

HEPATITIS A VIRUS AMONG CAPTIVE PANAMANIAN OWL MONKEYS

SIR,—Infectious hepatitis due to hepatitis A virus (HAV) has long been recognised as a major cause of human morbidity. However, research has, until recently, been hampered by the lack of both a simple assay to detect viral antigen and antibody and a readily available laboratory animal model. We describe here a sustained outbreak of HAV in a colony of owl monkeys (*Aotus trirangatus*) at

Gorgas Memorial Laboratory in Panama. The owl monkeys within this colony appeared uniformly susceptible to HAV and the course of infection largely mirrored that seen in man, in terms of incubation period, faecal shedding of virus, and acquisition of humoral antibody.

To determine the prevalence of anti-HAV antibody within the colony, sera were collected from fifty owl monkeys which had been held for various lengths of time. Thirty-eight (76%) of the sera contained anti-HAV antibody ('HAVAB' radioimmunoassay; Abbott Laboratories). The highest antibody prevalence rates were among animals who had been held in captivity the longest. None of 10 animals held less than 30 days, but all of thirty-five held longer than 150 days were seropositive. Of 38 animals with anti-HAV antibody, 17 (45%) had IgM anti-HAV antibody.¹ IgM anti-HAV was uniformly present in seropositive animals held for less than 300 days but was not detected in any animal held for over 600 days.

Forty monkeys, which arrived between Oct. 1 and Dec. 31, 1980, were followed up prospectively by periodic collection of serum and faecal samples. All forty lacked anti-HAV antibody when they arrived in the colony. Between 16 and 30 days after arrival, 21 blood samples were drawn from different monkeys and all lacked anti-HAV antibody. Between days 31 and 45, fourteen animals were tested and one had acquired anti-HAV antibody. However, between days 46 and 60, thirteen of sixteen animals (81%) tested had acquired anti-HAV antibody. Thirteen sera from monkeys which had seroconverted were further examined and all were found to contain IgM anti-HAV antibody.

Faecal specimens collected every 2–4 days between last negative and first positive serum samples were examined for HAV antigen by solid-phase radioimmunoassay.² Six separate cages containing a total of ten monkeys were examined. Antigen was detected in faeces collected from all six cages after an interval of 4–10 days. There was no evidence of persistent viral shedding. Cessation of faecal viral shedding coincided with or slightly preceded acquisition of humoral antibody. HAV was also detected in four (29%) of fourteen livers examined from monkeys which died in captivity and which lacked anti-HAV antibody when last tested (0–7 days before death). To our knowledge, these represent the first recoveries of HAV from non-experimentally infected hosts.

These data provide strong evidence that transmission of HAV occurred within the owl monkey colony at Gorgas Memorial Laboratory. Although the mechanism of transmission within the colony has yet to be established, a faecal-oral route is likely. We do not yet know whether the virus is human HAV or a closely related virus of *Aotus*, but either way our observations suggest that *Aotus* may be a useful host for investigation of the pathogenesis of HAV and for the production of HAV antigen, and that it may also provide an epidemiological model which might explain the occasional presence of anti-HAV antibody in other species of non-human primates.

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