# BIOENGINEERING
# Tenure-Track Faculty
# Candidate Seminar

**Date:** Tuesday, February 11
**Time:** 12:00 pm - 1:00pm
**Location:** Exploratory Hall, Room L111
(*Videoconferencing to SciTech, K. Johnson Hall Rm 254)*

## Sasha Cai Lesher-Perez, Ph.D.

**Biography:** Sasha Cai obtained his B.S. degree in Biomedical Engineering from the University of Wisconsin, Madison, and a Ph.D. in Biomedical Engineering from the University of Michigan, Ann Arbor. At the University of Michigan, he worked in the area of microfluidic circuits, as well as three-dimensional cultures and biosensors, under the supervision of Professor Shuichi Takayama as a GEM, Cellular Biotechnology, and NSF GRFP Fellow.

After his PhD, Sasha Cai worked as an F31 Recipient and Ford Fellow at the University of California, Los Angeles, in Professor Tatiana Segura's lab, researching the interface between microfluidic systems and hydrogel microparticles. His work aimed to integrate synthetic hydrogels into microfluidic devices to enable a different modular control into on-chip 3D cell culture platforms.

In his most recent position, as a Marie Curie Individual Fellow working at Elvesys SAS, a microfluidic instruments and controls startup in Paris, France, Sasha Cai oversaw 3 Industrial PhD students and worked on the development of cytotoxicity assays, and microfluidic systems for organ-on-chip platforms using new proprietary thermoplastics as an elastomeric alternative for PDMS that would enable industrial scale-up.

His future research program will integrate concepts of fluidic controls, biomaterials, tissue engineering, to establish three-dimensional perfusable in vitro culture platforms. These platforms will be used to apply timed oscillations in three-dimensional culture platforms to elucidate the role of hormonal dysregulation in metabolic and neurological disorders and diseases.

**Title:** Micro-construction -- Building homes of the future for the modern cell

**Abstract:** Efforts to close the gap between in vitro to in vivo model systems have produced technologies that more effectively evaluate spatial, structural, and mechanical control mechanisms (e.g., the move from two-dimensional to three-dimensional cultures). While biology works in rhythms, the methods for in vitro models are almost completely void of this temporal component. Timed oscillations applied in vitro primarily rely on two-dimensional cultures due to the difficulty of perfusing three-dimensional tissue cultures. However, this approach disregards cellular and tissue function in the more native, three-dimensional state.

One prime example are hormones; glucocorticoids fluctuate as a function of both the circadian rhythm and stress. Yet the majority of published work regarding glucocorticoids applies bolus treatments, which does not capture the basal and stressed oscillations expected in vivo. Of specific interest to me is the role of stress on hormonal dysregulation and disease progression in association with chronic and heightened stress.

In this talk, I will cover my previous work on two technology platforms. First, I will discuss the development of microfluidic self-regulating circuits as a tool to produce modular chemical profiles on-chip at different timescales. Microfluidic self-regulating circuits are small-footprint systems with embedded fluidic operations that enable multiple biological experiments to be conducted in parallel in a user-friendly fashion. Second, I will describe microparticle building blocks for the generation of customizable porous scaffolds. Fabricating microparticles enables **a bottom-up approach to engineer the chemical, mechanical, and geometric properties of these tissue-culture scaffold building blocks. Finally, I will discuss my future research program in which these two technologies will merge to implement hormonal rhythms in easily perfusable three-dimensional culture systems. This work will parametrize the role of stress-associated glucocorticoid dysregulation on disease development and progression, with an initial goal of determining the plasticity of pancreatic secretory (beta, alpha, gamma) cells and the transition from a stress-associated compensatory state to a pre-diabetic state.**