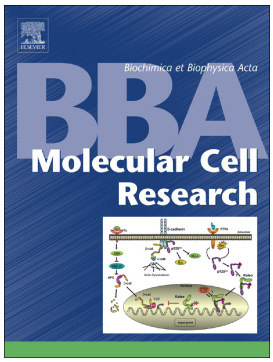

Translationally controlled tumor protein (TCTP) is required for TGF-β1 induced epithelial to mesenchymal transition and influences cytoskeletal reorganization

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Translationally controlled tumor protein (TCTP) is required for TGF-β1 induced epithelial to mesenchymal transition and influences cytoskeletal reorganization.

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Abstract

Epithelial-mesenchymal transition (EMT) is a programed course of developmental changes resulting in the acquisition of invasiveness and mobility in cells. In cancer, this course is used by epithelial cells to attain movability. Translationally controlled tumor protein (TCTP) has been extensively characterized following the observation on tumor reversion ensuing its depletion. However, the role of TCTP in cancer progression is still elusive. Here, we demonstrate for the first time that TCTP is a target of transforming growth factor-β1 (TGF-β1), a key regulator of EMT in A549 cells. We here present changes in expression patterns of intermediate filament markers (vimentin and cytokeratin 18a) of EMT following TCTP knockdown or over expression. The TCTP over-expression in cancer cells is associated with mesenchymal characters, while downregulation promotes the epithelial markers in the cells. Interaction of TCTP with β-catenin seems to stabilize β-catenin, preparative to its nuclear localization highlighting a role for β-catenin signaling in EMT. Moreover, the induction of urokinase plasminogen activator (uPA) following ectopic expression of TCTP leads to destabilization of ECM. The cells knocked down for TCTP show diminished invasiveness and migration under TGF-β1 treatment. The present results for the first time demonstrate that TGF-β1 dependent TCTP expression is required for EMT in cells.

Keyword: TCTP, cytoskeleton, EMT, cancer, TGFβ1, cell signaling.

1.0 Introduction