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Doxycycline inducible shRNA mediated knockdown of S100A11 impairs the migratory, invasive and proliferative capacity of adenocarcinoma cells

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Non-Small Cell Lung Cancers (NSCLCs) make 85% of lung cancers and adenocarcinomas are the most common type. Currently our understanding of the molecular signatures that drive the progression of lung adenocarcinoma is incomplete. S100A11, a member of the S100 protein family, contributes to the progression of lung adenocarcinoma; however the precise mechanism underlying its implication is unclear. We used doxycycline regulated knockdown of S100A11 to uncover its role in the cellular function of H1975 cells. Knockdown of S100A11 significantly reduced the migratory as well as invasive capability of H1975 cells. Con-focal microscopic analyses suggested a dysregulation in cytoskeletal organization and impaired lamellipodia formation. Further, CCK-8 assay showed high reduction in the proliferative capacity of S100A11 knocked down H1975 cells. Moreover, *in vivo* tumorigenic assay resulted in decreased tumor volumes in mice administered with doxycycline. Together, we report that S100A11 is an important molecule for the tumorigenic properties of H1975 cells and it may serve as a potential therapeutic target to attenuate the progression of lung adenocarcinomas.

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