Summary of Research Area

The general theme of my research focuses on the investigation of the cellular and molecular mechanisms underlying tumor development with the goal of identifying new therapeutic targets for treatment. This is articulated around three main points.

- Understanding the mechanism by which Wnt/β-catenin signaling pathway promotes tumorigenesis.

Previous studies have shown that constitutive activation of the Wnt/β- catenin signaling pathway is central for the development of colorectal cancer. Understanding this process would lead to design of better agents to treat this condition or others due to constitutive activation of Wnt/β-catenin signaling pathway.

We identified the coding region determinant-binding protein (CRD-BP) as a *bona fide* transcriptional target of Wnt/β-catenin signaling pathway, and demonstrated that its induction is responsible for a variety of pleiotropic effects of Wnt/β-catenin signaling in human colorectal cancer cells. CRD-BP has also been associated with the most aggressive form of many cancers. Advanced colorectal cancers are notoriously resistant to drugs.

My current studies are focused on the regulation of expression and function of CRD-BP; its role in tumor development and resistance to chemotherapeutics.

- Study the cross-talk between Wnt and Hedgehog signaling pathways in cancer development.

The Glioma-Associated Oncogene Homolog 1 (GLI1) is the transcriptional activator of the Hedgehog signaling pathway. Our previous studies showed that GLI1 is also a target of CRD-BP. Wnt/β-catenin signaling activation induces elevation of CRD-BP, which, in turn binds and stabilizes GLI1 mRNA, causing an elevation of GLI1 expression and transcriptional activity. Activation of GLI1 is a key step in the initiation of the tumorigenic program leading to Basal cell carcinoma (BCC).

My current research is focused on the role of CRD-BP in BCC development.

- Delineate the cellular and molecular mechanisms governing breast cancer cells fusion and its impact in metastasis.

Although cancer cell fusion has been suggested as a mechanism of cancer metastasis, the underlying mechanisms defining this process are poorly understood.

Our recent findings suggest a mechanism by which hypoxia-induced apoptosis stimulates fusion between mesenchymal stem/stroma cells and breast tumor cells resulting in hybrids with enhanced migratory capacity that may enable their dissemination to distant sites or metastases.

The focus of this study is to investigate the cellular and molecular mechanisms of apoptosis-driven cancer cell fusion. This study might provide new strategies to developing alternate drugs for preventing metastatic spread.