



Reply to contribution on the topic of hypovitaminosis D in chronic hepatitis C

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To The Editor,

We would like to thank Basyigit, *et al.* for their comments of on our recent study that aimed to determine pre-treatment 25(OH)D serum level among hepatitis C virus (HCV) infected individuals and its impact on virological response and laboratory data. Our main results were hepatitis C patients showed lower vitamin D level and it was associated to laboratory findings; however baseline 25(OH)D level was not independently associated with sustained virological response (SVR).

We agree authors highlighting the influence of several serum interleukine level, vitamin D receptor (VDR) polymorphisms and malnutrition in vitamin D status and viral response to therapy with interferon and ribavirin. Unfortunately, these data were not available in our cohort. El Hussein, *et al.*¹ found a negative correlation between vitamin D and serum level of IL-23 and IL-17 and MCP-1 in patients with hepatitis C genotype 4 suggesting vitamin D could be involved in immune response and fibrogenesis. In addition, studies conducted among Caucasian individuals from Europe demonstrated that genetic polymorphisms of vitamin D pathway were associated with the rate of SVR in patients infected by HCV genotypes 1-5.^{2,3} In spite of some studies demonstrated a role of vitamin D status on SVR rate in patients undergoing interferon-based antiviral therapy,⁴⁻⁶ a recent meta-analysis by Kitson, *et al.*⁷ demonstrated that baseline 25(OH)D level is not associated with SVR to peginterferon plus ribavirin, regardless of genotype. These controversies could be attributable to difference in genetic background, sun exposure and vitamin D ingestion. In order to reach definite conclusions about

the role of vitamin D on response to interferon and ribavirin it is mandatory to conduct high quality prospective clinical research studies.

CONFLICT OF INTEREST

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