How reproducible are rat steatosis models using high-fat diets? Reply

To the Editor:

In their Letter to the Editor, Marsman et al. [1] raised the question of the reproducibility of Lieber–Decarli (LDC) high-fat diets for the induction of fatty liver in rats [2]. The data presented in the letter revealed a striking difference in gains in body weight between the rats in Marsman’s study and those in Lieber’s study. The rats (Wistar, sex unknown, N = 5) reported by Marsman et al. had gained 98 g after 21 days of the LCD high-fat diet ad libitum, whereas in Lieber’s study, the rats (male Sprague–Dawley, N = 22) had gained 173 g. Thus daily body weight gain in Marsman’s study (4.2 g) was only half of that in Lieber’s study. (8.2 ± 1.1 g), suggesting an improper methodology in liquid diet feeding, despite, as it was claimed, “following the authors’ exact methodology”. Furthermore, no caloric intake of the high-fat diet or the control standard diet were indicated in Marsman’s letter, while Lieber et al. reported a total caloric intake of 1767 ± 42 kcal in 3 weeks and 86 ± 1.7 kcal daily and a comparable intake of the standard diet. Indeed, gains in body weight of rats (4.2 g/day) in Marsman’s feeding protocol were nearly identical to those of rats (4.7 ± 0.1 g, N = 12) fed a restricted high-fat diet (two-thirds of the amount spontaneously consumed by rats fed ad libitum) described by Lieber et al. Since dietary restricted rats developed only modest steatosis compared to panlobular steatosis in rats fed ad libitum, one may wonder whether the lack of fatty liver induction in Marsman’s feeding protocol is the result of a lower caloric intake caused by an insufficient consumption of the high-fat diet, reminiscent of the dietary restriction protocol in Lieber’s study. Furthermore, body weight gains in either ad libitum or dietary restricted feeding, and associated fatty changes were reproduced in a later study published in Journal of Hepatology [3]. It is imperative that Marsman et al. reassess the liquid diet feeding methodology to ensure that both caloric intake and gains in body weight are equivalent to those described by Lieber et al. [2,3]. Until then it is presumptuous to conclude that the Lieber–Decarli high-fat diet model is not reproducible for the pathogenesis of hepatosteatosis in rats.

References


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doi:10.1016/j.jhep.2009.05.019

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Close monitoring of serum HBV DNA levels and liver enzymes levels is most useful in the management of patients with occult HBV infection

To the Editor:

We read with interest the study by Zacharakis et al. [1] regarding the role of serial measurement of HBV DNA levels in Greek patients with chronic HBeAg(−) hepatitis B infection. We agree that serial HBV DNA level assessment may be crucial in the prediction of the natural history of HBeAg(−) patients, indeed, HBV carriers who are negative for HBeAg may replicate HBV to low levels and reactivation of HBV may occur spontaneously or as a result of immunosuppression. Similarly, HBsAg negative patients with occult HBV infections may reactivate in several contexts and such HBsAg negative patients were also shown to be associated with high risk of severe liver disease including cirrhosis or hepatocellular carcinoma. We collected data concerning occult HBV infections studying a group of 26 patients (15 men, mean age 48) suffering from cryptogenetic hepatitis. Patients remained negative at the time of diagnosis for HBsAg by three different immunoassays (AgHBs plus, Bio-rad, HBsAg Ultra, bioMerieux, HBsAg Enzy-