Carbonic anhydrase III promotes transformation and invasion capability in hepatoma cells through FAK signaling pathway.

賴基銘

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摘要

Abstract

Carbonic anhydrase III (CAIII) is distinguished from the other members of the CA family by low carbon dioxide hydratase activity, resistance to the CA inhibitor acetazolamide, and a predominant expression in the liver of males. In this report the effects of CAIII expression on liver cancer cells invasiveness were explored. Overexpression of CAIII in the HCC cell line SK-Hep1 resulted in increased anchorage-independent growth and invasiveness. And siRNA-mediated silencing of CAIII expression decreased the invasive ability of SK-Hep1 cells. Furthermore, CAIII transfectants showed elevated focal adhesion kinase (FAK) and Src activity. Silencing of FAK expression in CAIII transfectants led to suppression of HCC cell invasion. More importantly, the CAIII transfectants acidified the culture medium at an accelerated speed than the control cells did. Taken together, these data suggest that the CAIII-promoted invasive ability of HCC cells may probably be mediated through, at least in part, the FAK signaling pathway via intracellular and/or extracellular acidification.