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INTEGRIN-LINKED KINASE: A POTENTIAL PLAYER IN OVARIAN CANCER METASTASIS

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Epithelial ovarian cancer (EOC) is the fifth leading cause of overall cancer death among American women. In 2008, 15,520 deaths were directly attributed to EOC, and an additional 21,650 cases were diagnosed. When EOC is diagnosed prior to metastatic dissemination, the overall 5 year survival rate is 93%; however, over 75% of women with EOC are diagnosed with metastasis already present, dropping the survival rate to less than 20%. The process of EOC metastasis is described as follows: epithelial cells detach from the primary tumor into the peritoneal cavity where they form multicellular aggregates (MCA), adhere intraperitoneally and undergo localized invasion into the interstitial collagen-rich submesothelial matrix, where they proliferate to anchor secondary lesions. The exact mechanism that controls the transition from detached cells to peritoneally anchored metastatic lesions is still unknown. Studies have shown that β1 integrin activation is a key event in ovarian carcinoma metastatic dissemination and regulates expression of several gene products involved in metastasis. Activation of a β1 integrin cytoplasmic domain interactor, integrin-linked kinase (ILK), has been shown to regulate several biological processes that suppress anoikis and promote invasion, two key events in ovarian cancer metastasis. This study attempts to characterize the overall expression of ILK in ovarian cancer cell lines and tissues through immunofluorescence analyses, Western blotting, and real time PCR. Preliminary results suggest that ILK activity is stimulated by collagen binding; and enhanced nuclear localization has been observed on collagen surfaces.