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Research interests:

A number of clinically important pro-inflammatory and pro-angiogenic regulators lack a signal peptide in their primary structure and thus are secreted through poorly understood nonclassical mechanisms, which are independent of endoplasmic reticulum and Golgi. Among them are FGF1, FGF2, IL1 α , IL1 β , MIF and other. Understanding the mechanisms of nonclassical secretion has critical implications for cardiovascular biology, oncology and treatment of proinflammatory disorders. Our laboratory uses the methods of cell and molecular biology and studies on animal models to understand the mechanisms of nonclassical protein export and its regulation by stress, Notch signaling and thrombin receptors.

The following problems are addressed in the ongoing projects:

1. How non-classical release complexes assemble and translocate through the cell membrane? Here we are dealing with the enigma of the secretion, which does not use exocytotic vesicles.
2. What are the signaling pathways underlying the inhibition of non-classical secretion by Notch signaling? Our hypothesis is that in stationary tissues Notch signaling inhibits the non-classical release of polypeptide regulators and thus prevents hyperplasia and inflammation.
3. What are the effects of the stimulation of non-classical release upon tissue repair, angiogenesis, tumor formation and inflammation? These studies are pursued on in vivo models.