



Robert M. Berne CVRC Seminar

The Robert M. Berne Cardiovascular Research Center Presents

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Cellular and molecular mechanisms driving aberrant development in a novel model of Venous Malformation

Patients with venous malformation (VM) develop vascular lesions consisting of massively enlarged and dysmorphic veins. These lesions cause significant disfigurement, chronic pain, and complications such as bleeding and coagulopathy. VMs are associated with somatic, gain-of-function mutations in the endothelial cell receptor kinase TIE2. The cellular and molecular mechanisms by which TIE2 mutations drives formation of VM are still not fully understood. To unravel these mechanisms, we have developed the first-to-date murine model of TIE2 p.L914F-driven VM.

**Thursday September 24, 2026
11:00 AM-12:00 PM
MR5 3005**

****Refreshments served****

Hosted By: Joshua Wythe, PhD

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