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NFATc2-SOX2 COUPLING SUPPORTS CANCER STEM CELLS AND MEDIATES DRUG RESISTANCE OF LUNG ADENOCARCINOMA

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Cancer stem cells (CSC) are dynamic cancer cell subsets that display enhanced tumor functions and resilience to treatment but the mechanism of CSC induction or maintenance in human lung cancer is not fully understood. Calcium signaling integrates exogenous and endogenous stress stimuli leading to cellular responses that overlap with cancer functions. We investigated the role and mechanisms of the calcium pathway transcription factor NFATc2 in lung CSC, and found NFATc2 enhanced CSC phenotypes including tumorspheres, motility, tumorigenesis, as well as *in vitro* and *in vivo* responses of lung cancer cells to chemotherapy and targeted therapy. In human lung cancers, high NFATc2 expression predicted poor tumor differentiation and adverse patient survivals. Since pluripotency factors can modulate widespread transcriptomic changes through epigenetic reprograming, we investigated their candidacy as mediators of NFATc2 on CSC regulation. We found NFATc2 transactivated SOX2 through a novel 3' enhancer locus, while inhibiting SOX2 in cancer cells that overexpressed NFATc2 led to suppressed CSC functions. Targeting NFATc2-SOX2 coupling provides a novel approach for the long term treatment of lung cancer through TIC elimination.

BIOGRAPHY

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