# BIOCHEMISTRY SEMINAR

## Tec Kinases in Immune-Cell Signaling


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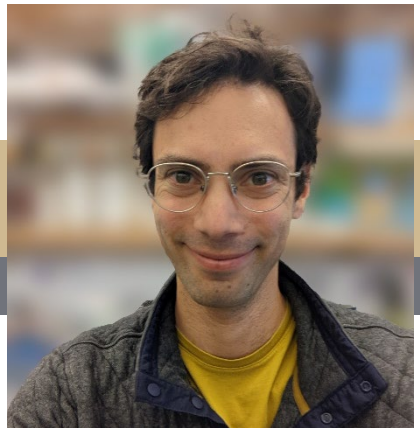


**Abstract:** One of the remarkable success stories in cancer treatment is the development of the small-molecule kinase inhibitor ibrutinib, which targets the Tec-family kinase BTK. In B cells, BTK is activated at the plasma membrane, where it phosphorylates phospholipase C γ (PLCγ). This phosphorylation event marks a transition from receptor-proximal to cell-wide signaling. BTK is the gatekeeper of this transition. Despite its major contributions to B-cell development, signaling, and cancer, how the structure of BTK regulates its cellular functions remains unknown. I developed a suite of high-throughput and single-molecule methods to investigate the function of BTK in B cells and the related T-cell kinase ITK in T cells. In three vignettes, I will describe how (1) BTK activation depends on dimerization in some cellular contexts but not others; (2) the SH2 domain of BTK evolved to support autoinhibition; and (3) the PH–TH lipid binding module of ITK allows ITK to prime T-cell activation prior to T-cell receptor engagement. Collectively, my studies shape our understanding of the evolution and mechanism of these fascinating kinases.


Monday, March 24, 2025 | 3:30 pm | BRWN 4102

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