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DEPARTMENT OF BIOMEDICAL ENGINEERING

2025 Fall Seminar Series

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11am-12pm
AUST 445

ABSTRACT:

Evolution of collagen homeostasis in stromal fibroblasts underlie acquired resistance to cancer malignancy among placental mammals

Among mammals, placental invasion is correlated with vulnerability to malignancy. Animals with invasive placentation (e.g. humans and rodents) are more vulnerable to malignancy of carcinomas, while those will no placental invasion (e.g. bovine) also can contain cancer metastasis locally. We have advanced an evolutionary framework to explain this correlation, termed Evolved Levels of Invasibility (ELI) which posits that it is the stromal evolution into a highly resistive state which has limited placental invasion, and secondarily, carcinoma dissemination. However, this framework is only correlative, and experimentally validated in vitro. Evolutionary hypotheses are extremely challenging to demonstrate and confirm in the natural ecosystem. However, we have developed mouse models of "stromal avatar", which can allow us to test the ELI framework, as well as mechanistically demonstrate the causal genetic correlates underlying the vastly differential rates of invasion across mammals. In vivo, bioluminescent A375 cancer cells co-mixed with hSkFbs or bSkFbs were implanted subcutaneously into SCID-beige mice and the progressive tumor development were observed and quantified using BLI. We found the hSkFbs group mice tumor size was smaller than bSkFbs group mice, but developed distal metastatic locii, as well as exhibited lower survivability. We further explored the genetic underpinnings underlying these differences by differential transcriptomics of hSkFbs, bSkFbs with or without the skin cancer cells, finding vast differences in gene expression encoding collagen subtypes, as well as matrix metalloproteinases. These included Collagen III and MMP3 being higher in cows, while Collagen VII and MMP14 being higher in humans. Using gene perturbation combined with a quantitative nanotextured stromal invasion assay, as well as in vivo dissemination assays, we demonstrate the causality of these genes as critical regulators of stromal resistance in bSkFbs, while promoting vulnerability to invasion in hSkFbs. Our data support the idea that the evolution of stromal derived collagen rich ECM niche regulate the cancer metastases, which could provide important insights to develop rational antimetastatic therapeutics.

BIOGRAPHY

Dr. Yamin Liu is currently pursuing her 2nd Ph.D. at the Department of Biomedical Engineering, at the University of Connecticut (UConn), mentored by Dr. Kshitiz. Her research focusing on how the tissue microenvironment impact on disease susceptibility and progression, with particular emphasis on intercellular communication, cell-extracellular matrix interaction.

Dr. Liu holds her 1st Ph.D. in Clinical Pharmacy from China Pharmaceutical University and has received extensive interdisciplinary training across biomedical engineering, molecular and cellular biology, pharmacology, and medical sciences. She completed postdoctoral research in cardiac molecular biology at both Johns Hopkins University and the University of California, San Francisco (UCSF). She has published over 40 research articles in broad scientific outlets, including high-impact journals (i.e., Cell Reports, PNAS, JCI Insight, Nature Communications, and Cell Systems). Besides, She also a recipient of Predoctoral Fellowship from the American Heart Association (AHA).

For questions, please contact
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