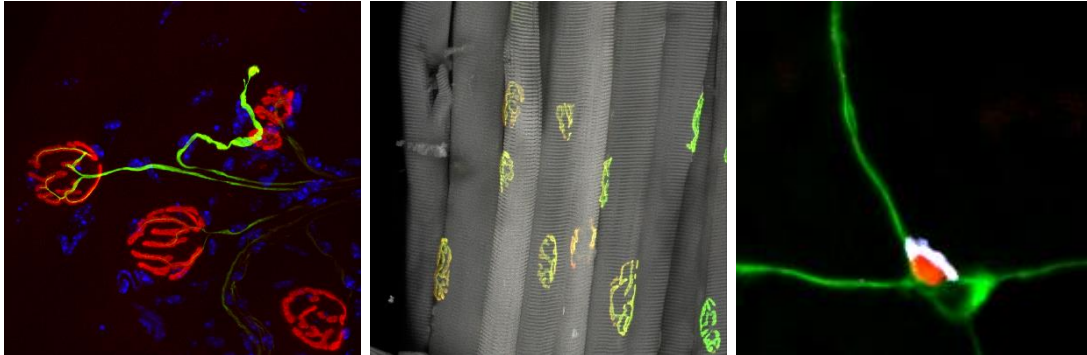


MASTER POSITION



Group of Prof. Castets (CMU, UNIGE) – Muscle and Neuromuscular Junction

The group focuses on the mechanisms involved in skeletal muscle homeostasis, and on the pathomechanisms leading to muscle deterioration in neuromuscular diseases and systemic conditions. In particular, the group investigates the signaling pathways regulating the maintenance of 1) neuromuscular junctions (NMJ), and 2) muscle stem cells (MuSCs). We are looking for highly motivated master students, who will manage one of the two following projects:

1) CaMKII isoforms in skeletal muscle: specific roles in muscle function and NMJ maintenance

Ca²⁺-associated pathways have been shown to be key players in the regulation of NMJ maintenance in skeletal muscle. In particular, Ca²⁺/calmodulin-dependent protein kinases II (CaMKII) are thought to regulate the expression of synaptic genes and the dynamics of synaptic proteins at the NMJ. However, the roles of the different CaMKII isoforms remain largely unknown. The aim of the project is to investigate the functions of specific isoforms of CaMKII in muscle, by using different mouse models, cell culture, proteomic analyses and *in vivo* functional assay. As we recently established that some of these CaMKII isoforms are deregulated in neuromuscular diseases, the project may unveil yet-unknown pathomechanisms leading to muscle dysfunction in these conditions.

2) Metabolic profiling of MuSCs in healthy and pathological conditions, towards improvement of cell therapy

– MuSCs are essential for muscle regeneration, and MuSC dysfunction is associated with numerous pathological conditions. Based on previous reports and recent results, we hypothesize that MuSC cell fate is driven by metabolic switches, and that metabolic perturbations contribute to MuSC dysfunction. The aim of the project is to characterize MuSC metabolism, using a novel *in situ* method developed in the lab. The student will screen changes in MuSC metabolism upon muscle injury, as well as in several pathological conditions, using muscle biopsies from patients and different mouse models. The results will bring new insights on the pathomechanisms leading to MuSC decline in disorders, and will help to design strategies to improve MuSC-based cell therapies, by modulating MuSC metabolism.

For more details: <https://www.unige.ch/medecine/phym/en/research/perrine-castets/>

List of publications: <https://www.unige.ch/medecine/phym/en/research/perrine-castets/publica/>

Please send your CV and letter of motivation to: perrine.castets@unige.ch

Prof. Perrine Castets, PHYM department, University of Geneva