Owen McCarty, PhD, FAHA

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Bio:

A native of Rochester, Dr. McCarty received his B.S. in Chemical Engineering from SUNY Buffalo, and a Ph.D. degree in Chemical Engineering from Johns Hopkins University, where his research focused on the identification and characterization of tumor cell receptors for blood platelets and leukocytes. He performed his postdoctoral research on platelet cell biology in the Pharmacology Department at the University of Oxford and University of Birmingham, UK in the group of Dr. Steve Watson. Dr. McCarty joined Oregon Health & Science University in 2005, where he holds an appointment as a Professor in the Departments of Biomedical Engineering and Cell, Developmental & Cancer Biology and the Division of Hematology & Medical Oncology in the OHSU School of Medicine. Dr. McCarty serves as the Chair of the Biomedical Engineering Department and a fellow of the American Heart Association.

Research focus:

Hemostatic plug formation upon blood vessel breach is initiated by platelet recruitment, activation and aggregation in concert with thrombin generation and fibrin formation. However, a similar process can also lead to pathological processes including deep vein thrombosis, ischemic stroke, or myocardial infarction, among others. We have developed narrow mechanism-specific agents targeting the intrinsic pathway of coagulation and demonstrated that experimental thrombosis and platelet production in primates is interrupted by selective inhibition of activation of coagulation factor (F)XI by FXIIa. Our current studies are focused on defining the role of the endothelium in inactivating FXI, as well as studies on whether inhibiting FXI is beneficial in a non-human primate model of sepsis. We have recently published the first data from our Phase 1 clinical trial on the safety of inhibition of FXI, and are testing the efficacy of FXI inhibition in dialysis in a Phase 2 clinical trial. The understanding of the mechanisms by which the intrinsic pathway of coagulation promotes thrombus formation may support the rationale for the development of selective, safe and effective antithrombotic strategies targeting FXI.