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## *Ets1* and *ESE1* reciprocally regulate expression of ZEB1/ZEB2, dependently on ERK1/2 activity, in breast cancer cells

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The epithelial–mesenchymal transition (EMT) is a crucial morphological event that occurs during progression of epithelial tumors. We reported previously that levels of the  $\delta$ EF1 family proteins (ZEB1/ $\delta$ EF1 and ZEB2/SIP1), key regulators of the EMT, are positively correlated with EMT phenotypes and aggressiveness of breast cancer. Here, we show that *Ets1* induces ZEB expression and activates the ZEB1 promoter, independently of its threonine 38 phosphorylation status. In the basal-like subtype of breast cancer cells, siRNAs targeting *Ets1* repressed expression of ZEBs and partially restored their epithelial phenotypes and sensitivity to anti-tumor drugs. *ESE1*, a member of the Ets transcription factor family, was originally characterized as being expressed in an epithelial-restricted pattern, placing it within the epithelium-specific ETS subfamily. *ESE1*, highly expressed in the luminal subtype of breast cancer cells, was repressed by activation of MEK-ERK pathway, resulting in induction of ZEBs through *Ets1* upregulation. Conversely, *Ets1*, highly expressed in the basal-like subtype, was repressed by inactivation of MEK-ERK pathway, resulting in reduction of ZEBs through *ESE1* upregulation. These findings suggest that *ESE1* and *Ets1*, whose expressions are reciprocally regulated by MEK-ERK pathway, define the EMT phenotype through controlling expression of ZEBs in each subtype of breast cancer cells.

### Biography

Nguyen Duy Sinh has 20 years of experience in treatment of cancer patients, he was awarded Ph.D. from University of Yamanashi, Japan and his research interests include radiotherapy, progression of progression of epithelial tumors.

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