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### *Insights into migraine mechanisms from genetic mouse models of the disease*

**Vendredi 18 novembre 2022**

12 h à 13 h

En ligne

<https://umontreal.zoom.us/j/88066173443?pwd=ajlvdzA0a09FSGIFc3NuRUdFNVhNdz09>

#### Research Interest

Migraine is a common disabling brain disorder affecting more than 10% of the population. The primary cause of migraine lies in the brain, but the nature and mechanisms of the brain dysfunction(s) in migraine remain unclear and controversial, and drug therapy for preventing and treating migraine remains unsatisfactory for many patients (despite the great societal and personal costs: cf WHO ranking of migraine as one of the 20 most disabling diseases and the enormous cost to the economy: 27 billions per year in Europe). Recent findings point to cortical spreading depression (CSD) as a key player in the pathogenesis of migraine, as CSD underlies migraine aura and may also trigger the mechanisms for migraine headache (Pietrobon and Striessnig, 2003 Nat Rev Neurosci; Pietrobon, 2005 Neuroscientist). Unique insights into the pathophysiology of migraine can be gained by studying the molecular and cellular mechanisms of familial hemiplegic migraine (FHM), a monogenic subtype of migraine with aura, whose typical attacks have headache and aura symptoms similar to those of the common forms of migraine (Pietrobon, 2007 Neurotherapeutics). The aim of Pietrobon's project is to study, in particular, the mechanisms of FHM type 1 (FHM1), that is caused by missense mutations in the CACNA1A gene, encoding the pore-forming subunit of a neuronal voltage-gated Ca<sup>2+</sup> channel (CaV2.1 or P/Q-type). CaV2.1 channels are located in somatodendritic membranes and in presynaptic terminals throughout the brain, where they play a dominant role in initiating fast synaptic transmission at most central synapses (Pietrobon, 2005 Curr Opin Neurobiol).

**Entrée libre**

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