HORIZONTAL TRANSMISSION OF HEPATITIS VIRUSES

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This paper will deal with the epidemiology of the hepatitis viruses other than perinatal or nosocomial infections. The current explosion of literature on the hepatitis A and B viruses often obscures the fact that a considerable corpus of knowledge concerning the epidemiology of the viruses existed before the discovery of hepatitis B surface antigen and the detection of hepatitis A virus. Nevertheless the development of techniques for the early detection of markers of hepatitis A and B virus infections has enabled epidemiological studies of these infections to be put on a more scientific basis. Many countries now require notification of viral hepatitis or infectious jaundice, and are able to produce data on the incidence of clinical hepatitis spanning at least a decade. Nevertheless it is apparent, from studies of antibody prevalence rates within populations that there is considerable under-reporting of hepatitis. This may be due to a combination of factors. In the absence of a high degree of incentive or motivation many clinical cases are not reported to the appropriate authority. There are undoubtedly a large number of subclinical or anicteric cases which escape notice. There is possibly not such an assiduous pursuit of the contacts of clinical cases as is practiced for instance in Departments of Genito-Urinary Medicine with respect to sexually transmissible diseases.

Hepatitis A

Hepatitis A virus is transmitted by the faecal-oral route. Infectious virus particles appear in the faeces late in the incubation period of the disease. Viral excretion reaches its maximum some days before the onset of clinical signs

and symptoms and is declining by the time that jaundice appears. It is important to realize that the most active disseminator of virus is to all intents and purposes a healthy individual who may or may not progress to the development of clinically apparent jaundice. There is no evidence for the existence of a chronic carrier state of hepatitis A virus. Infection is acquired by the ingestion of faecally contaminated food or drink.

McCollum (1967) proposed a five-phase model of the epidemiology of hepatitis A. In his first phase infection, with the development of lasting immunity, is nearly universal in very young children. At this age the majority of infections are anicteric. This phase appears to be operative in under-developed sub-tropical and tropical countries and is probably due to the lack of adequate sanitary facilities. In the second phase exposure in very young children is less common and the infection is seen predominantly in children in the 5-14 year age groups.

Phase three of this classification recognises increasing numbers of cases amongst adults, often occurring as waves of infection which increase until the fourth phase – that of sustained high hepatitis rates in adults – is reached. Thereafter the fifth phase of evolution is characterized by a decline of infection within the population as a whole.

Koff (1978) is of the opinion that within the United States hepatitis A has evolved from a disease of children to a predominantly adult disease consistent with progression from the second to the fifth phases of McCollum's hypothesis. Within populations it has been demonstrated that socio-economic factors also influence the prevalence of antibodies to hepatitis A. Szmuness *et al.* (1976) have shown that the prevalence of antibodies in New York was less in higher than in middle or lower socio-economic groups.

Mortimer (1980), reviewing the literature, emphasizes that there is in Europe a North to South gradient of antibody prevalence in adults form 3% positive in Sweden to 90% positive in Spain. Furthermore Frosner *et al.* (1978) have shown that in Germany the prevalence of antibody to hepatitis A virus has changed markedly. 51% of a group of 20-29 year old Germans were antibody positive in 1965 but in 1975 only 11% of this group was immune. It may be gratifying to correlate the decrease in prevalence of hepatitis A antibodies in the younger age groups with improvements in the standards of hygiene in the population as a whole but in the absence of an effective vaccine this trend carries with in certain penalties. An increasing number of the population will reach adulthood without immunity and will be at risk of developing clinical disease when challenged by the virus. Furthermore such individuals travelling to areas of high endemicity, on business or pleasure, are increasingly liable to become infected and may well introduce the virus to

their native community on their return. Skinhøj *et al.* (1981) use the term "travellers' hepatitis" to describe such infections. Finally it is probable that pooled normal immunoglobulin from these populations will contain progressively diminishing titres of antibody and will therefore be less effective as a passive immuno-prophylactic.

An outbreak of viral hepatitis that was probably waterborne was described in England by Plowright (1896). Since that time numerous accounts of waterborne hepatitis have been described although it was Neefe and Stokes (1945) who first demonstrated the presence of the infective agent in contaminated water. Contamination of municipal water supplies by raw sewage was responsible for an epidemic of hepatitis in Dehli in 1955 which gave rise to 28,000 cases. The presence of alcohol in drinks does not appear to affect the virus. Philp *et al.* (1973) describe an outbreak associated with a punch named mai tai.

Foodborne transmission requires only that the foodstuff be contaminated with sufficient virus to provide an infective dose. The nature of the foodstuff is relatively unimportant. It is known that the virus can withstand acid and although rapidly inactivated at 100 °C can survive at 60 °C for several minutes. The virus is closely related to the enteroviruses and may like them be able to withstand the processes of sewage purification practiced in many parts of the world. Among foodstuffs molluscs in particular enjoy a well deserved notoriety as vectors of hepatitis A infections. Many small outbreaks of hepatitis have been associated with consumption of these shellfish either raw or cooked. Using poliovirus to contaminate clams Digirolamo *et al.* (1970) found virus survival rates of 7-13% after steaming, frying, baking or stewing. It is unlikely that virus replication occurs in the offending molluscs; more probably the bivalves accumulate the virus particles but clear them from these systems only very slowly.

Sexual activity involving as it does intimate connection between individuals would apparently provide excellent opportunities for the transmission of viruses. It is therefore surprising that only a few viruses are known to be regularly transmitted in this manner. Conrad *et al.* (1964) suggested the possibility of heterosexual transmission of hepatitis A virus. They found that 22 of 25 military personnel who developed presumed type A hepatitis in Korea admitted to sexual exposure to the native population.

Theoretically the homosexual activities of oro-genital and ano-genital sex would favour the transmission of an agent spread by the faecal-oral route. Szmuness *et al.* (1976) investigating the prevalence of hepatitis A antibody in various sub-groups of the adult population of New York found that the prevalence of antibody in homosexual males did not differ from a heterosexual

group of comparable socio-economic class. This finding was confirmed by Coleman *et al.* (1979) in West London. In this survey the prevalence of hepatitis A antibody did not increase with age. However Høybye *et al.* (1980) observed that of 31 homosexual men admitted to a Department of Infectious Diseases in Copenhagen during 1976-1978 with acute viral hepatitis, 21 had hepatitis A, 9 had hepatitis B and 1 was neither A nor B. A prospective study of the acquisition of hepatitis A antibodies by heterosexual and homosexual men was performed by Corey and Holmes (1980). They found that the initial prevalence of hepatitis A antibody was significantly higher among homosexuals (30%) than among heterosexual men (12%). The attack rate for hepatitis A during the period of study was 11% among the susceptible homosexual men whereas no heterosexual men contracted hepatitis A infection. The oral role of oral-anal exposure correlated with both the initial presence of antibody and with the acquisition of antibody. An oral role in oral-anal activity was performed by all homosexual men who acquired hepatitis A during the study compared with 42% of those who did not. This study therefore confirms the suspicion that some aspects of homosexual activity may facilitate the transmission of hepatitis A virus. It is not known to what extent oral-anal activity is practised by homosexuals in the United Kingdom.

Hepatitis B

A large proportion of the earlier literature on the epidemiology of hepatitis B was devoted to transmission by transfusion, infection, haemodialysis and similar activities within the wide field of medical practice. It is surely presumptuous to assume that the evolution of a virus is dependent upon advances in medical technology. The epidemiology of hepatitis B contrasts with that of hepatitis A because chronic carriers of the virus occur in the population. It is estimated that there are some 200,000,000 chronic carriers of hepatitis B virus throughout the world (Dienstag, 1980). The majority of these individuals are from areas which do not enjoy those benefits of medical practice which are associated with the transmission of hepatitis B virus in developed countries. There must therefore be more natural routes of transmission of hepatitis B virus.

Hepatitis B surface antigen, a marker of hepatitis B virus, has been demonstrated in blood, saliva, breast milk, seminal fluid, and in menstrual and vaginal discharges. An early report supporting the concept of contact transmission of hepatitis B by Propert (1938) described two cases of jaundice in children two months after they were exposed to a group of children with jaundice due to administration of a contaminated measles anti-serum. Bradley

(1946) reported that the husbands of two patients who had apparently contracted syringe-transmitted jaundice developed jaundice 66 and 104 days after icteric disease in their wives. Hepatitis was also described by Freeman (1946) in four wives of men who had jaundice following yellow fever vaccine.

In the early 1960's Koff and his colleagues (Koff, 1978) observed two instances in which jaundice developed in the sexual partners of patients with post-transfusion viral hepatitis. Vahrman (1970) suggested that hepatitis might be sexually transmitted. Similarly Hersch *et al.* (1971) reported transmission of hepatitis B surface antigen positive hepatitis from a number of men to their intimate female contacts, observing at the same time that asymptomatic carriers of the antigen were capable of transmitting the disease. Heathcote and Sherlock (1973) found that 25 per cent of 51 patients in whom parenteral infection seemed unlikely had had sexual contact with jaundiced or HBsAg positive patients. Fulford *et al.* (1973) examined serum samples from 974 patients attending a department of sexually transmitted diseases and showed a higher prevalence of markers for hepatitis B virus amongst these patients. In the female patients greater promiscuity was associated with a higher probability of having antibody. Further studies amongst prostitutes (Papaevangelou *et al.*, 1974; Frösner *et al.*, 1975) confirmed a higher prevalence of antibody amongst these women and showed that the acquisition of antibody was not merely age dependent but more closely reflected the number of years experience of prostitution. Vahrman (1973) drew attention to the fact that many of the young men admitted to his wards with acute viral hepatitis were practising homosexuals. This observation has been subsequently confirmed many times (Fulford *et al.*, 1973; Jefferies, 1973; Szmuness *et al.*, 1975; Coleman *et al.*, 1977).

Approximately 5% of male homosexuals attending one clinic were found to be positive for hepatitis B surface antigen. As was found among prostitutes the likelihood of acquiring markers for the hepatitis B virus in homosexuals also appears to be age related. More significantly the male homosexual carriers have been shown to be more likely to be positive for the hepatitis B antigen and to carry surface antigen at higher titres than carriers found by screening blood donors. Few homosexuals with markers for hepatitis B virus give a history indicative of acute viral hepatitis. Due to the extreme versatility of the sexual practices indulged in by homosexuals it is very difficult to ascribe with any precision a particular practice which is more likely to transmit the virus. Lim *et al.* (1977) however suggest that the risk of transmission does appear greater in those who regularly assume the passive role during rectal intercourse. Among chronic asymptomatic carriers of hepatitis B virus individuals may be identified who have abnormal liver function tests. Liver biopsy

on these individuals reveals that a high proportion have chronic active liver disease, either chronic active hepatitis or cirrhosis (Ellis *et al.*, 1979; Viola *et al.*, 1981). There appears therefore to be a considerable body of evidence which points to sexual activity as a major route for the transmission of hepatitis B virus. This means of transmission assumes greater significance among those who are, for one reason or another, sexually promiscuous.

It is also possible that transmission of hepatitis B virus in some parts of the world may also be due to tattooing, ritual scarification, and circumcision with unsterile instruments. Indeed there have been numerous accounts of mini-outbreaks of hepatitis associated with tattooists' parlours and with ear-piercing in the Western World. Repeated biting by blood-sucking arthropods may also, in certain contexts, serve to transmit the virus. Bed bugs (*Cimex hemipterus*) collected in West Africa have been shown to contain hepatitis B surface antigen and the possible vector role of these arthropods has been discussed by Zuckerman (1977). Recently Hislop *et al.* (1981) have demonstrated an increased prevalence of serological markers of hepatitis B virus amongst histologically confirmed alcoholic liver disease, but were unable to offer an explanation for these findings.

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