

THE GIS SPEAKER SERIES

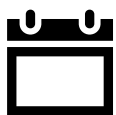


Cancer cells hijack RNA processing to transform cells inside and out

Professor Katherine BORDEN

Northwestern University

Host: Shobha Vasudevan



Thursday 15 October 2026
10.00am – 11.00am



GIS L2 – Seminar Room
60 Biopolis Street, Genome, Singapore 138672

About The Speaker

Katherine L.B. Borden, PhD, FRSC, FCAHS, Professor, Northwestern University, recently moved from Université de Montréal; Hospira Chair Foundation for Translational Cancer Biology. She received her PhD from Yale University in protein biophysics with Fred Richards and postdoctoral training in structural and cancer biology at The Crick Institute (formerly NIMR) and Cancer Research UK (formerly the Imperial Cancer Fund) in London. Her work focusses on understanding the mechanisms and impact of dysregulated RNA metabolism in cancer. She studies the eukaryotic translation initiation factor eIF4E as an exemplar. Her studies unearthed novel modes by which cancer cells hijack genetic information, in the form of messenger RNA, and demonstrated new mechanisms by which RNA maturation and RNA export influence cell physiology and support cancer growth. Her molecular snapshots elucidated new mechanisms for regulation and engagement of these steps. These findings drove first-in-class clinical trials demonstrating that targeting eIF4E and represents a promising avenue for the development of novel therapeutics, revealed novel modes of drug resistance and showed these were targetable in patients. These achievements were recognized by awards including the Stohlman Scholar award from the Leukemia and Lymphoma Society USA, 3 term Tier 1 Canada Research Chair, Distinguished Scientist of the Canadian Society for Clinical Investigation, CSMB Canadian Science Publishing Senior Investigator Award and is a Fellow of the Royal Society of Canada and Fellow of the Canadian Academy of Health Sciences. She has over 150 publications and given more than 160 invited lectures.

About The Seminar

RNA metabolism can be hijacked by cancer cells to re-write and/or amplify the message sent from the DNA. This in turn can underpin the oncogenic phenotype. The eukaryotic translation initiation factor eIF4E is an m7G cap-binding protein which can escort messages through various RNA metabolism steps including translation. Indeed, it has been known since the 1990s that eIF4E is present in both the nucleus and cytoplasm of eukaryotic cells. In the nucleus, eIF4E binds ~3000 transcripts and influences multiple stages of their processing. eIF4E-targeted mRNAs can act in the same biochemical pathways whereby eIF4E coordinates biochemical responses as seen in RNA regulons. These pathways include the extracellular hyaluronan-CD44-ezrin signalling pathways. Identification of these eIF4E-targets led to the finding that eIF4E enhances cell motility in cancer cells which is consistent with its frequent association with metastatic disease. We also observe evidence for new forms of localized translation in pseudopods of cancer cells to prime these cells for motility and a new role for the motility machinery for translation there. Further, we will discuss that clinical targeting of eIF4E in high-eIF4E AML patients leads to objective clinical responses including complete remissions corresponding to eIF4E targeting in these patients and new modalities of drug resistance that can arise.