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Hair cells are mechanosensory receptors in the inner ear responsible for transducing oscillations in air pressure into neural signals that are then encoded as sound. Fibroblast growth factor 20 (FGF20) signaling has been implicated in the differentiation of hair cells. Mice lacking a functional copy of *Fgf20* are deaf and have decreased hair cell numbers. However, the mechanisms that regulate differentiation downstream of FGF20 and its receptor, fibroblast growth factor receptor 1 (FGFR1), are not known. There are five major pathways that serve as downstream effectors of FGFR1 in other systems: PLCγ, MAPK (p38), MEK1/2, PI3K, and STAT. But, which one is involved in differentiation in the cochlea? To answer this question, we treated murine cochlear explants with inhibitors that block each major signaling pathways individually during the timeframe when FGFR1 signaling was found to be critical for differentiation, E14.5-E16.5. We found that inhibiting signaling through PLCγ, MEK1/2, and MAPK (p38) did not result in significant changes in total hair cell number. However, treating cochleae with LY294002, a PI3K inhibitor, did result in a decrease in hair cells similar to inhibition of FGFR1 signaling and phenocopies the *in vivo Fgf20* knockout phenotype. This suggests that PI3K signaling may play a role in mediating hair cell differentiation. Furthermore, AKT is a common downstream effector of PI3K. Inhibiting AKT, however, showed no decrease in hair cells suggesting that an alternative pathway is responsible for differentiation. We are currently experimenting with alternative signaling pathways downstream of PI3K.