European **Rev**iew for **Med**ical and **Pharmacol**ogical **Sci**ences 2019; 23: 3149-3150

Letter to the Editor

Comment on "LncRNA 00152 promotes the development of hepatocellular carcinoma by activating JAK2/STAT3 pathway"

Dear Editor,

We read with great interest the paper of Li et al[1] concerning the role and the mechanism of Long non-coding RNA (LncRNA) 00152 and JAK2/STAT3 pathway in the pathogenesis of hepatocellular carcinoma (HCC). A significantly higher expression of lncRNA 00152 in HCC tissues was found respect to normal liver tissues. Moreover, lncRNA 00152 expression was positively correlated with tumor stage and tumor size, whereas negatively correlated with the overall survival of HCC patients. Finally, Western blot results showed that lncRNA 00152 knockdown upregulated the protein expression levels of JAK2 and STAT3 in HCC cells.

Chronic hepatic diseases have a major impact on the quality of patients' life and increase the risk of developing life-threatening diseases as HCC[2]. Several therapies have been developed for the treatment of HCC, including surgery, radiotherapy, chemotherapy[3], antiangiogenic treatment[4] and molecular targeted therapy[5]. However, the 5-year survival of HCC is relatively low due to a high degree of malignancy and rapid progression. Therefore, it is of great significance to reveal the pathogenesis of HCC, eventually developing new therapeutic targets and improving clinical outcomes of affected patients. Several studies have indicated the regulatory effect of lncRNAs on multiple diseases, including HCC. Even if underlying mechanisms of this relationship with hepatocarcinogenesis are unknown, many authors explored their expression profile and biological role. Whang et al[6] observed that high FEZF1-AS1 expression was correlated with aggressive phenotypes and poor prognosis. Conversely, FEZF1-AS1 knockdown markedly inhibited the proliferation, migration and invasion of HCC cells and HCC tumor growth.

Likewise, Lv et al[7] results indicated that there was a marked rise in lncRNA taurine upregulated gene 1 (TUG1) expression in HCC tissues and cells. Interacting with miR-144, TUG1 contributed to proliferation and migration of HCC cells via activating the JAK2/STAT3 pathway *in vitro.*

Also lncRNA SNHG16 presented much higher expression levels in HCC tissues and cells, particularly in advanced stages of HCC. In Lin et al[8] study, enhanced SNHG16 expression was strongly related to poor prognosis. SNHG16 was confirmed to exert its carcinogenesis by miR-4500/STAT3 axis. The influence of insulin factors and insulin resistance on the development of chronic liver diseases is known[9]. The liver-enriched LncRNA LINC01093 directly binds insulin-like growth factor 2 mRNA-binding protein 1 (IGF2BP1), lead to the degradation of GLI1 mRNA, further affecting expression of GLI1 downstream molecules involved in HCC progression. LINC01093 overexpression significantly suppresses HCC cell proliferation and metastasis[10].

In conclusion, all these studies demonstrated that oncogenic lncRNA in human HCC could be used as a valuable new therapeutic target for HCC treatment.

Conflict of interest

The authors declare that they have not any personal or financial conflicts of interest.

Corresponding Author: Stefania Milione MD; e-mail: stefania.milione@gmail.com

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S. Milione[1], F. Foglia[1], M. Rinaldi[2]

[1]Department of Advanced Medical and Surgical Sciences, University of Campania Luigi Vanvitelli, Naples, Italy
[2]Multidisciplinary Department of Medical, Surgical and Dental Sciences, University of Campania Luigi Vanvitelli, Naples, Italy