presumably, vaccination should also be offered to risk groups such as immigrants—until it is proved that they do not mount an adequate immunological response to the vaccination.

Dronning Igrids Hospital,
3900 Godthaab, Greenland T. BENNIKE

PREVALENCE OF VIRUS A EXPOSURE AND VIRAL HEPATITIS A IN INDIA

SIR,—Hepatitis A virus (HAV) infection is highly prevalent in poor countries with bad hygienic conditions. Its incidence is rapidly declining in developed countries as living conditions have improved and proper sewage systems and safe drinking water have been provided. Studies in Asia, Oceania, the Middle East, and Southern Europe suggest that hepatitis A is endemic in these regions so presumably people become infected at a young age and acquire immunity.[1,2] We have looked for HAV antibodies, as a marker of exposure to HAV infection, in 310 healthy persons of different ages and socioeconomic status and living in different environments. We also studied 485 patients with hepatitis to determine the frequency of HAV as an aetiological agent in overt disease.

TABLE I—HAV EXPOSURE IN DIFFERENT CATEGORIES OF HEALTHY PEOPLE IN INDIA

Group	Positive for anti-HAV
Cord blood	25/25 *(100%)*
Children, 1–5 yr	50/73 *(68%)*
Children, 5–10 yr	47/52 *(90%)*
Medical students and resident doctors	21/24 *(88%)*
Staff nurses	36/43 *(84%)*
Adults over 60 yr	24/25 *(96%)*
Tribal population	64/68 *(94%)*
Total	267/310 *(86%)*

TABLE II—HEPATITIS A VIRUS MARKERS IN PATIENTS WITH CLINCAL HEPATITIS

Groups	Anti-HAV positive	IgM anti-HAV positive
Acute viral hepatitis		
Adults	13/13 *(100%)*	34/180 *(19%)*
Children <10 yr	. .	18/27 *(67%)*
Subacute hepatic failure	. .	1/24
Fulminant hepatitis	15/15 *(100%)*	6/65 *(10%)*
Epidemics		
Kerala	16/16 *(100%)*	56/70 *(80%)*
Azamgarh	85/89 *(95%)*	0/39
Haryana	. .	18/82 *(22%)*
Total	129/133 *(97%)*	133/485 *(27%)*

Past exposure was investigated by detection of IgG antibodies in the serum by radioimmunoassay ('HAVAB'; Abbott Laboratories) while acute infections were studied by demonstration of IgM anti-HAV by the technique of Møller and Mathiesen.[3]

100% of cord bloods were positive for HAV antibodies, while only two-thirds of preschool children were positive. Children above the age of 5 years, adults, and the elderly had nearly the same positivity rates. Tribal populations, living in remote villages away from the unhygienic conditions of cities and having no centralised system of water supply or sewage disposal, had, surprisingly, the same high anti-HAV positivity rate as that recorded in the urban population.

IgG HAV antibodies as a marker of past exposure to virus A were positive in almost all patients with hepatitis but the IgM HAV specific antibody positivity rate had an interesting distribution. 80% of sera from an epidemic of hepatitis in a primary school were positive, while in the Azamgarh epidemic, where mainly the adult population was affected, none was positive. 22% of patients from another epidemic in Haryana were positive. 19% of adults with sporadic acute viral hepatitis were positive while the corresponding figure for paediatric patients in this group was 67%. Only 4% of patients with subacute hepatic failure and 9·5% of those with fulminant hepatitis were positive for IgM-HAV specific antibodies.

This study suggests that, after the age of 5 years, more than 90% of people in India are exposed to HAV, antibody to this virus persisting until old age. Socioeconomic factors such as environment (urban, rural, or tribal) do not influence the frequency of virus A exposure in India. Sporadic and epidemic hepatitis in children is mostly due to HAV infection while in adults HAV is a less important aetiological agent. Serious hepatitis (i.e., the subacute and fulminant forms) is seldom due to HAV.

These results raise two important questions—since 68% of children had, by the age of 5 years, been exposed to HAV why was both sporadic and epidemic hepatitis in the 5–12 age group still predominantly due to virus A; and why is the prevalence of HAV exposure in a tribal population as high as that in an urban population when the living conditions and hygiene of the two populations are completely different?

Department of Gastroenterology
and Human Nutrition,
All-India Institute of Medical Sciences,
Ansari Nagar, New Delhi-110029, India B. M. GANDI
Y. K. JOSHI
B. N. TANDON

MALIGNANT MELANOMA: TAMOXIFEN AND OESTROGEN RECEPTORS

SIR,—The long-term remission after treatment with tamoxifen in a patient with malignant melanoma, reported by Dr Mirimanoff and colleagues (June 20, p. 1368) raises again the question of the efficacy of this form of therapy. The patient was elderly, as were three of the four responders reported by Nesbit et al.[1] Nevertheless, the long delay (6 months) between the onset of treatment and appearance of partial remission must raise doubts about the role of tamoxifen in this case. Although subsequent remission appeared to coincide with tamoxifen therapy the spontaneous regression which we have observed several times in cutaneous metastases of malignant melanoma makes evaluation of such a single case extremely difficult.

Following reports of measurable cytoplasmic oestrogen receptors (ER) in some 40% of malignant melanomas,[2,3] we were surprised to find that none of 10 consecutive melanoma samples which we tested with a reliable assay contained cytoplasmic ER ($p = 0{\cdot}006$ for expected 40% incidence). A later report, by Creagan et al.,[4] of 4 ER positive cases in 34 melanomas tested is, however, in keeping with our experience. None of the 4 ER positive tumours in that study had an assay value in excess of 5 fmol/mg: in our laboratory these results would have been reported as ER negative since we use a cut-off point of 8 fmol/mg. Although an occasional malignant melanoma may contain cytoplasmic ER protein, Creagan's results support our conclusion that such cases are too uncommon for this to be of practical value.

Creagan and his colleagues[5] gave tamoxifen to 25 patients with

1. Szmuness W, Dienstag IL, Purcell RH, et al. The prevalence of antibody to hepatitis A antigen in various parts of the world: a pilot study. *Am J Epidemiol* 1977; **106:** 392–98.
2. Wong DC, Purcell RH, Rosen L. Prevalence of antibody to hepatitis B virus in selected populations of the South Pacific. *Am J Epidemiol* 1979; **110:** 227–36.
3. Møller AM, Mathiesen LR. Detection of immunoglobulin M antibodies to hepatitis A virus by enzyme-linked immunosorbent assay. *J Clin Microbiol* 1979; **10:** 628–32.

1. Nesbit RA, Woods RL, Tattersall MHN, Fox RM, Forbes JF, Mackay IR, Goodyear M. Tamoxifen in malignant melanoma. *N Engl J Med* 1979; **301:** 1241–42.
2. Fisher RI, Neifield JP, Lippman ME. Oestrogen receptors in human malignant melanoma. *Lancet* 1976; ii: 337–39.
3. Chaudhuri PK, Walker MJ, Briele HA, Beattie CW, Das Gupta TK. Evidence of estrogen receptors in benign naevi and human malignant melanoma. *JAMA* 1980; **244:** 791–93.
4. Creagan ET, Ingle JN, Woods JE, Pritchard DJ, Nai-Siang Jiang. Estrogen receptors in patients with malignant melanoma. *Cancer* 1980; **46:** 1785–56.
5. Creagan ET, Ingle JN, Green SJ, Ahmann DL, Nai-Siang Jiang. Phase II study of tamoxifen in patients with disseminated malignant melanoma. *Cancer Treat Rep* 1980; **64:** 199–201