**Weekly Colloquium**
Tuesday, 3/21/2017, 12:30pm, Billings Building – Rosedale Conference Room

**“Bioelectronic Medicine: The Future of Medicine”**

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**Research Abstract:**
I began my research career in the laboratory of Kevin J. Tracey, MD, studying the mechanisms of pathological inflammation. My early work focused on validating HMGB1 as a therapeutic target, and gaining a broader understanding of its molecular mechanisms of disease pathogenesis. I have also studied the “inflammatory reflex,” a neural mechanism through which the central nervous system detects and regulates immune responses to infection and injury. More recently, through the pursuit of my doctoral dissertation I discovered the “neural tourniquet,” a field of technology that uses electrical nerve stimulation to control traumatic hemorrhage. This technology has been validated in murine, rat and porcine models of peripheral and internal (non-compressible) trauma models. My current research focuses on the molecular mechanisms of the neural tourniquet, and the development of new devices to activate this novel mechanism to control bleeding. This technology has been licensed by Sanguistat, Inc., a startup biotechnology company focused on the clinical development and eventual commercialization of a novel, transcutaneous electrical nerve stimulator for potential use in surgery, trauma, and bleeding disorders. As chief scientific officer of Sanguistat, Inc., I am directly responsible for the scientific development of our technology and its advancement to the clinic. I received my B.S. in biotechnology from William Paterson University in 1996 and a doctoral degree in molecular microbiology from Stony Brook University in 2009. I am author or co-author of approximately 80 peer-reviewed publications, book chapters and meeting abstracts, and co-inventor on a patent that describes vagus nerve stimulation to control hemorrhage.

**Recent Publications:**
Vagus nerve stimulation regulates hemostasis in swine.

Transcutaneous vagus nerve stimulation reduces serum high mobility group box 1 levels and improves survival in murine sepsis.

Splenectomy inactivates the cholinergic antiinflammatory pathway during lethal endotoxemia and polymicrobial sepsis.