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Postdoc position available: BIOCHEMICAL MECHANISMS OF HEDGEHOG AND WNT SIGNALING

A postdoc position is available in our group to study signal transduction mechanisms in the Hedgehog and Wnt pathways. These pathways control the development of nearly every organ in our bodies, and, when misregulated, lead to widespread birth defects and cancers. Surprisingly, we still know very little about the biochemical steps underlying Hedgehog or Wnt signaling. We are tackling these longstanding questions by merging membrane protein biochemistry and biophysics in vitro with cell biology and embryology in living systems.

This position is ideal for a graduating Ph.D. in biochemistry, biophysics, or cell biology. It's a great opportunity for trainees looking to explore the interface between biochemistry, developmental biology, and cancer. Experience with GPCRs (or other membrane receptors), lipids, *in vitro* reconstitution, or live-cell imaging is desirable but not necessary.

Our recent studies have provided foundational insights into the activation and downstream coupling mechanisms for SMOOTHENED (SMO), a class F GPCR in the Hedgehog pathway and a major oncology drug target (Deshpande et al, Nature 2021; Arveseth et al, PLOS Biol 2021; Happ et al, biorxiv 2021). These insights have revealed new principles for how lipids can bind and activate GPCRs, and how GPCRs can control intracellular kinases. Our work is also leading to new ways to therapeutically modulate class F GPCR signaling in cancer and other diseases. There are lots of exciting follow-up studies to pursue as postdoc projects in all of these areas!

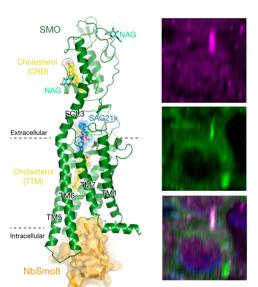
The University of Utah offers a cutting-edge, collaborative scientific environment in a beautiful and affordable metropolis with world-class outdoor activities.

Our work is funded by R35 and R03 grants from the NIH.

Candidates should send a cover letter, CV, and list of 3 references to Ben Myers at benjamin.myers@hci.utah.edu.



One of the many perks of life in Utah: year-round access to jaw-dropping natural scenery.



Left: 2.8 Å structure of a nanobodystabilized physiological active state of SMO bound to sterol ligands (Desphande et al, *Nature* 2019). Right: SMO (magenta) controls PKA signaling by directly binding and inactivating PKA catalytic subunits (green) in primary cilia (Arveseth et al, *PLOS Biol* 2021; Happ et al, *biorxiv* 2021).