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Correspondence

To the Editor-in-Chief:

We were most interested in the paper by Galun et al,[1] suggesting a possible pathogenic role of hepatitis B virus (HBV) in the development of hematopoietic and lymphoid malignancies. In 1971, one of us noted an association between cirrhosis of the liver and malignant lymphomas of lymphocytic and plasmacytoid types.[2,3] In 1977, we postulated that HBV might induce a constant stimulation of the lymphoid system resulting under certain circumstances in the emergence of a malignant clone.[4] At that time, we were unable to further substantiate this hypothesis which, admittedly, seemed farfetched to the majority of our colleagues.

In 1980, Talamo et al[5] reported an autopsy case of primary hepatic lymphoma confined to the liver in a patient with chronic active hepatitis, cirrhosis, and hepatocellular carcinoma associated with HBV infection. They speculated that HBV could have been the common cause of both tumors located in the liver.[5] In retrospect, this case might constitute the missing link between virus-associated liver diseases and hematopoietic/lymphoid malignant proliferations, but, to our knowledge, it remained an isolated case report in the literature. During the ensuing decade, the use of highly sensitive techniques for viral detection demonstrated the hepatotropism and lymphotropism of HBV, as well as of hepatitis C virus (HCV), making it easier to accept the idea that viruses that are both hepatotropic and lymphotropic might induce malignant lymphomas. Recently, Ferri et al[6] reported a significantly higher incidence of HCV in the serum of patients with non-Hodgkin's lymphoma as compared to patients with Hodgkin's disease and the healthy controls. The presence of HCV in one third of their cases of non-Hodgkin's lymphoma led them to suppose that "this lymphotropic virus, in a particular patient subset, probably in association with genetic and/or environmental factors" could play a possible role in the development of these lymphomas. We await with eager interest further confirmation of an old hypothesis, based on pathological observations, but ahead of its time to allow objective validation.

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To the Editor-in-Chief:

I read with great interest the "In Memoriam" for Dr. Donald B. Hackel, whom I knew and admired as a resident at Duke University some years ago.

I wish to add a note about Dr. Hackel's early research career, which was not mentioned in his biographical sketch. With Dr. Walter Heymann, of Heymann nephritis fame, Dr. Hackel participated actively in many of the landmark studies[1–4] that described the Heymann model, which contributed to the understanding of immunopathogenesis of renal disease.