Dear Editor,

We consider the paper by Wang et al, published on European Review for Medical and Pharmacological Sciences and titled “Up-regulation of LINC00346 inhibits proliferation of non-small cell lung cancer cells through mediating JAK-STAT3 signaling pathway” as another important piece in the understanding of the complicated mechanisms that underlie neoplastic proliferation.

Lung cancer (LC) is one of the most common cancers in both sexes. It is also one of the deadliest forms of cancer in the world; so, it is crucial find a new therapeutic target. In particular, this article focuses attention on the expression level of LINC00346 in non-small cell lung cancer (NSCLC) tissue and its relationship with cell proliferation. Long noncoding RNA is a kind of RNA molecule with a length transcript of more than 200 nt, which has the function to regulate the gene expression at epigenetic level, but does not have the protein-encoding capacity. More and more reports have demonstrated that IncRNAs play an important role in the development of a variety of carcinomas with a crucial role in cell proliferation, differentiation, apoptosis, and in all pathological processes. However, despite the high number of IncRNAs identified, to date, little is known about their role in cancer for their vast majority and, unlikely, only a small fraction of them have been characterized. LINC00346 is one of these and have been found to be also upregulated in hepatocellular carcinoma (HCC), breast cancer, and bladder cancer tissues.

This research evaluated 70 samples of tumor tissues and para-carcinoma tissues from patients operated between January 2014 to December 2016 with a pathological diagnosis of NSCLC without any chemotherapy, radiotherapy and targeted therapy before operation. The qRT-PCR analyses conducted showed that the LINC00346 expressions were upregulated in 50 out of 70 tissues of NSCLC patients. In the experiment, second part cells transfected with sh-LINC00346 were studied for changes in cell proliferation capacity. The experiment result demonstrated that LINC00346 interferes with proliferation capacity of NSCLC cells.

The confirmation of the effect of LINC00346 on tumorigenic ability was obtained by an in vivo analysis. The results of proliferation assay demonstrated that the proliferation rate of transplanted tumor formed by cells transfected with sh-LINC00346 was significantly decreased compared with that in control group. From these results and according to literature data, it would seem that the proliferative interference mechanism LINC00346 mediated would act via JAK-STAT3 signaling pathway; in fact, Western blotting analyses highlight a reduction of expression of molecular markers of JAK-STAT3 signaling pathway such as JAK1, STAT3 and STAT5.

The importance of this research lies in having defined possible pathways of pharmacological resistance and in identifying a potential oncogene and a possible therapeutic target. Moreover, study like this could help to determine whether IncRNAs can serve as prognostic markers in NSCLC.
**Conflict of Interest**
The Authors declare that they have no conflict of interests.

**References**


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