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Regulation of stemness gene expression and tumorigenicity by an endogenous matrix metalloproteinase inhibitor RECK

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Reversion-inducing cysteine-rich protein with Kazal motifs (RECK) gene encodes a membrane-anchored glycoprotein that exhibits strong inhibitory activity against various matrix metalloproteinase (MMPs) and a disintegrin and metalloprotease 10 (ADAM10). RECK functions as a tumor suppressor by inhibiting migration, invasion, and angiogenesis. However, whether RECK can modulate the stem-like phenotypes of cancer cells is not known. In this study, we demonstrate that RECK reduces the expression of stemness genes by inhibiting Notch1 activation. In addition, RECK suppresses sphere formation and sphere size of CD133-positive cancer cells. More importantly, RECK reduces tumorigenic activity of these cells *in vivo*. Conversely, knockdown of RECK in non-tumorigenic GI2 cells increases stemness and CD133 expression and sphere forming ability. Collectively, these results indicate that RECK represses stemness gene expression and tumorigenicity by inhibiting ADAM-mediated Notch1 shedding and activation.

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