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***Salle de conférence Bâtiment 84, Ecole Polytechnique***

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## *Unconventional type 1 Myosins in animal Left-Right asymmetry: from chirality induction to asymmetry suppression*

The presence of bodily Left-Right (LR) asymmetries is a commonly observed feature throughout the animal kingdom, but the mechanisms through which LR asymmetries are established have long been thought to differ among different species. While a directional, cilia-driven fluid flow in an anatomical structure that functions as a central LR Organizer (LRO) achieves symmetry breaking in many vertebrates, other species such as the fruitfly Drosophila establish LR through a mechanism that involves neither cilia nor a central LRO. Our lab established that in spite of these major differences, the unconventional type 1 Myosin Myo1D acts as an evolutionary conserved regulator of animal LR asymmetry that controls both cilia-dependent (zebrafish) and cilia-independent (flies) mechanisms of symmetry breaking (Juan et al, Nat.Com. 2018).

In addition to Myo1D, the zebrafish genome encodes the closely related protein Myo1G. In contrast to Myo1D that regulates the morphogenesis of the ciliated LRO, Myo1G regulates the subsequent transfer of laterality information from the central LRO to target tissues (Kurup et al, Nat.Com. 2024). While these findings further substantiate an important function of Myosin1 proteins as key regulators of animal LR asymmetry, the fact that neither cilia nor a central LRO are present in Drosophila raises the question whether a common, Myosin1-dependent cellular mechanism that underlies the establishment of LR asymmetry in different animal species still remains to be identified.
To address this issue, our current work focuses on the importance of Myosin1 proteins for the chiral deformation of embryonic tissues, a morphological event required for the morphogenesis of all lateralized organs. Of particular interest, our recent work reveals that proper embryonic development requires the activity of both chirality-inducing and asymmetry-supressing pathways.

Juan T, Géminard C, Coutelis JB, Cerezo D, Polès S, Noselli S, Fürthauer M. Myosin1D is an evolutionarily conserved regulator of animal left-right asymmetry. Nat Commun. 2018 9(1):1942. doi: 10.1038/s41467-018-04284-8.

Kurup AJ, Bailet F, Fürthauer M. Myosin1G promotes Nodal signaling to control zebrafish left-right asymmetry. Nat Commun. 2024 15(1):6547. doi: 10.1038/s41467-024-50868-y.