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Aurora B, Eg5, and MPS1 as Potential Biomarkers in Ovarian Carcinoma

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Abstract

Background: Ovarian carcinoma remains one of the most lethal gynecological malignancies, largely due to late diagnosis, high recurrence rates, and the development of resistance to conventional therapies [1]. Mitotic regulators, such as Aurora B, Eg5, and MPS1, play critical roles in cell division and chromosomal stability and are frequently dysregulated in cancer. Their aberrant expression may contribute to tumor progression and therapeutic resistance, highlighting their potential as clinically relevant biomarkers [2,3,4]. **Methods:** This study aimed to evaluate the expression of the mitotic proteins Aurora B, Eg5, and MPS1 by immunohistochemistry in ovarian carcinoma cell models, comparing parental OVCAR8 cells with OVCAR8 cells exhibiting dual resistance to carboplatin and paclitaxel. Caspase 3, an apoptosis marker, and Ki-67, a proliferation marker, were also included. For result interpretation, both the percentage of stained cells and staining intensity were considered. **Results:** Immunohistochemical analysis revealed distinct expression patterns of the mitotic proteins Aurora B, Eg5, and MPS1 between parental OVCAR8 cells and their carboplatin- and paclitaxel-resistant counterparts. Aurora B showed a clear increase in the percentage of positively stained cells in resistant cells compared to parental cells, indicating a more pronounced expression in the resistant phenotype. Eg5 and MPS1 were detected in both sensitive and resistant cell lines, with no marked differences in the proportion of positive cells; however, subtle variations in staining intensity were observed, with some resistant cells displaying increased intensity. In addition, Ki-67 expression was higher in resistant cells, consistent with an enhanced proliferative profile. In contrast, caspase-3 staining showed no substantial differences between parental and resistant cells under the conditions evaluated. **Conclusions:** Our findings suggest an association between enhanced expression of mitotic regulators and the acquisition of chemoresistance in ovarian carcinoma cells, particularly in the case of Aurora B. The increased Ki-67 expression further supports a more proliferative phenotype in resistant cells, while the absence of marked differences in caspase-3 indicates that apoptotic activity is not substantially altered under these conditions.

Keywords: ovarian carcinoma; mitosis; Aurora B; Eg5; MPS1

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