Shed microvesicles derived from oncogenic H-RAS-mediated epithelial-mesenchymal transition in MDCK cells induce invasion in fibroblasts

Abstract:

Epithelial mesenchymal transition (EMT), a process whereby cells lose their epithelial phenotype and acquire mesenchymal traits, is critical in embryogenesis, organ fibrosis and cancer metastasis. Using oncogenic H RAS transformed MDCK (21D1) cells as a model of EMT, we previously showed that endosomally derived extracellular vesicles (EVs) called exosomes are profoundly reprogrammed for their protein cargo and function. However, the composition and function of another major EV subtype called shed microvesicles (sMVs) 50-1300 nm) that are derived from outward budding of plasma membrane, remains unknown. Overarching aim: To investigate the functional role of shed microvesicles released during HRAS \(^{G^{12}V}\) mediated Epithelial-mesenchymal transition.