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Letter to the Editor

In regard to Zhou et al. "MiR-24 promotes the proliferation and apoptosis of lung carcinoma via targeting MAPK7"

Dear Editor,

Zhou et al[1] have recently published an extremely interesting study about a possible role of MiR-24 in the proliferation and apoptosis of lung cancer cells. In this study, they explain how MiR-24 targets MAPK7. It is of the outmost importance to understand biological mechanisms underlying lung carcinogenesis in order to improve lung cancer treatment and to better use the available therapies. Nowadays, about 80% of cancer-related deaths in lung cancer are due to tumour progression; so if we take into account the global incidence and mortality rate of such a tumour[2], we must regard it as one of the most pressing issues of public health worldwide. As for other tumours, it is absolutely necessary to stratify patients according to molecular parameters in addition to traditional clinic criteria (such as staging or histology) with the aim of optimizing available medical[3,4] and radiation treatments[5,6]. To know in advance the possible response induced by an anticancer therapy in tumour microenvironment is a crucial element in determining its choice.

Since MiRNA are stable in blood, overexpressed in cancer compared to normal tissues and easily quantifiable, they could be used to select patients for risk-targeted therapy, to assess treatment response and to detect early relapse.

In the study by Zhou et al[1], MiR-24 expression was correlated with tumour size and clinical staging and it was inversely proportional to overall survival of patients.

Indeed, as authors underline, MiR-24 expression affects the transcription of target genes and significantly influences cancer-associated signalling pathways, which are involved in proliferation, cell cycle control, and apoptosis of lung cancer cells.

MiR-24 overexpression increases viability and proliferation of cells and inhibits their apoptosis. Consequently, an oncological treatment (chemotherapy, target therapy or radiotherapy) could be significantly influenced by MiR-24 expression.

For example, authors state that the overexpression of Mir-24 inhibits apoptosis, consequently influencing radio- and chemo-response, and eventually protecting cells against treatment-induced apoptosis. It might be interesting to know if there is a significant variation of MiR-24 expression[7] in lung cancer cells after irradiation or exposition to an anticancer drug. It is well known that intrinsic and acquired resistance to therapeutic agents significantly hinders chemotherapy and / or radiotherapy efficacy. Regarding this, some works have compared the potential for inducing drug resistance of different modalities of administering a chemotherapeutic drug. For example, it has been showed that conventional administration of chemotherapy may increase the risk of drug resistance compared to other approaches such as metronomic delivering[8,9]. Likewise, it is known that exposure of cancer cells to fractionated radiotherapy can select a cancer subpopulation with modified sensitivity to subsequent radiation[10]. Therefore, considering the possibility of using different chemotherapeutic routes and several modalities of delivering radiotherapy, dosing MiR-24 in lung cancer patients could indicate us the most suitable and effective strategy to contrast or to prevent chemo- and radio-resistance, with the purpose of increasing tumour control probability. If such evidence is confirmed by clinical trials in the future, dosing MiR-24 could work as a predictive biomarker for chemo- and radio-response and it could be a valuable tool for clinicians in management of lung cancer patients. Variations of Mir-24 expression after radiotherapy or chemotherapy

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in lung cancer could be used to guide the association of different modalities of treatment (chemotherapy, target therapy, immunotherapy) with the purpose of maximizing the possibilities of lung cancer control. Furthermore, in the future, it will be hopefully possible even to modify MiRna expression with the aim of using it as a treatment, alone or as associated to other therapies.

Conflict of interest

The authors declare no conflicts of interest.

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