372 THE MEDICAL JOURNAL OF AUSTRALIA Vol 161 19 September 1994

# Should Aboriginals in the "Top End" of the Northern Territory be vaccinated against hepatitis A?

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***Objective:*** **To determine the level of immunity to hepatitis A virus infection in rural Australian Aboriginal populations in the "Top End" of the Northern Territory.**

***Methods:*** **A total of 344 sera, for which details of donors' age, sex and domicile were available, were collected and tested for hepatitis A total antibody in a delinked seroprevalence study.**

***Results:*** **Overall, 337/344 samples (97.97%) tested positive for hepatitis A total antibodies — 18/20 samples (90%) in the 1–5 year age group; 85/88 (96.6%) in the 6–10 year age group; 98/98 (100%) in the 11–15 year age group; 32/33 (97.0%) in the 16–20 year age group and 104/105 (99%) in the older than 20 year age group.**

***Conclusion:*** **Hepatitis A is hyperendemic in the rural Aboriginal communities studied and the virus is acquired predominantly in the first five years of life. Symptomatic hepatitis A infection is uncommon in this population. We suggest that hepatitis A vaccination for rural Aboriginal children is not indicated as it would not reduce clinical disease rates and may produce a cohort whose immunity could decrease over the following 10 years. Although vaccination is appropriate for non-immune individuals working in remote communities, emphasis must be placed on the inequities in health infrastructure and education underlying the high transmission rates in Aboriginal children.**

**(Med J Aust 1994; 161: 372-373)**

Hepatitis A virus (HAV) infection is now preventable by vaccine. Although improved sanitation and personal hygiene have reduced its incidence in most urban areas of Australia, foci of infection persist and, in special circumstances, HAV vaccination is likely to be beneficial. Recent epidemics of HAV in Melbourne and Sydney[1,2] highlight the fact that the disease is still present in the community, with substantial morbidity.

It is less well known that HAV is endemic in the Northern Territory (NT), where the incidence was 88 per 100 000 in 1992 compared with the Australian average of 13 per 100 000.[3] Clinical disease is almost exclusively confined to the non-Aboriginal population and is an important occupational hazard for medical, nursing and child-care staff. Informal reports and occasional surveys have suggested that the disease is hyperendemic in rural Australian Aboriginal populations, but before this study no detailed HAV seroprevalence data have been published from the NT.

## Methods

Permission for an anonymous, delinked seroprevalence study was obtained from the Joint Institutional Ethics Committee of the Royal Darwin Hospital and the Menzies School of Health Research, which operates to National Health and Medical Research Council guidelines and is advised by an independent Aboriginal subcommittee. Sera collected in various surveillance programs were obtained from Disease Control in Darwin and the Menzies School.

Samples were selected to only include sera from Aboriginal people living in remote communities in the "Top End" of the NT. No clinical data apart from age, sex and region were available because of the design of the study. Duplicate testing for hepatitis A specific total antibody was performed at the Victorian Infectious Diseases Reference Laboratory by a microparticle competitive enzyme immunoassay (IMX HAVAB, Abbott Diagnostics, North Chicago, Ill., USA).

## Results

A total of 346 samples were tested. Two results from individuals aged less than one year were not included to eliminate the possibility of detection of maternal antibody. There were 203 males and 141 females (mean age 18.4 years). The results are summarised in the Table.

Hepatitis A total antibody was detected in 337/344 samples (97.97%; 95% confidence interval (CI), 95.85%–99.18%). There was no antibody in 7/344 samples (2.03%; CI, 0.8%–4.15% [four males, ages 1, 7, 7 and 16 years; and three females, ages 1, 9 and 43 years]).

## Discussion

These results confirm anecdotal reports that HAV is hyperendemic in Top End NT rural Aboriginal communities and is acquired early in life. Icteric disease caused by HAV in Aboriginals is uncommon, consistent with asymptomatic infection in early childhood.[4] In contrast, icteric HAV disease is common in the non-Aboriginal population of the NT, especially in those in close contact with children from rural communities and those in urban day-care centres. In 1993, 115 cases of HAV were notified in the NT, an incidence of 66 per 100 000. From October 1991 to March 1994, 18 staff in NT hospitals were diagnosed as having acute HAV.

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**Positivity for HAV specific antibody by age group**

| Age (years) | No. tested | No. (%) HAV IgG positive | 95% CI |
|---|---|---|---|
| 1–5 | 20 | 18 (90 00%) | 68.29–98.77 |
| 6–10 | 88 | 85 (96.60%) | 90.37–99.29 |
| 11–15 | 98 | 98 (100%) | 96.31–100.00 |
| 16–20 | 33 | 32 (96.97%) | 84.23–99.92 |
| > 20 | 105 | 104 (99.05%) | 94.82–99.98 |
| Total | 344 | 337 (97.97%) | 95.85–99.18 |

HAV = hepatitis A virus; CI = confidence interval.

Several recent HAV serosurveys have shown that the prevalence of immunity in places previously considered to be high prevalence areas (including South-East Asia) has fallen progressively in the last 10 years.[5-12] In developed countries HAV total antibodies are present in less than 20% of those aged under 20 years who were born in those countries.[10,13,14] Non-immune individuals place themselves at substantial risk of infection when they visit endemic areas or are involved in high risk activity, such as faecal–oral contact during sex. A transitional situation of moderate transmission exists in some regions where standards of sanitation and hygiene have improved, a situation reported to be the case in some urban Aboriginal communities.[15]

Reasons for the hyperendemicity of HAV in the communities we studied include overcrowding, poor sanitation, inadequate water supply and poor understanding of personal hygiene. The high birth rate in these communities maintains a supply of susceptible individuals. The prevalence of enteric diseases such as shigellosis and salmonellosis is also alarmingly high[3] and associated with overcrowding.[16]

The newly released HAV vaccine (Havrix, SmithKline Beecham, Dandenong, Vic.) is highly immunogenic and protects against HAV,[17] but the duration of immunity is unknown and boosters may be required to maintain protective immunity. It has been argued that the vaccine should be incorporated into the routine childhood vaccination schedule with the aim of eradicating HAV,[18] a policy already adopted in the NT for hepatitis B virus (HBV). However, there are important differences between the epidemiology and natural history of HAV and HBV, especially in the NT Aboriginal population, and a different approach for HAV is indicated.

Sensible policies for targeting those at highest risk of disease are essential because the vaccine is costly and widespread vaccination could be counterproductive if protection wanes and disease transmission continues at high rates. Only 1.5% of rural Aboriginal people over 10 years of age were susceptible to HAV (our data). To our knowledge, this reflects the highest rate of seropositivity for HAV reported. Thus, vaccination of Aboriginals from the Top End would lead to little, if any, decrease in clinical disease in that population and would produce a cohort whose immunity might decrease over the following 10 years. This susceptible group within an endemic area may then, as older children or adults, manifest icteric disease with all its morbidity.

HAV vaccine should be offered in the NT to the susceptible group (non-Aboriginals less than 50 years of age) and persons at high risk (those working with children in hospitals, remote communities or transitional urban settings). At present the cost of the vaccine probably justifies prevaccination testing for antibodies in groups with some immunity, but studies to ascertain immunity levels in these groups are required.

We do not believe that it is appropriate to vaccinate NT rural Aboriginal populations given the almost universal natural immunity by 10 years of age and the persisting disadvantageous conditions of living, which dictate the high levels of transmission early in life. This policy will need to be reviewed with the changing social circumstances of Australian Aboriginal people and further seroprevalence studies will be required.

Living conditions in the communities studied are typical of many other remote communities in the Northern Territory, northern Queensland and Western Australia. Probably, similar seroprevalence rates exist elsewhere, but local vaccination policy will depend upon regional epidemiology. Meanwhile the benefits of HAV vaccination for affluent communities should not distract from efforts to address the enormous inequities in health infrastructure and education underlying our findings.

**Acknowledgements**

This study was a joint investigation by Disease Control, Northern Territory Department of Health and Community Services, and the Menzies School of Health Research in Darwin in collaboration with Theodora Efandis, Suellen Nicholson and Dr Alan Breschkin from the Victorian Infectious Diseases Reference Laboratory. The authors also gratefully acknowledge the assistance of Dr John Hargrave, Dae Sharrock, Jo Bex and Yvonne Wood.

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*(Received 11 Apr, accepted 5 Jul 1994)*