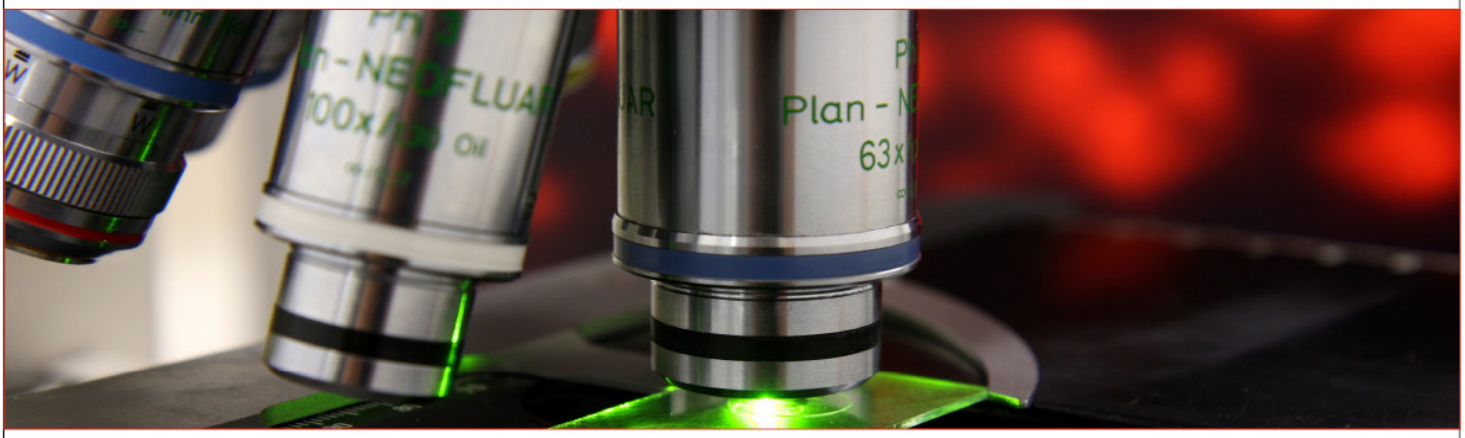
SÉMINAIRES ET CONFÉRENCES



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A truncated SARS-CoV-2 nucleocapsid protein enhances virus fitness by evading antiviral responses

Viruses face selective pressure to evade cellular antiviral responses to control the outcome of an infection. Since its emergence in humans, SARS-CoV-2 accrued many mutations; however, the functional consequence of many such genetic changes remains unexplored. Here, we show that SARS-CoV-2 produces a truncated form of the nucleocapsid protein, called N*M210. Due to the acquisition of a viral transcription regulatory sequence (TRS) in the N gene, certain variants markedly increase N*M210 production. N*M210 is a dsRNA binding protein which inhibits multiple arms of the cellular antiviral response including interferon induction and stress granule formation. Using a panel of recombinant SARS-CoV-2 viruses (rSARS-2) that increased or decreased N*M210 production, we show that enhanced N*M210 production increases virus fitness, in part due to its ability to potently block stress granules. To evade the cellular antiviral response, SARS-CoV-2 has evolved a mechanism to increase the production of a truncated form of the N protein which limits activation of dsRNA-induced antiviral responses, tipping the balance in favour of the virus in the battle for control of the cell.

Vendredi, 11 juillet 2025, 11h30

Pavillon Joseph-Armand-Bombardier, Salle : 1035

ET

Lien ZOOM

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