The study of Zhu et al. evaluated the expression level of long noncoding RNAs small nucleolar RNA host gene 17 (SNHG17) in hepatocellular carcinoma (HCC) using reverse transcription quantitative PCR in tissue specimens and cell lines. The results showed the SNHG17 expression was significantly upregulated in cancer regions of HCC compared with adjacent regions. Moreover, increased SNHG17 expression level was correlated with tumor size, TNM stage and poor survival prognosis in HCC patients, whereas the inhibition significantly correlated with cell cycle G0/G1 phase arrest and apoptosis of HCC cells.

The possible role of SNHG17 in HCC was studied in a recent paper of Ma et al.; the results showed that SNHG17 lncRNA is up-regulated in HCC tumor tissues. This over-expression reported the proliferation, invasion and migration of HCC cells in vitro and in vivo. Furthermore, SNHG17 sponged miR-3180-3p, thereby regulating its functions and in turn up-regulating RFX1 which is a gene that has been encoded for a member of the regulatory factor X (RFX) transcription factor family. The encoded transcription factor contains an N-terminal activation domain and a C-terminal re-enrollment domain and can activate or repress target gene expression depending on the cellular context. This transcription factor regulated a wide variety of genes involved in immunity and cancer, including MHC class II genes and genes that may be involved in cancer progression. Globally, the results of this study may provide new insights into the molecular mechanisms involved in HCC and the use of the SNHG17/miR-3180-3p/RFX1 axis as a promising therapeutic target for HCC.

The interaction between lncRNA, oncogenes and cancer progression has been evaluated by other studies conducted on oral squamous carcinoma, colorectal and prostate cancer. In these cases, the influence of lncRNAs in cancer progression was confirmed by the regulation of different miRNAs. Zhao et al. evaluated SNHG17 mRNA expression level in different prostate cancer cell lines. In particular, SNHG17 increased tumor cell growth and aggressiveness by stimulating tumor cell proliferation, survival, invasion and resistance to chemotherapy and promoted in vivo tumor growth in a xenograft mouse model. Furthermore, the SNHG17 induced in vitro and in vivo were associated with activation of the β-catenin pathway.

Globally, the factors involved in the carcinogenesis and progression of HCC are not entirely clear. Viruses represent an important trigger either with a possible indirect action with the development of cirrhosis, or with epigenetic interactions. HCC remains an important health problem due to its high worldwide incidence and mortality. Furthermore, radical treatment such as liver transplantation, possible in cases where diagnosis is early, is burdened by risks of infectious, oncological or toxic complications by immunosuppressive therapy. Currently, the prevention and surveillance of viral and metabolic diseases still remain the best option for a cure; diet, lifestyle and involvement in screening of all risk groups should always be guaranteed. Unfortunately, pharmacological treatment does not allow a very wide range of effective choices. Thus, the identification of new targets such as SNHG17 may represent an important turning point.
Conflict of Interest
The Authors declare that they have no conflict of interests.

References