
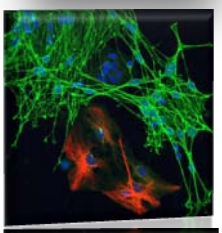
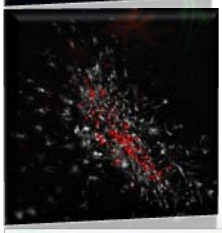

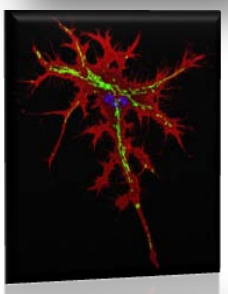

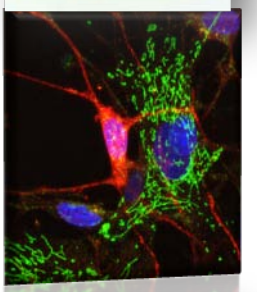

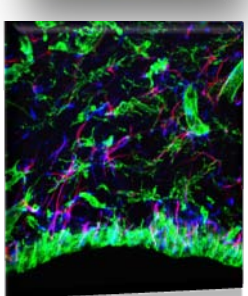

# VII JORNADAS CIENTIFICAS

## INSTITUTO CAJAL 21 al 23 de diciembre de 2009

**<u>21 de diciembre:</u> Visita al Instituto de investigadores postdoctorales en el extranjero**

9:30h. Reunión de bienvenida con la Dirección del Instituto.
9:45-13:00. Visitas a laboratorios.

**<u>22 de diciembre: Jornadas Científicas</u>**

**Sesión mañana**

9:30h. **Anahí Hurtado Chong**: "El papel de IGF-I en la neurogénesis de la zona subventricular y el bulbo olfatorio" (Grupo C. Vicario)

10:00h. **Maria Victoria Gomez-Gaviro**: "Betacellulin promotes cell proliferation in the neural stem cell niche" (Postdoctoral en NIMR-MRC, London. <u>Anfitriona: Paola Bovolenta</u>)

10:30h**. Marta Navarrete**: "Comunicación entre astrocitos y neuronas mediada por endocannabinoides" (Grupo A.Araque)

11: 00h**. M. Laura Ceci**: "Efecto del microambiente en la migración de las células de Cajal-Retzius" (Grupo De Carlos)

11:30h. Café

12:00h. **Carolina Cabezas**: "Transient GABAergic phenotype of newborn hippocampal granule cells" (Postdoctoral en el Institut du Fer a Moulin, París. <u>Anfitrión: Alfonso Araque</u>)

12:30h. **Jorge García Marqués**: "Astropistas para la migración neuronal" (Grupo López-Mascaraque)

13:00h. **Jorge Rubén Cabrera**: "RET acts as a cadherin like protein via its cleavage by caspase" (Postdoctoral en CNRS UMR523, Lyon. <u>Anfitrión: Juan J. Garrido</u>)

**Sesión tarde**

16:00h. **Ana M. Fernández**: "Papel de calcineurina/Foxo en la inflamación asociada a la enfermedad de Alzheimer" (Grupo I. Torres-Alemán)

16:30h. **Alfonso Perez-Escudero**: "Deviations from the minimum-wire configuration in the nervous system of C. elegans". (Grupo: G. Polavieja)

17:00h. **Patricia Martínez Morales**: "Desarrollo Temprano de la Cresta Neural". (Grupo A. Morales)

17:30h. **Julia Makarova**: "Generadores de campo local: tirando de la madeja" (Grupo O. Herreras)

**<u>23 de diciembre</u>**

11:30h. Claustro Científico
13h. Acto Institucional
14h. **Cóctel de Navidad para todo el personal**

**CAROLINA CABEZAS**
**Anfitrión: Alfonso Araque**
Institut du Fer a Moulin, Paris

Dentate gyrus (DG) granule cells (GCs) have been suggested to co-release GABA and glutamate both in the immature hippocampus and in some pathological conditions in the adult. However, direct functional evidence for GABA release from these cells is still missing.
Using a transgenic mouse strain that expresses GFP under control of the GAD67 promoter, we show GFP expression in the DG is mostly restricted to a fraction of granule cells. GFP+ granule cells are most abundant in the young hippocampus but persist at a lower density in the adult. Expression of GAD67 is restricted to recently (15-28 days) differentiated GCs that also express markers of neuronal differentiation such as doublecortin. Combining whole-cell recordings and single-cell RT-PCR, we found these rather immature neurons yet are able to fire action potentials, are synaptically integrated in the DG network and express the molecular machinery required to release GABA. However, photostimulation of individual GFP+ granule cells elicited pure glutamatergic, unitary PSCs both in postsynaptic mossy cells and hilar interneurons, suggesting GABA released at terminals from GAD67+ GCs fails to recruit postsynaptic GABAA receptors. Further experiments are conducted to evaluate whether such receptors may be present at synapses formed onto CA3 pyramidal cells.
We conclude that expression of GAD67 in GCs reflects a specific differentiation state of individual GCs throughout life, and suggest GABA released from their terminals may contribute to their maturation and/or functional integration rather than convey synaptic inhibition to their postsynaptic targets.

**JORGE RUBÉN CABRERA**
**Anfitrión: Juanjo Garrido**
CNRS UMR523, Lyon

RET acts as a cadherin like protein via its cleavage by caspase

RET is a tyrosine kinase receptor (TKR) which is implicated in numerous cellular mechanisms including proliferation, neuronal navigation, migration or differentiation upon binding with GDNF Family Ligands (GFLs). Surprisingly RET is not inactive in the absence of GFLs. RET has been shown to act as a "dependence receptor", activating cell death in the absence of its ligands through a mechanism requiring receptor intracellular caspase cleavage. However in vivo data suggests that RET is not always associated to the balance "cell death-survival" but also provides positional information. We thus investigated whether RET could take advantage of its caspase cleavage to participate on this latter effect. Our data show that RET is early cleaved by caspases in sympathetic neurons. This cleavage releases a N-terminal truncated fragment which behaves as a cadherin-like protein. RET fragment caspase-truncated promotes cell aggregation, modifies cadherin protein environment and inhibits neuronal navigation. In conclusion, we propose that depending on the cell context the caspase cleavage of RET provides two RET fragments: one intracellular domain that triggers cell death in apoptotic permissive settings and one membrane anchored ectodomain with cadherin-like activity. We suggest that this latter function is notably important for the adequate development of the superior cervical ganglion.

**MARÍA VICTORIA GÓMEZ-GAVIRO**
**Anfitriona: Paola Bovolenta**
NIMR-MRC, London

Betacellulin promotes cell proliferation in the neural stem cell niche

In the adult mammalian brain, neural stem cells (NSC) reside in specialized niches that include the subventricular zone (SVZ), the dentate gyrus and the olfactory bulb. In their niche, NSCs are found in proximity to blood vessels and endothelial cells enhance NSC proliferation and neurogenic capacity in vitro. We carried out a comprehensive analysis of the interaction between NSCs and endothelial cells. We show that betacellulin (BTC) induces NSC proliferation and prevents spontaneous differentiation in culture. When infused into the lateral ventricle, BTC induces expansion of NSCs, progenitor cells and neuroblasts. BTC also enhances regeneration of the SVZ niche following depletion of proliferating cells using AraC. Blockade of endogenous BTC, made by endothelial cells and by the choroid plexuses results in a decrease in the number of stem/progenitor cells in the SVZ. Our results suggest that BTC could be a good candidate for regenerative therapies in the future.