Llamado a Contrato de Investigación

Instituto de Investigaciones Biológicas Clemente Estable (IIBCE)

Departamento de Neurofisiología Celular y Molecular

El Departamento de Neurofisiología Celular y Molecular del IIBCE llama a postulantes para **un contrato de investigación** en el marco del proyecto WFL-UY-13/23 #290 “The role of connexins in self-repair induced by endogenous spinal progenitors” financiado por Wings for Life. Spinal Cord Research Foundation. El contrato será por 30 horas semanales por el plazo de un año, renovable por igual período (hasta un máximo de 3 años) en caso de cumplir satisfactoriamente con los informes de actuación. El sueldo nominal es de $ 35.000, el cual que se actualizará por el porcentaje de ajuste de salarios de la Administración Central. Los postulantes deberán poseer una licenciatura -o formación equivalente- en alguna de las siguientes disciplinas: Biología, Bioquímica, Biología Humana, Medicina o Química.

Las personas interesadas deberán enviar antes de las 17:00 h del 2 de febrero de 2024 la siguiente información a russoblanc@gmail.com, haciendo referencia al número del proyecto:

- Currículum vitae
- Escolaridad de grado (y posgrado si correspondiera)
- Carta de motivación

Las personas seleccionadas en la evaluación de méritos serán invitadas a una entrevista a coordinar oportunamente.

Descripción del proyecto

Spinal cord injury produces devastating conditions because of limited self-repair. Although the mammalian spinal cord lacks the ability for self-repair of non-mammalian species, its ependyma reacts to injury by generating new cells that integrate the scar to limit the damage. The manipulation of the ependymal stem cell niche to maximize endogenous repair requires understanding the mechanisms by which proliferation, migration and differentiation of ependymal progenitors are controlled. We recently showed that communication via connexins (Cxs) plays a key role in the early response of ependymal cells to injury. From a translational point of view, understanding the cellular and molecular mechanisms of Cx-mediated plasticity may be important to design strategies to boost the contribution of the ependyma to repair. The central hypothesis of this proposal is that the optimization of the response of the ependyma to injury will shift the balance towards a pro-regenerative scar. We have shown that similar to stem cell niches in the embryo, early in life Cx43 and Cx26 are expressed in ependymal cells which are in general functionally coupled via gap junctions. Cx26 and gap junction coupling decrease as the ependymal stem cell niche becomes dormant in adulthood, but this down-regulation is overruled by injury. In addition to gap junctions, there is a population of permeable Cx43 hemichannels in ependymal cells that may play a part in the earliest stages of injury. Communication via Cxs is involved in wound healing and repair in different tissues by the regulation of proliferation, migration and differentiation. For example, Cx26 in the skin promotes proliferation whereas Cx43 is downregulated in the margins of healing skin wounds but its persistence impairs healing. Based on our previous studies and preliminary results, we hypothesize that

-as in other tissues-Cx43 might act deleteriously whereas Cx26 would promote a better response of ependymal cells to injury that will lead to a scar permissive for regenerating axons. Our specific aims are: To investigate the role of Cx26 and Cx43 in the re-activation of ependymal cells after SCI. Aim 2: To determine the influence of Cxs on the contribution of the ependyma to tissue repair and functional recovery. Aim 3: To explore the effects of Cx26 upregulation in healing and functional recovery after SCI. To pursue these aims, we will apply a multi-technical approach that will provide a better understanding of the mechanisms by which Cx43 and Cx26 regulate the reaction of the ependyma to injury and their impact in the formation of on the glial scar. In vitro and in vivo approaches combining patch clamp recordings, immunohistochemistry, confocal and electron microscopy, DIVER imaging and optogenetics to apply both loss-of-function and gain-of-function approaches using transgenic mice models. We believe the new knowledge on the role Cxs in the ependymal stem cell niche will provide valuable clues to design new therapeutic approaches aimed at increasing the self-repair capabilities of the spinal cord to help improve functional recovery.