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BCL6 induces EMT by promoting the ZEB1-mediated transcription repression of E-cadherin in breast cancer cells

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B-cell CLL/lymphoma 6 (BCL6), a transcriptional repressor, is involved in the development and progression of breast cancers with uncertain mechanism. It was found that the expression of BCL6 positively associates with poor survival of breast cancer patients. The purpose of this study is to investigate the potential effect and mechanism of BCL6 in the regulation of epithelial-mesenchymal transition (EMT), a critical cellular process for controlling the development and progression of breast cancers. We found that BCL6 promoted invasion, migration and growth by stimulating EMT in breast cancer cells. BCL6 induced EMT by enhancing the expression of transcriptional repressor ZEB1 which bound to the E-cadherin promoter and repressing the E-cadherin transcription. Deletion of ZEB1 protected against the pro-EMT roles of BCL6 by restoring the expression of E-cadherin in these cells. Moreover, inhibition of BCL6 with BCL6 inhibitor 79-6 suppressed these functions of BCL6 in breast cancer cells. These findings indicate that BCL6 promotes EMT via enhancing the ZEB1-mediated transcriptional repression of E-cadherin in breast cancer cells. Moreover, we recently find that BCL6 is involved in the dormancy-reactivation pathway from the published clinical breast cancer database, and BCL6 initiates and maintains cancer stem cell activity. Targeting BCL6 has therapeutic potential against the development and progression of breast cancer.

Biography

Bing Cui has completed his PhD at the age of 27 years from Peking Union Medical College and postdoctoral studies from University of California, San Diego. He has published 29 papers and filed 8 patents. As first author, the investigator's papers have been published in several preeminent peer review journals such as *Cancer Research*, *PNAS*, *Blood* etc. As a principal investigator, Dr. Cui set up a new lab and research team in Institute of Materia Medica, Chinese Academy of Medical Sciences in 2015.

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