Abstract

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Calsequestrin-2 regulates migration and invasion in breast cancer cells

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Abstract

Calsequestrin (CASQ) is a Ca2+-binding protein localized in the endoplasmic/sarcoplasmic reticulum (ER/SR), an intracellular Ca2+ release and storage of muscle. CASQ2 forms a complex with Ryanodine Receptor-2 (RyR2) luminal calcium release channel and the intrinsic membrane proteins Triadin and junction in cardiac muscle. Ca2+ is a sequester and regulator of diverse cellular processes and specific Ca2+ channels play important roles in cell proliferation and invasiveness of breast cancers. To know the role of CASQ2 in breast cancer cells, we established CASQ2 over-expressing stable cells in Hs578T cells using retrovirus. CASQ2 over-expressing Hs578T cells showed higher level of migration and invasion rate compared to Hs578T, which indicated that overexpression of CASQ2 related with cellular functions. We also found that CASQ2 overexpression elevates extracellular signal-related kinase (ERK) expression. In epidermal growth factor (EGF) treated cells, CASQ2 over-expressing Hs578T had higher phosphorylated ERK compared to Hs578T. The results from this study show a possible cause of migration and invasiveness in breast cancer cells. Taken together, these findings demonstrate that CASQ2 could be a new therapeutic target for breast cancer.

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Biography

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