Silencing of MTA1 in endothelial cells induced tumor regression by inhibiting angiogenesis via downregulation of S100A4

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(119/120 letters)

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Metastasis-associated proteins, such as S100A4 and MTA1, have been extensively studied for over two decades, but correlation between the two proteins remains obscure. A recent report suggesting that silencing of S100A4 in endothelial cells significantly suppresses in vitro tube formation and tumor angiogenesis in a xenograft cancer model, motivated us to examine MTA1 from the same perspective. In the present study, we showed that knockdown of MTA1 in mouse endothelial MSS31 cells using murine MTA1-specific small interference RNA (mMTA1 siRNA) markedly reduced in vitro tube formation. Interestingly, expression of S100A4 was concomitantly downregulated by mMTA1 siRNA treatment, suggesting that MTA1 is involved in S100A4 gene expression. Additionally, intratumoral administration of the mMTA1 siRNA to a human pancreatic cancer cell (PANC-1) xenograft significantly reduced the tumor volume. Histological analysis revealed a reduction in the intratumoral vessel density due to mMTA1 siRNA treatment. Altogether, the data suggested that silencing of MTA1, as well as S100A4, in endothelial cells could be used as a new strategy for tumor regression, by inhibiting tumor angiogenesis.

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