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FAK expression and signaling in bladder cancer cells. The activation of the nonreceptor tyrosine kinase focal adhesion kinase (FAK) and its downstream signaling pathways is implicated in the tumorigenesis of many solid tumors. We have previously shown that FAK is expressed in a range of human urothelial carcinomas. In the current study, we examined the expression of FAK and its phosphorylated form (FAK-Tyr397) in a panel of human urothelial carcinoma cell lines and examined the effect of its suppression using specific small interfering RNA. In addition, the expression of FAK, focal adhesion kinase-associated phosphoprotein (FAP) and paxillin were also assessed in a cohort of urothelial tumors. FAK expression was detected in all the cell lines, whereas the expression of FAK-Tyr397 was detected in all except one of the cell lines. The suppression of FAK by small interfering RNA reduced cell migration and reduced matrix invasion in bladder cancer cells. In addition, FAK suppression resulted in a reduction in the phosphorylation of Akt and Src. FAK expression and signaling in bladder cancer are associated with poor patient survival. In addition, the suppression of FAK in bladder cancer cells results in a reduction in cell migration and matrix invasion, as well as a reduction in

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