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FGF18 as a potential biomarker in serous and mucinous ovarian tumors

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Fibroblast growth factor 18 (FGF18) has been suggested to play important roles in promoting progression of ovarian high-grade serous carcinoma. Our aim was to investigate FGF18 expression in the whole spectrum of serous and mucinous ovarian tumors, highlighting differences in expression within the adenoma-carcinoma sequence and differences between type-1 and type-2 tumors. We also aimed to test the prognostic significance of this expression and its relation to microvessel density (MVD). We evaluated the immunohistochemical expression of FGF18 and CD31 in 103 ovarian tumors and statistically analyzed their association with clinicopathological variables and patients' outcome. FGF18 score increased significantly within the adenoma-carcinoma sequence for serous and mucinous tumors. MVD increased significantly only among serous tumors. FGF18 and MVD correlated significantly (overall and among serous tumors only) and were significantly higher in type-2 than type-1 tumors. Cox regression models were built. Independent predictors could not be determined due to multicollinearity between the predictors. However, the combination of International Federation of Gynecology and Obstetrics (FIGO) stage, ovarian carcinomatype and or FGF18 score achieved the highest predictability of poor prognosis. FGF18 could play a role within the adenoma-carcinoma sequence in type-1 tumors and might modulate angiogenesis among serous tumors. Our findings further augment the differences between type-1 and type-2 tumors. The combination of FIGO stage, ovarian carcinoma type and or FGF18 score could predict poor prognosis among ovarian carcinoma patients. Our work identifies FGF18 in ovarian neoplasia as a promising field of research, although evaluation of the performance of the developed models is still needed.

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The role of JAK/STAT signaling pathway in cell proliferation and anti-apoptosis in ESCC cells in vitro and *in vivo*

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Esophageal cancer is one of the most common malignant tumor, awarded the fourth deadliest cancer in China, which has been a serious threat to human health. Recently, accumulating evidence has demonstrated that JAK/STAT signaling transduction pathway plays an essential role in carcinogenesis, which is mainly involved in cell growth, survival, differentiation and inhibition of apoptosis, thus making it a good target for cancer chemotherapy. Although constitutive JAK/STAT activation has been reported in many human tumors, the effect of JAK/STAT signaling pathway in esophageal squamous cell carcinoma (ESCC) is still poorly understood. To explore the role of JAK/STAT signaling pathway in ESCC, the ESCC cell lines and SCID mice were used. First, We detected the status of JAK/STAT signaling pathway in several ESCC cell lines and used stattic- the inhibitor of stat3, to evaluated the role of this pathway in carcinogenesis of ESCC. In vitro studies revealed that stattic inhibited the JAK/STAT signaling pathway and suppressed the ESCC cells proliferation, promoted the ESCC cells apoptosis and increased the ESCC cells sensitivity to chemotherapeutic drugs. Under these conditions, we detected the therapeutic effect of stattic using an *in vivo* PDX model to further study the role of JAK/STAT signaling pathway in ESCC tissues. The results *in vivo* studies have shown that stattic could efficiently inhibit the expression of stat3, leading to stay proliferation and induction of apoptosis *in vivo*. This finding will provide theory bases for the opinion that JAK/STAT signaling pathway could be considered potential therapeutic target for ESCC and stattic might possess the value as protective agent in ESCC chemotherapy.

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