Abstract:

Vascular devices such as vascular grafts, hemodialyzers and oxygenators suffer from high failure rates due to both thrombosis and neointimal growth. These devices can activate blood and often require the use of systemic antithrombotics to reduce the incidence or severity of device failure and thrombotic events. Currently marketed antithrombotic drugs can help reduce clot formation on vascular devices, but all increase the risk of bleeding. Dr. Hinds research focuses on blood material interactions to reduce both thrombosis and neointimal growth on these devices. Her laboratory has developed surface modification methods to encourage in vivo healing responses. She is part of a larger team focused on studying the role of coagulation factors (F)XII and XI of the contact activation pathway, which play important roles in not only the initiation but also the propagation of the thrombotic process. We focus on FXII and FXI because (1) there appears to be a causal relationship between contact activation and vascular device failure, and (2) targeting the contact activation pathway as a therapeutic approach is less likely to have detrimental bleeding side effects for patients. Dr. Hinds utilizes clinically relevant non-human primate models to study these blood material interactions and the in vivo long term healing responses. Her approaches are being applied to improve the design and development of vascular grafts, stents, hemodialyzers and extracorporeal membrane oxygenators (ECMO).