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Fibronectin promotes migration and invasion of ovarian cancer cells through up-regulation of FAK pathway: Cross talk with PI3K/AKT cascade

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Back ground: Ovarian cancer is the leading cause of death from gynecological malignancy and the fourth most common cause of cancer death among American women. The objective of this study is to understand the mechanism of fibronectin (Fn), to stimulate ovary cancer cell migration and invasion through up-regulation of FAK pathway.

Method and Material: The human ovarian cancer cells OVCAR-3 and A2780/CP70 were cultured and treated with fibronectin (10 μg/ml), Trans-well plates were used to conduct the migration assay, real-time RT-PCR for FAK mRNA expression, FAK siRNA for blocking FAK expression, Western blot analysis for P-FAK, P-PI3K and P-Akt. One-way ANOVA was used to determine the overall significance within data groups. P-value of <0.05 was considered significant.

Result: Fibronectin treated ovarian cancer cells OVCAR-3 and A2780/CP70 had increased ability to the migration and invasion by using trans-well plate's migration assays compared with untreated cells. Fibronectin significantly promotes cell migration and invasion through the phosphorylation of FAK. Ovarian cancer cells lines treated with fibronectin showed increased signaling regulation of FAK (p-PI3K/P-Akt) furthermore siRNA FAK treatment reduced the levels of p-PI3K/ P-Akt after induced by fibronectin.

Conclusion: Our results indicate that FAK inhibition can suppress ovarian cancer cells migration and invasion through decreased PI3K/AKT pathway may or may not prove to be a therapeutic target, or a biomarker for ovarian cancer, and future needed.

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