



INFANT MORTALITY IN THE RURAL HLABISA HEALTH DISTRICT — ESTIMATION USING THE PREVIOUS BIRTH TECHNIQUE

To the Editor: The infant mortality rate (IMR) is recognised as being a key health factor indicator. However, owing to a lack of appropriate surveys there are few reliable estimates of the IMR in rural South Africa. Estimates of the IMR range from 15 to 71 per 1 000 total births, depending on setting and methodology used.¹

One validated methodology is the 'previous birth technique', in which women attending antenatal clinics are asked about their previous birth experience. Those with a previous live birth are asked whether that child is still alive, and the West model life table is used to estimate the IMR from these data (D Bradshaw — unpublished report, Medical Research Council). The same tables provide estimates of the under-5 mortality rate.

Hlabisa health district in rural northern KwaZulu-Natal is home to approximately 250 000 Zulu-speaking people. The Hlabisa Nursing Service provides a mobile primary health care service through 16 mobile clinic points across the district. Our previous work^{2,3} suggests that these clinics serve a population representative of the district as a whole. A previous survey indicates that approximately 95% of pregnant women in the district receive antenatal care.⁴

For a 1-year period ending July 1998, all women attending the clinics for antenatal care were asked about their previous birth experience. Of 708 women whose last baby had been born alive, 42 reported that child to have since died. The estimated IMR is therefore 53 per 1 000 total births (95% confidence interval (CI) 42 - 71), and the under-5 mortality rate is estimated at 70 per 1 000 population (95% CI 53 - 98).

These are the first estimates of the IMR and the under-5 mortality rate in Hlabisa. They provide an important baseline against which changes in health status can be measured. It is predicted that the HIV epidemic is having a substantial impact on infant mortality in Africa.⁵ The relatively high rate reported here may already reflect this in part, as the HIV epidemic⁶ and its impact⁶ is already well established in KwaZulu/Natal. Simple and cheap survey methodologies such as this can provide important information on health status.

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D8/17 IN OBSESSIVE-COMPULSIVE DISORDER AND TRICHOTILLOMANIA

To the Editor: The finding that patients with Sydenham's chorea often demonstrate obsessive-compulsive disorder (OCD) has fostered increased interest in possible neuro-immunological mechanisms in OCD.¹ Increased expression of the B-lymphocyte antigen D8/17, which has been hypothesised to be a genetically inherited trait marker for susceptibility to rheumatic fever, has recently been demonstrated to be higher in OCD patients than in normal controls.^{2,3} To date, however, D8/17 expression has been studied in few psychiatric control populations.

Some authors have suggested that trichotillomania (TTM) is related to OCD insofar as it involves repetitive ritualistic behaviours and responds selectively to the serotonin reuptake inhibitor, clomipramine.⁴ Nevertheless, there are also many phenomenological and neurobiological differences between OCD and TTM.⁵ Of note, however, is a case report of Sydenham's chorea associated with hair-pulling,⁶ and reports that TTM may be exacerbated after streptococcal infection.⁷

We assessed D8/17 expression in 17 TTM patients (3 male, 14 female), 12 OCD patients (4 male, 8 female), and 22 normal controls (9 male, 12 female). All subjects were adults of Afrikaner descent. Eight of the 12 OCD patients and 14 of 17 TTM patients had onset of symptoms in childhood or adolescence. The Structured Clinical Interview for Axis I Disorders (SCID-I) was used to diagnose patients, and to exclude current or past psychiatric disorders in controls. D8/17 expression was measured using an indirect immunofluorescence assay, as described elsewhere.⁸

Analysis of variance of D8/17 levels showed a significant group difference ($F = 4.06$, $P = 0.02$). The average percentage of B cells expressing the D8/17 antigen was found to be significantly higher in patients with OCD ($29.2 \pm 15.0\%$) than in either TTM patients ($16.6 \pm 13.6\%$, $P = 0.03$) or controls ($17.9 \pm 10.5\%$, $P = 0.02$). Similarly, positive D8/17 expression (defined as having 12% or more D8/17+ cells) appeared more common in those with OCD (11/12) than in either TTM patients (10/17, $P = 0.06$) or controls (14/22, $P = 0.08$), although there was only a trend towards statistical significance.



These findings indicate that there may be differences in any possible neuro-immunological mechanisms underlying OCD and TTM. However these findings need to be qualified by emphasising the small sample size used, and by noting the possibility that there may be an overlap between OCD and a particular subgroup of TTM. While the mean percentage of B cells expressing D8/17 in OCD found here was similar to that noted in previous work on childhood-onset OCD,^{2,3} the relatively high levels obtained in both our controls and TTM patients^{2,3} may indicate that this is not an uncommonly expressed antigen in certain population groups, such as among Afrikaners.

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