Author's response to reviews

Title:IL-6, through p-STAT3 rather than p-STAT1, activates hepatocarcinogenesis and affects survival of hepatocellular carcinoma patients

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Author's response to reviews: see over
Dear Professors,

We would like to submit our manuscript entitled "IL-6, through p-STAT3 rather than p-STAT1, activates hepatocarcinogenesis and affects survival of hepatocellular carcinoma patients".

In this larger cohort study, we clearly elucidated that serum IL-6 rather than IL-27, TNF-α, and VEGF playing a definite role in liver function deterioration and tumor progression, as well as further affecting HCC patient survival. The mechanism of IL-6 biologic activity is chiefly through activation of p-STAT3 instead of p-STAT1 protein in the real world. From functional identification of IL-6/p-STAT3 pathways, we believe ELISA detection of circulating IL-6 and immunostain of tissue p-STAT3 as biomarker combined with current clinical biochemical data or images can provide clinicians with useful references for prognosis. Such an attractive immunotherapeutic strategy would reduce or prevent mortality in the future.

This manuscript has never been published, or previously submitted to any other journal in whole or in part, and the material will not be submitted for publication elsewhere until a decision has been made as to its acceptability for the BMC Infectious Diseases. All the authors have seen and approved the content and have contributed significantly to the work. We would appreciate your kind consideration of the manuscript for the publication in BMC Infectious Diseases.

With best wishes

Sincerely yours

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