

miRNA-145 and Canonical TGF- β 1 Signaling Mediate Post-transcriptional Repression of CFTR and Resistance of Δ F508-CFTR to Corrector Rescue in Human Bronchial Epithelial Cells

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The predominant cystic fibrosis (CF)-associated mutation, deletion of Phe508 (Δ F508) causes defects at the level of CFTR mRNA, protein translation, and channel function. Small molecules partially rescue the distal Δ F508-associated defects in vitro. Specifically, correctors rescue the biosynthetic processing of CFTR protein while potentiators improve the function of rescued CFTR channels; however, their efficacy is marginal in vivo. Most CF patients have increased levels of TGF- β 1, compared to non-CF controls. TGF- β 1 inhibits CFTR mRNA and may represent a prevalent key antagonist limiting responses to the corrector/potentiator therapy in the majority of CF patients. Mechanisms of CFTR repression by TGF- β 1 will be discussed.

Host: Carlos Farinha
(FunGP-BioISI)

When: November 28 🕒 **14h30**

Where: Building C8, room 8.2.38

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