Baicalein disturbs the morphological plasticity and motility of breast adenocarcinoma cells depending on the tumour microenvironment.

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During tumor invasion, cancer cells change their morphology and mode of migration based on communication with the surrounding environment. Numerous studies have indicated that paracrine interactions from non-neoplastic cells impact the migratory and invasive properties of cancer cells. Thus, these interactions are potential targets for anticancer therapies. In this study, we showed that the flavones member baicalein suppresses the motility of breast cancer cells that is promoted by paracrine interactions. First, we identified laminin-332 (LN-332) as a principle paracrine factor in conditioned medium from mammary epithelium-derived MCF10A cells that regulates the morphology and motility of breast adenocarcinoma MDA-MB-231 cells. Then, we carried out a morphology-based screen for small compounds, which showed that baicalein suppressed the morphological changes and migratory activity of MDA-MB-231 cells that were induced by conditioned medium from MCF10A cells and LN-332. We also found that baicalein caused narrower and incomplete lamellipodia formation in conditioned medium-treated MDA-MB-231 cells, although actin dynamics downstream of Rho family small GTPases were unaffected. These results suggest the importance of mammary epithelial cells in the cancer microenvironment promoting the migratory activity of breast adenocarcinoma cells and show a novel mechanism through which baicalein inhibits cancer cell motility.