QUANTITATIVE & SYSTEMS BIOLOGY COLLOQUIUM:

Decoding Immunopathogenic Cellular Crosstalk in Chronic Pancreatitis: Insights from Patient-Derived Samples

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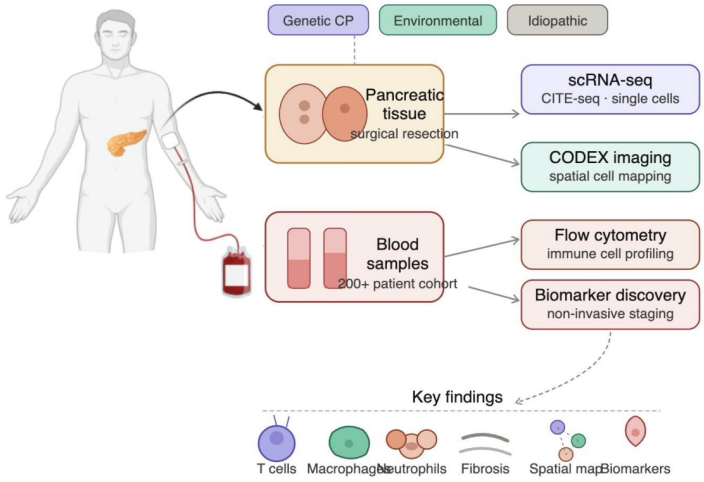
About The Speaker:

Bomi Lee, PhD is an Assistant Professor of Pediatrics at Stanford University, where she leads an independent research program in the Division of Gastroenterology, Hepatology & Nutrition. Driven by a deep commitment to improving outcomes for patients with debilitating pancreatic diseases, she investigates how the immune system contributes to the development and progression of chronic pancreatitis and pancreatic cancer. Her work draws on cutting-edge technologies — from single-cell sequencing to spatial tissue imaging — to uncover why the same disease can look so different from patient to patient, and to translate those discoveries into better diagnostics and targeted treatments. She is also affiliated with Stanford Bio-X, Stanford MCHRI (Maternal & Child Health Research Institute), and Stanford Diabetes Research Center, reflecting her passion for interdisciplinary science at the intersection of immunology, genetics, and clinical medicine.



Human samples as the cornerstone of CP research
Pancreatic tissue · blood cohorts · multi-platform immune profiling

Date:
3/19/2026

Time:
10:30 AM – 11:45 AM

Location:
SSB 130

Abstract:

Chronic pancreatitis (CP) is a long-term inflammatory disease of the pancreas that causes irreversible damage, chronic pain, and significant loss of quality of life. Despite being a serious condition, there are currently no approved treatments that stop or slow the disease, and doctors lack reliable tools to detect it early or predict how it will progress. One reason for this gap is that the pancreas is difficult to access, making it hard to study what is actually happening inside the organ at the immune level. Our research program set out to understand how the body's immune system behaves in CP — and whether different causes of the disease lead to different immune responses.

Methods: We collected pancreatic tissue samples from CP patients who underwent surgery, as well as from healthy organ donors for comparison. Patients were grouped by the likely cause of their CP: genetic (inherited mutations), environmental (alcohol and smoking), or unknown (idiopathic). We used a range of modern techniques to examine immune cells in the tissue and in blood samples, ranging from methods that identify and count different immune cell types, to advanced technologies that can analyze thousands of individual cells at once and map out how cells interact with each other in tissue.

Results: Our studies consistently showed that CP is not a one-size-fits-all disease — the immune system behaves very differently depending on the cause. In patients with genetic CP, a particular type of immune cell (T cells) dominated the pancreas and showed signs of responding to specific disease-related targets. In contrast, patients with idiopathic CP had a greater presence of macrophages, a different type of immune cell more associated with general inflammation. Using blood samples from a large national cohort of over 200 patients, we also identified immune signals in the bloodstream that could distinguish CP from earlier stages of the disease — pointing toward potential non-invasive biomarkers. Finally, using a cutting-edge spatial imaging approach, we mapped the physical arrangement of immune and structural cells in the pancreas and discovered previously unknown cell types and interaction patterns that differ across CP subtypes.

Conclusion: Together, these studies reveal that chronic pancreatitis has distinct immune "fingerprints" depending on its cause. This means that a single treatment approach is unlikely to work for all patients. By identifying the specific immune pathways active in each subtype, our work opens the door to developing more targeted therapies and better diagnostic tools — moving toward a more personalized approach to treating this understudied and debilitating disease.

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