JUILLET 2018

# Lettre mensuelle

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@SFBD_biodev.

## ACTUALITES :

## UN CONGRES SBCF-SFBD-ITMO organisé par des jeunes scientifiques en 2019

La SFBD et la SBCF ont confié à de jeunes scientifiques, l'organisation scientifique d'un congrès sur le thème:

**"Biologie Cellulaire et Biologie du Développement du futur", au printemps 2019, avec le soutien de l'ITMO.**

Les personnes ou groupes de personnes intéressées étaient invités à soumettre :

- le thème d'une session qu'ils souhaiteraient mettre en place pour ce congrès, avec le nom d'un conférencier invité de leur choix
- une courte lettre pour se présenter et expliquer leur motivation pour organiser ce symposium
- éventuellement une ou deux suggestions de keynote speakers pour le congrès.

20 propositions ont été reçues et sont en cours d'évaluation !!
Merci à tous pour vos candidatures et votre motivation.
A très bientôt pour la suite…

## 5 BOURSES DE VOYAGE pour le congrès joint SPBD, SEBD, SFBD à Porto en novembre prochain (deadline 31 juillet)

La SFBD distribuera 5 bourses de voyage d'un montant de 400 euros à des étudiants de thèse ou Post doc qui souhaitent particpiper au congrés co-organisé par les société Portugaise, Espagnole et Française de biologie du Developpement

The SFBD is happy to announce that it will distribute 5 travel awards (400€ each) for PhD students (year 2 onwards) and Post-docs to attend the joint meeting of the Portuguese, Spanish and French Societies for Developmental Biology, Porto, Nov 7-10 2018. The meeting offers a rich and diverse program which you can see here: http://devbiomeetingporto2018.pt/

# *Quoi de neuf en bio Dev ?*

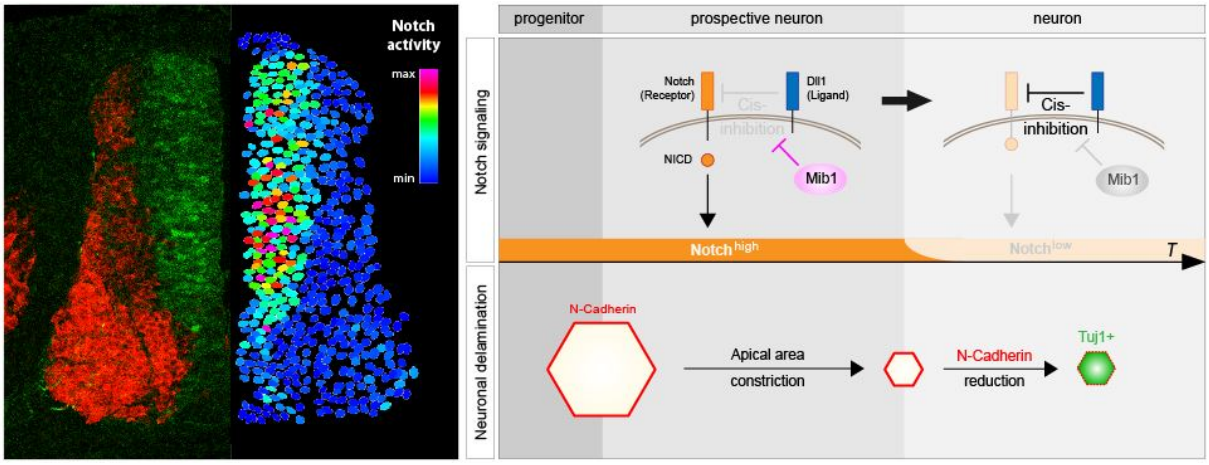
## Neurons maintain Notch activity transiently while they prepare to leave the neuroepithelium

During early development of the vertebrate central nervous system, neural progenitors are organized in a pseudo stratified layer of cells that forms the neural tube. The cohesion of this neuro-epithelium relies on the apical-basal organization of progenitors, which adhere to each other via a sub-apical junctional network. In particular, N-cadherin is a key component of this organization. When neurogenesis starts, neurons are born in a salt and pepper manner from asymmetric divisions of progenitors. They reorganize their morphology and relocate in the “mantle zone”, in the periphery of the tube. Leaving the apical surface involves the constriction of the apical domain and the reduction of adhesion through N-cadherin down-regulation. However, the signaling mechanisms allowing these events to be correctly coordinated and insuring the seamless delamination of nascent neurons remained to be explored

In this study*, we first investigated the dynamics of Notch activity during the transition period extending from mitotic exit to the actual delamination of the newborn neuron. In the classical view, neural progenitors display a high level of Notch activity, and switch it off as they differentiate. We had recently found that the ubiquitin ligase Mindbomb1 (Mib1) is inherited asymmetrically by the future neuron during asymmetric divisions of progenitors. Mib1 is required for the trans-activating function of the Notch Ligand Dll1, suggesting that expression of Dll1 and Mib1 inheritance by the future neuron contribute to the maintenance of high levels of Notch activity in surrounding progenitors, including its sibling. However, using a transgenic chick line that reports the activity of the Notch pathway with a fluorescent protein, we were surprised to observe that Notch signaling was still active in future neurons for many hours after they had exited the cell cycle. We therefore asked how this activity was controlled, and its importance for the differentiation process. Through a series of gain and loss of function experiments performed in clonal and non-clonal conditions in the chick neural tube, we were able to show that Mib1, inherited by the prospective neuron, not only is important to promote Notch transactivation in neighbors (by Dll1), but that in doing so, it also prevents Dll1 from cis-inhibiting Notch, such that the prospective neuron remains competent for activation of the Notch pathway.

However this is only transient: as the new neuron matures, it expresses increasing levels of Dll1. We propose that Dll1 in the differentiating cell progressively outgrows the capacity of Mib1 to convert it into a trans-activator, leading to Notch cis-inhibition by its own ligand. We then explored whether the maintenance of Notch activity in prospective neurons mattered for the developing tissue by artificially switching off Notch prematurely in prospective neurons. Strikingly, we found that they were now unable to organize the events that lead to delamination: they differentiated faster and detached too early without shrinking their apical domain, leaving behind holes that weakened the whole neuroepithelial organization, ultimately leading into its collapse, a phenotype also observed following Mib1 inhibition. Importantly, the phenotypes could be fully rescued by forcing apical constriction using the actin-binding protein Shroom3.

This study shows that Dll1 switches from an early trans-activating to a later cis-inhibitory activity in prospective neurons. This switch is controlled by Mib1, and its temporal regulation allows to couple the full constriction of the apical domain with the delamination of nascent neurons. Further work will be necessary to determine whether Notch signaling dynamics directly controls the delamination events or only provides the time window necessary for seamless delamination by delaying terminal differentiation.

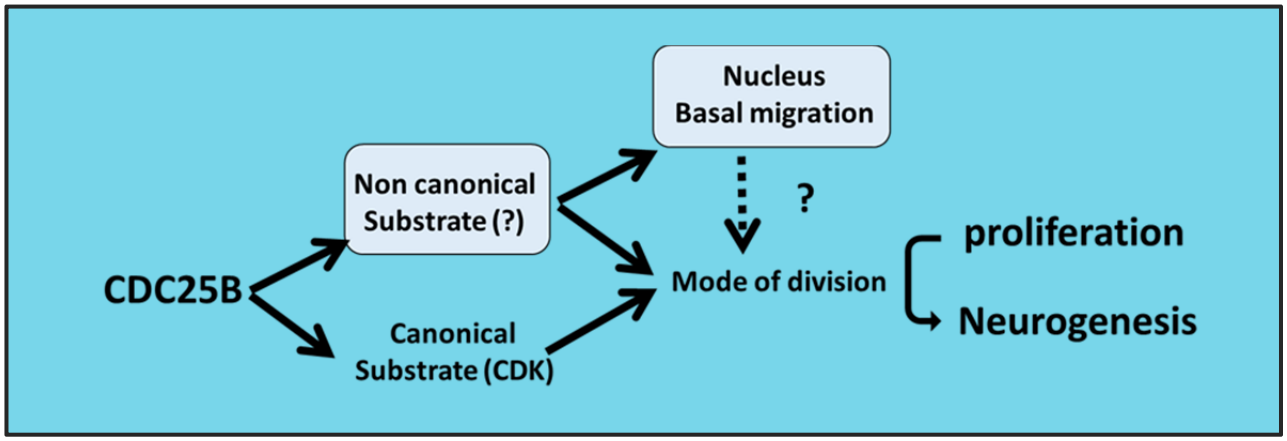



*Left: Notch signaling activity in the developing spinal cord was measured using a transgenic chick reporter line (Venus reporter, green).*
*Right: a model of the dynamic role of Mib1 and Dll1 in the regulation of Notch activity in differentiating neurons, and its temporal relationship with the morphological changes at the apical surface during the delamination process.*

* Baek C, Freem L, Goïame R, Sang H, Morin X#, Tozer S# (2018). Mib1 prevents Notch cis-inhibition to defer differentiation and preserve neuroepithelial integrity during neural delamination. *PLoS Biology, 16(4):e2004162.*

**Neurogenic decisions require a cell cycle independent function of the CDC25B phosphatase**

A fundamental issue in developmental biology and in organ homeostasis is understanding the molecular mechanisms governing the balance between stem cell maintenance and differentiation into a specific lineage. Accumulating data suggest that cell cycle dynamics play a major role in the regulation of this balance. The results presented in this paper show that the G2/M cell cycle regulator CDC25B phosphatase is required in mammals to finely tune neuronal production. In chick neural progenitors, CDC25B activity favors fast nuclei departure from the apical surface in early G1, stimulates neurogenic divisions and promotes neuronal differentiation. Moreover, a designed mathematical model indicates that within a limited period of time, cell cycle length modifications cannot account for changes in the ratio of the mode of division. Finally, using a CDC25B mutated form that cannot interact with CDK, the authors show that part of CDC25B activity is independent of its action on the G2 length.




**Bonnet** F, Molina A, Roussat M, Azais M, Vialar S, Gautrais J, Pituello F, Agius E. (2018) Neurogenic decisions require a cell cycle independent function of the CDC25B phosphatase. Elife. 2018 Jul 3;7. pii: e32937. doi: 10.7554/eLife.32937. [Epub ahead of print]

# *OFFRES DE POSTES*

***(voir détail des offres de group-leaders, post-docs et doctorants en fin de lettre)***

- **Postdoc position at the interface between microbial ecology, microbial genetics, chemical ecology, and plant development in Lyon** postdoc will carry out the project in the participating laboratories in the Lyon – St-Etienne area. Start date between October 1st 2018 and January 2nd 2019.

- **International PhD Fellowship "Gene Regulation by RNA modifications"** University of Grenoble, France, and University of Geneva, Switzerland Supervised by Dr. M.O. Fauvarque and Pr. R.S. Pillai

- **Studentship available in Claudio Stern's lab on "Dynamics of cell behaviour during somite formation"**
  **http://thenode.biologists.com/studentship-available-in-claudio-sterns-lab/jobs/**
  Starting date: 1 October 2018 or earlier by arrangement.

**If you want your offer to be advertised here, please send your ad to:** sophie.vialar@univ-tlse3.fr, cedric.maurange@univ-amu.fr,

# *SOUTENEZ LA SFBD !*

## DONS SFBD

**Dorénavant, pour chaque don fait à la SFBD, un reçu fiscal sera délivré qui permettra une déduction d'impôt de 66%N'hésitez plus !**

**Pour faire un don il faut aller ici: http://www.sfbd.fr/new-website/spip.php?article37**

## Training school


http://www.mbl.edu/education/courses/

## MEETING

aviesan
alliance nationale
pour les sciences de la vie et de la santé

ITMO BIOLOGIE CELLULAIRE, DEVELOPPEMENT ET EVOLUTION

sbcf
Société de Biologie Cellulaire de France

SFBD
SOCIÉTÉ FRANÇAISE DE BIOLOGIE DU DÉVELOPPEMENT

**PLACE AUX JEUNES SCIENTIFIQUES POUR L'ORGANISATION DU**

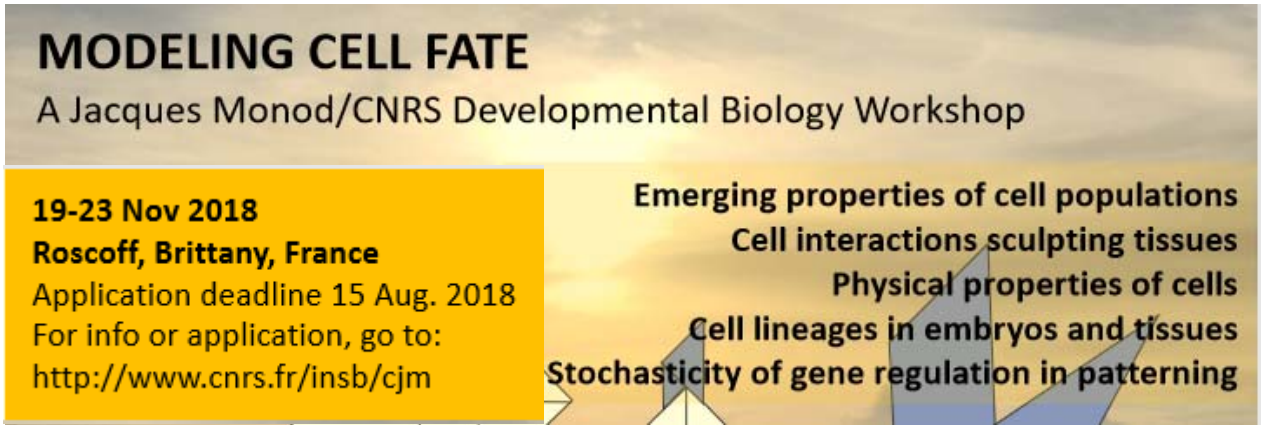
CONGRES 2019:

**"Biologie Cellulaire et Biologie du Développement du futur"**

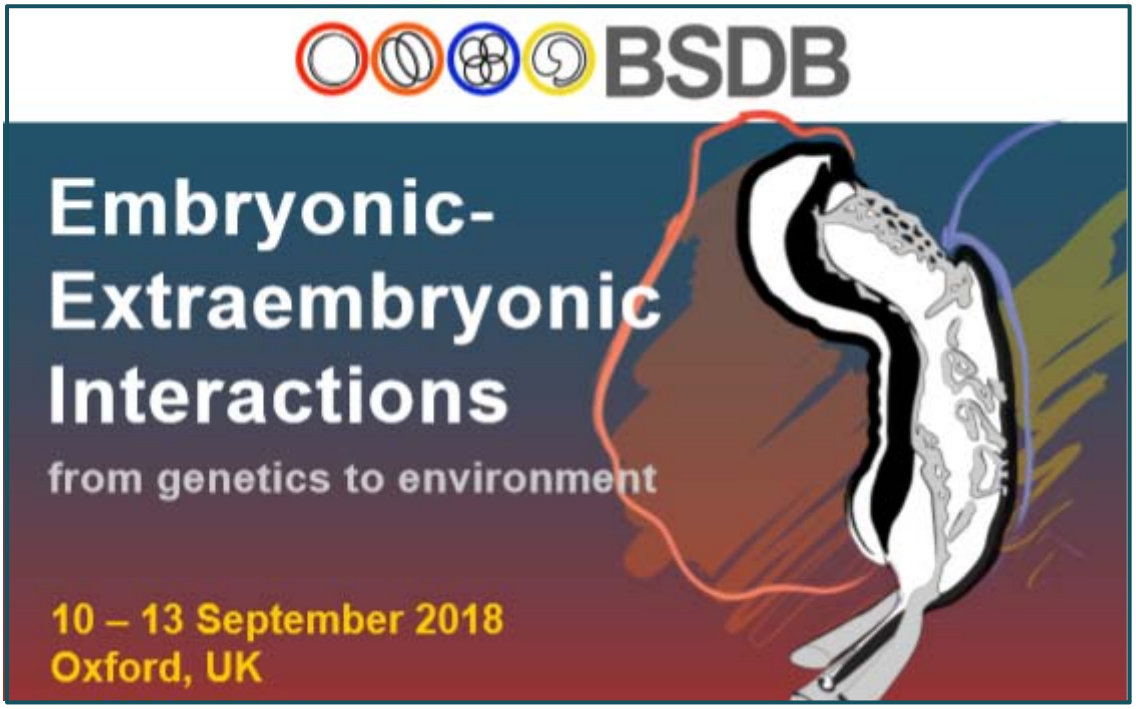
- **le thème d'une session** qu'ils souhaiteraient mettre en place pour ce congrès, avec le **nom d'un conférencier** invité de leur choix
- **une courte lettre pour se présenter et expliquer leur motivation** pour organiser ce symposium
- éventuellement **une ou deux suggestions de keynote speakers** pour le congrès.

Christine Lemaitre (christine.lemaitre@inserm.fr)
Sylvie Robine (sylvie.robine@inserm.fr) avant le vendredi 29 juin 2018.
https://itbcde.aviesan.fr/appel.html

## Jacques Monod/CNRS Workshop




http://www.cnrs.fr/insb/cjm




http://www.bsdbautumn2018.co.uk/

*La SFBD subventionne* ***des écoles thématiques et des congrès*** *(Participation forfaitaire ou prise en charge des frais d'un invité) : N'hésitez pas à nous contacter !!*


Http://frenchdrosophilameeting.com

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## *DETAIL DES ANNONCES ET DES OFFRES DE POSTE*