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Titre.

Interplay between the actin network and Myosin1C in cell shape remodelling during tracheal system morphogenesis in *drosophila*

Projet :

Epithelium remodelling is an essential mechanism for organogenesis during which cells change shape and positions while maintaining contact with each other. Similar mechanisms are used again during solid tumour invasion. Adherens junctions (AJs) mediate stable cohesion between cells but must be actively reorganised to allow morphogenesis. An essential component of AJ is the transmembrane protein E-Cadherin (E-Cad). Through its intra cellular domain, E-Cad is associated with different factors involved in the regulation of the membrane dynamics but also with cytoskeletal elements of the acto-myosin and the microtubule (MT) networks. This latter one represents a dynamic network crucial for cell migration, cell deformation, cell adhesion and molecular transport. This network contributes to the establishment, the maintenance and the remodelling of cellular contacts.

To further uncover fundamental processes essential during collective cell migration, we study the formation of the respiratory or so called tracheal system in the *drosophila* embryo. The tracheal network is an excellent model system to explore, *in vivo* in a whole organism, cellular junction remodelling during organ formation in 3D.

Recently, we showed that MTs are required for the apical targeting of newly synthetized E-Cad molecules. Thus the MT network contributes to maintain the proper E-Cad level that is essential to preserve the integrity of all tracheal branches.

We now focus our work on the role of the acto-myosin network and will address the followings points:

1- We will characterise the dynamics of actin distribution during tracheal morphogenesis using a range of *in vivo* markers and correlate this distribution to cell behaviour.

2- We will determine the actin functional requirement during tracheal cell migration. To approach this question, we have established new tools to depolymerise the actin network *in vivo*. We will evaluate their effect on tracheal morphogenesis.

3- We will determine the localisation and the functional importance of Myosin 1 family members, more precisely of Myosin1C. We will develop an original approach to deplete Myosin1C function only in tracheal cells. We will also test as to whether Myo1C depletion could be improved by modulating the depolymerisation of the actin network.

Publications de l'équipe, relatives au stage proposé

- Le Droguen P. M., Claret S., Guichet A. and **Brodu V**. (2015). Microtubule-dependent apical restriction of recycling endosomes sustains adherens junctions during morphogenesis of the Drosophila tracheal system. Development. 2015 142(2):363-74
- **Brodu V.** et al. (2010) A developmentally regulated two–step process generates a non–centrosomal microtubule network in *Drosophila* tracheal cells. Developmental Cell 18(5):790–801.