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Letter to the Editor

Estramustine phosphate induces prostate cancer cell line PC3 apoptosis by down-regulating miR-31 levels

Dear Editor,

We think that this paper by Wei et al[1], published on European Review for Medical and Pharmacological Sciences and titled ''Estramustine phosphate induces prostate cancer cell line PC3 apoptosis by down-regulating miR-31 levels" is another important piece for understanding the complex and intricate scenario of tumor proliferation.

Prostate cancer (PC) is one of the common malignancies afflicting men. PC is a multifactorial disease with a complex etiology that is not clear yet. Surely genetic causes as well as environmental and life style factors contribute to its development. Age, race, familial tendency and genetic aberrations are some of the established risk factors for prostate cancer PC incidence and mortality rates changes across the different parts of the world, perhaps due to healthcare differences in cancer screening and registration[2].

The main therapeutic option for advanced prostate tumors treatment depends on androgen deprivation therapy (ADT) with limited clinical outcomes. Although the initial response rate is high, the therapeutic benefits are short-lived, and, within 18-24 months, the treated patients progress to metastatic castration-resistant prostate cancer (mCRPC), an androgen-insensitive disease, for which curative therapy is not available[3,4]. Therefore, the CRPC represents a critical demanding task for the prostate cancer cure. A greater comprehension of the molecular apparatus involved in PCa distant metastasis formation and metastatic castration-resistance is of critical importance to develop novel therapeutic approaches[5,6]. Evidence of epithelial-mesenchymal plasticity plays a role in PCa metastatic progression. Microenvironment molecules are able to accommodate cellular and tissue growth in normal or altered conditions. In tumors, local cancer environment epithelial cells must transiently turn into a mesenchymal state for which these neoplastic epithelial cells take over the evolutionary conserved EMT process. This is only an example that clarifies how much there is to discover in this field to contain and block cell proliferation[7-9].

In this paper, the authors attempt to investigate the effect of estramustine phosphate (EP) on prostate cancer cell line PC3 *in vitro*, and the related mechanism to put the basis for EP clinical application. In the experiment prostate cancer, cell line PC3 were treated with estramustine phosphate; then, they were analyzed with flow cytometry to detect their cell growth and apoptosis. Furthermore, the reverse transcriptase-polymerase chain reaction (RT-PCR) was effectuated to test that miR-31 level demonstrated its decrease. Surely these results are only preliminary and they will have to validate in the clinical scenario. These hypotheses should be confirmed by *in vivo* studies to prove scientifically that EP can suppress PC3 cell growth inducing apoptosis by a down-regulation of miR-31 level. MiR-31 level infect was found higher in prostate cancer tissue compared with para-carcinoma tissue.

Conflict of interest

The authors declare no conflicts of interest.

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