Master Biologie Moléculaire et Cellulaire 'BMC',
Université Paris Cité - *UFR Sciences du Vivant*

Parcours : **Biologie et Développement Cellulaires 'BDC'**
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Fiche de Projet de Stage de M2, 2026-2027

Unité INSERM ou CNRS ou Université : **Institut Jacques Monod** **UMR-CNRS 7592** **Intitulé Equipe : Polarité et Morphogenèse** **ED d'appartenance : BioSPC** **Responsable de l'Equipe : Antoine GUICHET**	**Responsable du Stage : Véronique BRODU** **Contacts** Adresse : **Institut Jacques Monod** **Bât.Buffon - 15 rue Hélène Brion** **75205 PARIS CEDEX 13 - FRANCE** Email : **veronique.brodu@ijm.fr** Tel : **+ 33 1 57 27 80 78**

Titre du projet : Actin cytoskeleton dynamics during 3D cell intercalation in Drosophila tracheal morphogenesis

Résumé du Projet de Stage (en 300 mots maximum, mots clés en gras)

Morphogenesis relies on coordinated cell movements such as **intercalation,** which reshape cells and tissues. These processes depend on regulated interactions between **adherens junctions** (AJs) and the **actin cytoskeleton** to generate mechanical forces. E-Cadherin (E-C-ad, a core AJ component, cooperates with the actomyosin network to control cell rearrangements. These mechanisms are well characterised in two-dimensional (2D) systems, where Myosin II, drives contractile force generation but remain poorly understood in **three-dimensional (3D) tissues**. The *Drosophila* embryonic respiratory or so called tracheal system provides a powerful in vivo model to study **junction remodelling in 3D.**

We have recently characterised the actin dynamics during 3D cell intercalation in tracheal branch morphogenesis. *In vivo* imaging reveals a progressive enrichment of actin from the cytoplasm to the AJs, along with pulsatile dynamics at tricellular AJs (tAJs), sites of high tension. Strikingly, this actin redistribution occurs independently of Myosin II, challenging the prevailing view that actomyosin contractility drives force generation and suggesting an alternative mechanism of cell remodelling.

This project aims to define the subcellular origin of this tracheal actin network and to elucidate how its dynamic redistribution at AJs drives force generation during branch morphogenesis. We will combine advanced imaging, intracellular photomanipulation to dissect the underlying mechanisms

1- We will investigate **the origin of actin accumulating in pulses at tAJs** and enriching at AJs during intercalation. Using *Drosophila* lines expressing a photoconvertible actin marker, we will track actin pools in vivo to determine whether junctional actin derives from polymerisation, intracellular transport, or transient anchoring between cytoplasmic and junctional compartments.

2. We will determine if the association of the actin cytoskeleton with E-Cad enables **force transmission driving cell shape changes** and migration. By performing FRAP experiments on E-Cad, we will test whether E-Cad recycling depends on its association with actin. We will then measure forces exerted on the apical membrane by performing laser nanosurgery and measuring recoil dynamics.

Publications de l'équipe relatives au projet de stage (max 5)

- Girdin controls the pace of 3D tracheal cell intercalation by coupling adherens junctions to the actin cytoskeleton in Drosophila. Carvalho S, Laprise P, **Guichet A**, Brodu V. Development. 2026
- Microtubule-dependent apical restriction of recycling endosomes sustains adherens junctions during morphogenesis of the *Drosophila* tracheal system. Le Droguen PM, Claret S, **Guichet A**, Brodu V. Development. (2015)