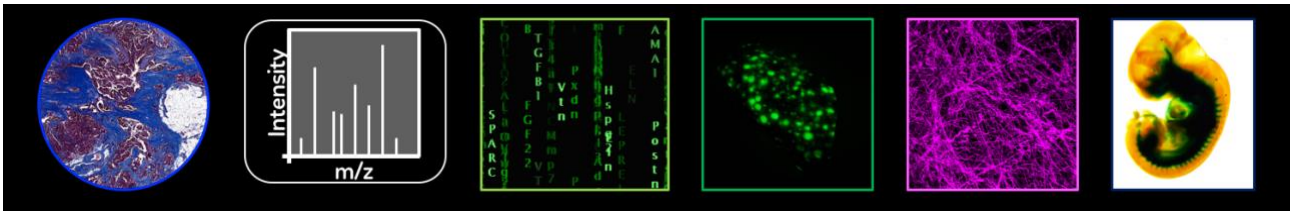

The Naba lab studies the role of the extracellular matrix (ECM) in development, health, and disease, with a particular focus on cancer. To do so, we utilize classical molecular, cellular, and developmental biology approaches in combination with cutting-edge proteomics and computational analyses. Our goal is to better understand how the ECM contributes to diseases, in particular fibrosis and cancer, so that we can exploit it to develop novel diagnostic and therapeutic strategies.
The Naba lab is a highly collaborative and dynamic group that strives for scientific excellence, and offers a stimulating and inclusive working environment conducive to learning and professional development.

Project 1: Elucidate the roles of SNED1, a novel extracellular matrix protein, in breast cancer metastasis

We previously identified a novel ECM protein, SNED1 (Sushi, Nidogen and EGF-like domain protein 1), in a proteomic screen aimed at discovering ECM proteins differentially expressed between highly and poorly metastatic mammary tumors. We reported that SNED1 was produced only by highly metastatic tumor cells, and not by poorly metastatic tumor cells or by stromal cells, and that it played a functional role in tumor dissemination, as, SNED1 knockdown decreased metastasis in a murine model of breast cancer. We further showed that the level of expression of SNED1 was a prognostic factor for hormone-negative breast cancer patients (Naba et al., eLife, 2014).
We are now working towards identifying the cellular and molecular mechanisms controlled by SNED1 and contributing to breast cancer metastasis.

Project 2: Understanding the role of SNED1 in craniofacial and skeletal development

The importance of SNED1 in breast cancer metastasis prompted us to evaluate its roles in normal development and physiology. To do so, we generated multiple mouse model lacking *Sned1* either globally or in a tissue-specific manner and found that *Sned1* is an essential gene, since global *Sned1* knockout results in early neonatal lethality. We also reported the first physiological functions of this gene as a regulator of craniofacial morphogenesis and skeletal growth (Barque et al., bioRxiv, 2020). Our current work focuses on determining the precise aspects of craniofacial and skeletal development controlled by *Sned1* and the potential contribution of *SNED1* in a rare human syndrome: the 2q37 deletion syndrome.

Through collaborative efforts, and as part of The Matrisome Project, initiated by Dr. Naba, our lab also continues to develop novel proteomic approaches and bioinformatic tools to further characterize the ECM of model systems and investigates its roles in diseases including cancer and fibrosis. Incoming trainees will all have the opportunity to work on these collaborations in addition to their own research projects.