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LETTER TO THE EDITOR

A potential explanation of the reported low prevalence of hepatitis B virus infection in patients with systemic lupus erythematosus

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Zhao et al. [1] reported a low prevalence of hepatitis B virus (HBV) infection in patients with systemic lupus erythematosus (SLE). These authors were unable to explain their finding, so I should like to offer one.

There have been two large prospective studies of HBV and hepatocellular cancer (HCC) [2, 3]. Incidental findings in each were that healthy volunteer HBV carriers had higher testosterone (T) concentrations than healthy uninfected volunteer controls ($P = 0.045$ and $P = 0.0006$, respectively, both two-way). The reason for this is not established and not directly relevant to the present note (though it may relate to variation in rates of viral replication in different hormonal environments, if we may be guided by mouse models [4–6]).

It has been recognised for many years that androgen deficiency can predispose to and accelerate murine lupus [7]. There is also some evidence that androgen deficiency may be associated with the development of SLE in humans [8, 9]. Accordingly, I suggest that the finding of Zhao et al. [1] may be explained by the low androgen levels of patients with SLE.

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