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# Sox2 induces Mesenchymal to Epithelial transition in somatic cells

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During late embryonic development mesodermal cells generate epithelial organs, such as kidney and ovary, via a process named Mesenchymal to Epithelial Transition (MET) (1). This process can also occur reversely giving rise to Epithelial to Mesenchimal transition (EMT) (2), which represents the mechanism by which metastasis originate from primary tumors. Many lines of evidence underlie a continuous interaction between these two programs. Indeed MET is believed to participate in the establishment and stabilization of distant metastases by allowing cancerous cells to regain epithelial properties and integrate into distant organs. It has been shown that fibroblasts undergo MET at some point during reprogramming to mimic undifferentiated ESCs (2).Since SOX2 is an essential factor to reprogram somatic cells to stem cell fate, we investigated if it might play a role in inducing MET in mouse embryonic fibroblasts. Using a lentiviral vector we found that Sox2 transduced cells changed morphology as early as two days after infection compared to their mock infected controls. Although cells showed an epithelial-like morphology, they were E-cadherin negative. Furthermore, Sox2 negatively regulated the expression of some of the mTOR pathway such as 4EBP1, EIF4E and S6. FACS analysis in Sox2-transduced somatic cells also revealed a cell cycle arrest in G1-phase. Together our data suggest a role of Sox2 in cell fate and commitment.

### References

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Key words

Sox2, MET, mTOR.