confirming the presence of cross-reactive epitopes since timothy does not occur locally.

Recent reports have implicated cross-reactive sugar molecules in IgE binding,[13,14] as first reported for the major allergen of Bermuda grass, *Cynd1*,[10] and the 60 kD allergen, BG 60,[15] of Bermuda grass. We have demonstrated carbohydrate moieties in Kikuyu, buffalo and *Eragrostis* extracts, and periodate treatment of these local pollen extracts resulted in reduced patient IgE binding, as well as the monoclonal antibody, anti-*Lol p 1* (2) in ELISAs. The carbohydrate moiety of B-cell epitopes may conceivably contribute to the cross-reactivity between these Panicoid and the unrelated Pooideae grasses.

Interspecies allergens have been proposed to account for the cross-reactivity between unrelated fruits and vegetables,[16-18] such as the ubiquitous panallergen profilin, an actin-binding protein, which plays an important role in pollen germination and tube growth, which is recognised by 20% of pollen-sensitive patients. Reactivity to the 14 kD protein in buffalo and *Eragrostis* extracts has been shown by many of our allergic sera. The presence of interspecific calcium-binding allergens was also demonstrated by inhibition of patient IgE binding in the presence of ethylenediamine-tetraacetic acid (EDTA) to Kikuyu, buffalo, and *Eragrostis* pollen extracts (data not shown).

The rising levels of pollution, comprising diesel exhaust particles (DEPs), industrial emissions and wood smoke from the burgeoning informal settlements, are reported to enhance both IgE production and cytokines associated with airway inflammation.[19,20] DEPs also increase the availability of allergens of respiratory size, as pollen grains have been shown to aggregate on these airborne particles,[21] while gaseous pollutants facilitate the release of the allergenic molecules.[22]

This confirms the important role played by buffalo and other indigenous grass pollens in pollinosis in this region. The cross-reactivity demonstrated between all four grasses underlies the importance of the dominant Panicoid family as a pollen sensitiser in southern Africa. Current testing panels and immunotherapy vaccines for this region are deficient in representatives from these important grass pollen families. Diagnostic and therapeutic strategies should take this into consideration. We propose that buffalo, Kikuyu and *Eragrostis* be included in the SPT panels, and further studies are underway to develop appropriate desensitising vaccines for the region.

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