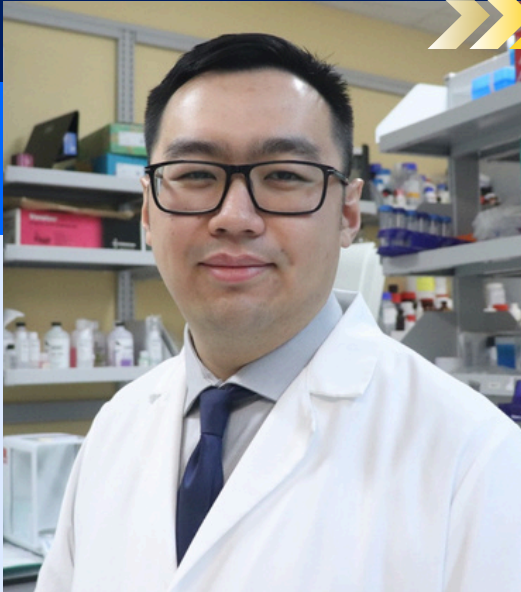




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Supervised by
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*“Outer Blood-Retina Barrier-on-a-Chip:
exploiting multifactorial vascular tissue
development cues to recapitulate
retinal pathophysiology”*

Seminar Abstract: Permanent blindness is a debilitating condition that profoundly affects quality of life. In 2020, it was estimated that about 43 million people suffer from permanent blindness globally. Retinal degeneration, which leads to irreversible blindness, can be caused by inherited disease, toxicity, systemic disease, physical trauma, or unhealthy lifestyle choices. Macular dystrophies such as age-related macular degeneration (AMD) are the leading cause of blindness in western countries. Dysfunction of the outer blood retinal barrier (OBRB), which comprises the retinal pigment epithelium (RPE), the Bruch’s Membrane, and the choriocapillaris, is implicated in retinal degeneration, but the etiology of retinal degeneration remains elusive. Animal-based models of the OBRB are severely limited by major physiological differences and practical constraints, reducing clinical translatability of interventions for retinal degeneration. Microphysiological systems (MPS) are promising alternative platforms that can integrate human-relevant tissues with biomaterials and microfluidic technologies to recreate dynamic tissue-specific microenvironments and support high-content biomolecular analyses. However, current MPS for the OBRB struggle to reproduce tissue-specific angioarchitecture, capture ECM-tissue interactions driving retinal (patho)physiology, or generate clinically relevant readouts of retinal toxicity and barrier function. Hence, improved in vitro models of the OBRB are needed.

To address these gaps, I leveraged engineered biomaterials, microfluidics, and the μ SiM (Microphysiological system enabled by a Silicon nitride Membrane) device platform to develop improved models of the OBRB. Matrix metalloproteinase (MMP)-degradable poly(ethylene glycol) (PEG) hydrogels were used to establish a biomaterial design framework in which degradability, mechanics, and crosslinker peptide chemistry are tuned in tandem to engineer tissue-specific microvascular networks like the choriocapillaris. The μ SiM was used to create a functional model of the OBRB (μ SiM-OBRB) that captures retinal pharmacokinetics and reproduces drug-induced retinal toxicity. Finally, injectable hydrogels were incorporated into μ SiM-OBRB to juxtapose RPE barriers with 3D microvascular networks and model pathological choroidal neovascularization. Collectively, these studies illustrate how biomaterials-based tissue engineering strategies can be integrated with microphysiological systems to develop human-relevant models of specialized microvascular tissues such as the OBRB.

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