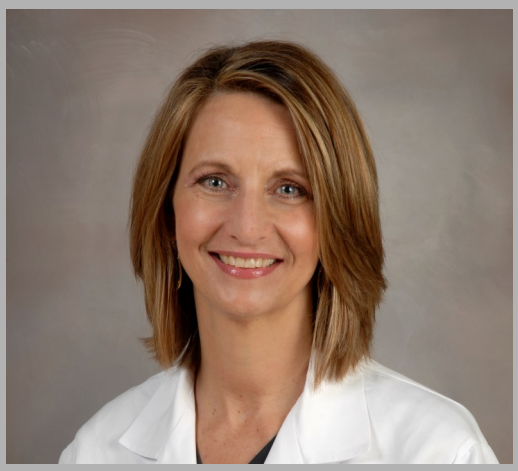
BIOENGINEERING
Spring 2021 Seminar

Date: Thursday, April 1
Time: 12:00 pm - 1:00pm
Location: Virtual
Join Zoom Meeting
https://gmu.zoom.us/j/98805494005?pwd=M1A2R1BaSEdqa2hhOUltTE5YeWxtdz09

Meeting ID: 988 0549 4005 Passcode: 454698



Rosemary Kozar, Ph.D.

Biography: I am currently Professor of Surgery, Director of Translational Research at the Shock Trauma, and Co-Director of the Shock Trauma Anesthesia Research (STAR) Center at the University of Maryland. As a trauma surgeon and surgical intensivist in a busy trauma and surgical center, I have gained extensive knowledge of the care of critically injured patient in hemorrhagic shock. My expertise is evidenced by publications, invited presentations, and national positions. I've held national leadership positions including the Executive Committee of the American College of Surgeons Committee on Trauma, AAST Board of Managers, President of the Shock Society and Vice-President of the Western Trauma Association. I am also on four editorial boards and have served on the SAT study section as well as on numerous NIH Special Emphasis Panels related to this topic.

My expertise lies in mechanisms of endothelial dysfunction after shock. I was the first to demonstrate that syndecan-1, the backbone of the endothelial glycocalyx was shed in severely injured patients in hemorrhagic shock. More recently, we have been examining intra-cellular mechanisms and discovered a novel microRNA that mediates endothelial injury by inhibiting syndecan-1.

Title: **Endotheliopathy, from bench to bedside**

Abstract: Trauma is the third leading cause of death across all age groups. Of these deaths, hemorrhage remains the number one cause of early trauma deaths, Current efforts to improve outcomes are focused on reversal of the dysfunctional endothelium that follows trauma and hemorrhage, coined the "endotheliopathy of trauma" (EoT). Syndecan-1 forms the structural backbone of a protective network on the endothelium referred to as the glycocalyx. We have shown that injury to the glycocalyx leads to shedding of the syndecan-1 ectodomain. Shedding is associated with enhanced shock, inflammation, and endothelial damage and is an independent predictor of mortality. In our efforts to define the mechanisms for syndecan-1 downregulation, we discovered that microRNA-19b (miR-19b) targets syndecan-1, phenocopies the effects of hemorrhage in reducing syndecan-1 expression, and contributes to the EoT, Our data also demonstrates that miR-19b may be a therapeutic target, as antagomiRs reverse the deleterious effects of syndecan-1 downregulation as do a number of resuscitative blood products. Our most recent investigations are examining the role of pathologic hyperadhesive VWF in the etiology of EOT and hemorrhage-induced coagulopathy.