CHEMICAL & BIOMEDICAL ENGINEERING SEMINAR ANNOUNCEMENT

von Willebrand factor: Friend or Foe?

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Room B135

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Jing-fei Dong, MD, PhD, is a full member of the Bloodwork Research Institute and a Professor of Medicine in the Division of Hematology and Oncology, Department of Medicine, the University of Washington School of Medicine. He received his medical training from Lanzhou Medical College and Tianjin Medical University in China (clinical certification in 1984) and his graduate training in Neurobiology from University of Birmingham in the United Kingdom (PhD in 1993). He completed his clinical fellowship in Neurosurgery in the Midline Center for Neurosurgery and Neurology, University of Birmingham School of Medicine (a Fellow of the Royle College of Surgeons in 1993). He received his basic science postdoctoral research training in hemostasis and thrombosis at the Gladstone Institute of Cardiovascular Sciences, University of California San Francisco. He was first appointed as an Assistant Professor in 1996 and moved up the rank to a professor with tenure and endowed professorships in Baylor College of Medicine. He served as the Chief of Thrombosis Division of the Department of Medicine and the director of the Cardiovascular Science Graduate Program at Baylor. His research focuses on the structure-functional relationship of the adhesive ligand von Willebrand factor and its cleaving metalloprotease ADAMTS-13, especially the role of VWF-ADAMTS-13 in the development of bleeding and thrombotic complications of patients with traumatic brain injury and those with terminal heart failure and supported by left ventricular assist devices.

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von Willebrand factor (VWF) is a glycoprotein serving as an adhesive ligand for hemostasis, which is the process of bleed arrest at the site of vascular injury. It is a large protein of 250 kDa that is made even larger by forming multimers containing 2-20 multimers of different sizes. VWF functions to capture platelets to the site of vascular injury to initiate hemostasis but also causes vascular occlusion (thrombosis). The hemostatic and prothrombotic activities of VWF is directly associated with its multimer sizes with large and very large multimers being prothrombotic. This VWF hyperadhesive activity can be reduced by the metalloprotease ADAMTS-13 (A Disintegrin and Metalloprotease with a Thrombospondin Type I Motif 13), which cleaves VWF to smaller multimers. ADAMTS-13 cleaved VWF multimers maintain hemostatic activity but no longer cause thrombosis. VWF activity is also regulated by hemodynamic and other physical forces by activating VWF to bind its receptor on platelets, by changing its multimerization, and by accelerating its cleavage by ADMATS-13. As an acute phase reactant, VWF is substantially released from injured or inflamed endothelial cells to propagate the traumatic brain injury-induced endothelial injury and intravascular coagulation by mediating vascular injury caused by extracellular vesicles and by activating platelets. In addition, the high shear stress blood flow driven by left ventricular assist devices (LVAD) results in the loss of hemostatic active VWF multimers by binding to platelets or excessively cleaved by ADAMTS-13. In both pathologies, recombinant ADAMT-13 modifies the VWF-induced pathologies. The state of the VWF-ADAMTS-13 interaction is therefore important for both hemostasis and thrombosis.