Engineering Functional Adipose Tissue

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https://asu.zoom.us/j/98112983378

Bio Sketch

Dr. Evangelia Bellas is an Assistant Professor in the Department of Bioengineering at Temple University. Prior to joining Temple University, Dr. Bellas was a postdoctoral fellow in Biomedical Engineering at Boston University and Bioengineering at University of Pennsylvania under the mentorship of Dr. Christopher Chen where she developed 3D in vitro adipose tissue disease models. She received her Ph.D. in Biomedical Engineering at Tufts University mentored by Dr. David Kaplan. Her Ph.D. research focused on developing long-term volume stable silk biomaterials for soft tissue regeneration. This work resulted in 2 patents and a start-up. Before starting her Ph.D., Dr. Bellas was at Massachusetts Institute of Technology under the supervision of Drs. Robert Langer and Daniel Kohane, where she worked on biomaterial, drug delivery solutions for prevention of peritoneal adhesions and controlled release formulations for long-term pain management. Her current research focuses on the development of fat-on-chip and (dys)functional adipose tissue models to study how vascularization and interactions with the microenvironment impact tissue health and function.

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Abstract

Obesity is a growing epidemic which affects 42% of U.S. adults and leads to co-morbidities, such as type 2 diabetes, cardiovascular disease, stroke, some cancers, among many others. Obesity is defined as excess adipose tissue, but how it works and how it exerts its effects on other tissues is poorly understood. Adipose tissue was once seen as a static, storage unit for energy, more recently, we have begun to appreciate this dynamic, complex tissue which regulates our metabolic homeostasis. Adipocytes, the primary cell type in adipose tissue, expand and shrink to accommodate energy (lipids) storage and release. This requires a dynamic matrix, which can be easily remodeled. In obese adipose tissue, adipocytes become hypertrophic as they store more lipids, and the vasculature does not increase to adapt to the growing tissue. This results in tissue hypoxia, leading to inflammation and fibrosis, and ultimately causing a vicious cycle of dysfunction. Our group develops adipose tissue disease models to mimic these dysfunctional states of hypoxia and fibrosis. We employ various bioengineering approaches to build these 3-dimensional engineered adipose tissue models, to study how fibrosis occurs, which pathways are implicated and how it leads to further dysfunction when the cell is physically constrained with pericellular collagen. We have also developed a vascularized adipose tissue model, demonstrating how vascularization is supported by healthy adipocytes and how direct contact between these cells regulates their function.