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Role of TBX3 on Epithelial Mesenchymal Transition (EMT) in BRAF mutant negative malignant melanoma

Shafat Hassan
Tianjin Medical University Cancer Institute and Hospital, China

TBX3 is overexpressed in a broad range of epithelial and mesenchymal-derived cancers. In melanoma, there is a link between oncogenes B-RAF, transcriptional repressor TBX3, and epithelial to mesenchymal transition (EMT). EMT is related to the upregulation of E-cadherin regulators. TBX3 is an additional regulator in BRAF mutation-positive melanoma. TBX3 may act as a significant regulator of oncogenic B-Raf signaling pathways and a promoter of metastasis in B-Raf mutation-positive melanoma. There is an essential correlation between EMT, TBX3, and EMT activation, and BRAF mutations. The mutant B-RAF can up-regulate TBX3 expression in malignant melanoma, which inhibits E-cadherin levels and promotes tumor cell invasion and metastasis. It is more rational to analyze the role of TBX3 in melanoma with low-frequency B-RAF mutation or no BRAF protein and whether it regulates epithelial-mesenchymal transition (EMT). We purposed to examine the effect of TBX3 on EMT in BRAF mutant negative melanoma. The functional loss of E-cadherin repression has been an assay mark of epithelial-to-mesenchymal- transition and several EMT transcription factors have been identified as the cause of this repression. EMT has been allied with up-regulation of the E-cadherin controllers, but it has acknowledged TBX3 as a supplementary tissue-restricted repressor of E-cadherin in melanoma. TBX3 may act as a significant controller of the oncogenic B-Raf signaling pathway and as a promoter of metastasis in B-RAF-mutant melanomas. In our research, we examined the role of TBX3 in melanoma without or low-frequency BRAF mutation and show up the first time that TBX3 still promotes EMT in BRAF mutant-negative cell lines. We have further discovered, with the support of histological and cytological experiments, the correlation between the expression of TBX3, E-cadherin, N-cadherin, vimentin, and the prognosis of the patients. Therefore, further analysis and research in the future will make it achievable to cure melanoma. The purpose of this study was to examine the role of TBX3 in melanoma without or low-frequency B-RAF mutation and the effect of TBX3 on epithelial-Mesenchymal transition (EMT) in BRAF mutant negative cells. Our results verify, TBX3 is a key molecule regulating epithelial-to-mesenchymal transition (EMT) in malignant melanoma, and TBX3 exhibits a positive regulatory role in promoting EMT regardless of the presence or absence of BRAF mutants. For the first time, we have suggested that TBX3 can still do its function of promoting tumor evolution without BRAF mutations.

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