

## ABSENCE OF CIRCULATING INTERFERON IN ACUTE VIRAL HEPATITIS

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Recent research has shown the importance of the interferon (IFN) system in the control of viral infections. However, while some viruses induce significant IFN levels in target organs or in the circulation, others are poor IFN inducers (1). The hepatitis viruses were thought to be among the latter (2), but recently Levin and Hahn (3) observed that patients with acute viral hepatitis (type A, B and non-A non-B), have significant levels of circulating IFN and that a defective IFN production characterizes the fulminant course of infection.

First time when we tested the sera of 20 patients with acute viral hepatitis (6 type A, 8 type B and 6 non-A non-B), we found low antiviral activity in two patients only (8 U/ml in one case of type A and in one of non-A non-B; normal controls  $\leq$  4 U/ml).

To investigate further this issue after reading Levin's and Hahn's paper (3), we have recently tested another 31 patients (6 type A, 18 type B and 7 type non-A non-B). Six of these with type B hepatitis had a fulminant course. Blood samples were collected within 7 days after beginning of symptoms and stored at  $-70^{\circ}\text{C}$ . Hepatitis A was diagnosed by determination of anti-HAV IgM, hepatitis B by serum conversion from HB<sub>e</sub>Ag to HB<sub>e</sub>Ab or by detection of anti HB<sub>e</sub>Ag IgM and non-A non-B hepatitis by exclusion of HAV, HBV, CMV and EBV antigens. Serial twofold dilutions of the sera were tested for antiviral activity in human amnion cells (WISH), using the Sindbis virus haemagglutinin yield reduction test, according to Oie *et al.* (4). Significant IFN levels ( $\geq$  4 U/ml) were not observed in any sample. As Levin and Hahn (3) found IFN using bovine fibroblasts and not human cells, we tested 15 sera for antiviral activity (5 of each group) in calf embryonic testis fibroblasts, determining the dilution at which the cytopathic effect of vesicular stomatitis virus (VSV) was inhibited by 50%. Again no antiviral activity was observed.

In accordance with previous reports (5, 6, 7) our results show that patients with type A, B and non-A non-B acute viral hepatitis do not have IFN in the circulation within 7 days from the onset of symptoms, irrespective of the course of the disease. On the other hand IFN administration to patient with chronic hepatitis seems to reduce HBV replication (8). Therefore, the capability of the virus to replicate for a long time in chronic carriers and, during the prodromic phase, in symptomatic patients would be difficult to explain, if the virus were a good IFN inducer.

## References

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