The immune complement system forms a first line of defense against pathogens and clears damaged host cells. Complement activation is tightly controlled, and excessive complement activation can result in the deleterious attack of healthy host tissue such as the vascular endothelium. Complement activation can occur via three pathways (i.e., classical, lectin, and alternative pathway), all of which converge on the induction of the terminal pathway ultimately resulting in the formation of the membrane attack complex (MAC, C5b-9), which deposits on target cells, forms membrane pores, and causes osmotic cell lysis. My overall research program is focused on investigating the mechanisms of complement-mediated damage on the vascular endothelium (and other cells/tissues) at a cellular and molecular level, as well as potential cellular defense mechanisms resulting from such insult. Unravelling these mechanisms in detail will not only advance our insights into the relation between inflammation and the vasculature but will likely also be applicable to several other tissues/organ systems. Overall, our preliminary results point towards a new fundamental concept of complement as energy switch on the cellular level.

“Translational research in complement-mediated research”

The Centre for Blood Research
VIRTUAL SEMINAR SERIES

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Professor of Pediatrics, University of Toronto
Senior Scientist, Cell Biology Program, SickKids Research Institute

Wednesday, October 6, 2021
11:00am - 12:00pm PT