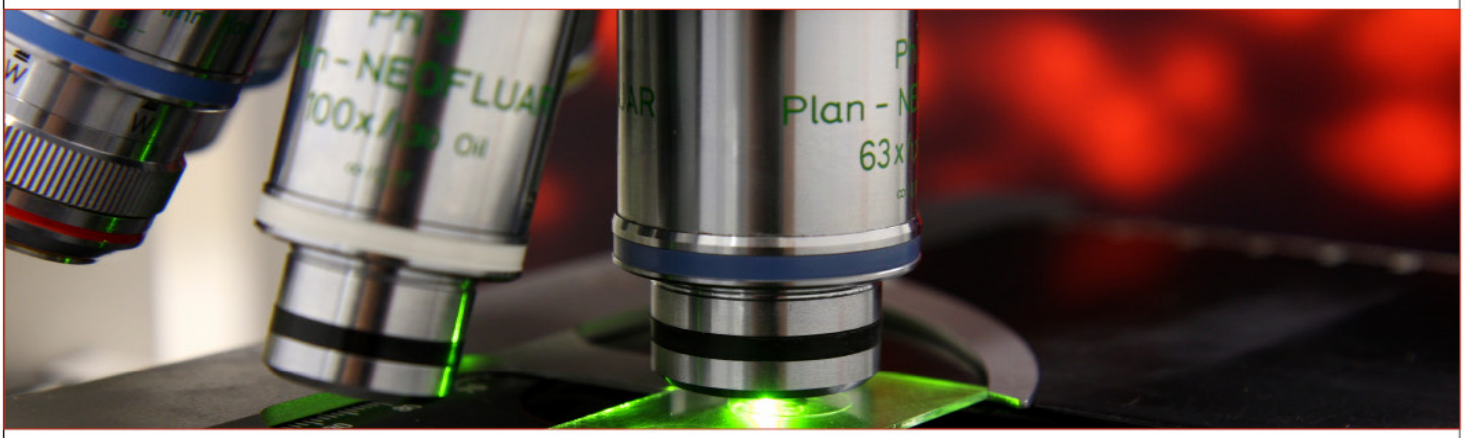
SÉMINAIRES ET CONFÉRENCES



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Targeting splicing-factor regulatory networks in cancer and aging

Alternative splicing expands proteomic diversity by allowing a single gene to generate multiple protein isoforms. In cancer, this process is frequently hijacked to produce oncogenic variants that promote tumor growth, invasion, and therapy resistance. Rather than being a byproduct of transformation, aberrant splicing can act as a primary driver—and a therapeutic vulnerability. Our work addresses three central questions: which splicing events are pathogenic, what drives these changes in tumors, and how they can be targeted. We examine the transcriptional and post-transcriptional mechanisms regulating splicing factor levels and show how their disruption in cancer alters isoform expression. Using splice-switching approaches, we demonstrate that restoring normal isoform expression induces selective anti-tumor effects. Finally, we explore how aging—the greatest risk factor for breast cancer—modulates splicing and gene expression in mammary tissues. Together, our findings reveal new principles of post-transcriptional regulation and uncover tractable vulnerabilities in the splicing machinery.

Mardi, 8 juillet 2025, 11h30

Pavillon Joseph-Armand-Bombardier, Salle : 1035

ET

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