Call for expression of interest

POSITION: Post-Doc

RESEARCH TITLE: Neurophysiological basis of itch sensation: ion channels regulating sensory neuron excitability as molecular targets for acute and chronic itch

PROJECT: Chronic itch is an unpleasant, multifactorial and complex symptom associated with a long list of chronic pathologies: eczema, psoriasis, allergic dermatitis, renal failure, liver failure, nerve damage due to herpes and diabetes or Hodgkin's lymphoma, affecting 16-21% of the population according to age. In addition, itching is associated with scratch-induced skin lesions and disorders such as anxiety, depression, and insomnia, representing an important health and socioeconomic problem due to the lack of drugs for non-histaminergic itch.
In all these conditions, pruritic stimuli activate specific sensory neurons that send the message to the spinal cord and later to different areas of the brain, where the sensation of itch is perceived and trigger the scratching response. The recent description of the specific sensory neuron subpopulations involved in this process has started to elucidate some of the neural mechanisms involved in itch signaling. Several ion channels and membrane receptors are involved in this process. From recent RNAseq databases and the functional characterization of pruriceptive neurons, we have identified a new pharmacological target that can be used to decrease neuronal activation and relieve itch sensation. The project aims to functionally characterize these mechanisms, the molecular players involved, validate the target identified and develop new pharmacological tools to treat chronic itch, for which effective drugs are lacking. The project will combine several different techniques from the molecular level (qPCR, in situ hybridization (RNAscope)), to the functional level, including electrophysiological recordings, behavioral assays and animals models and optopharmacology.

REQUIREMENTS: A PhD in neuroscience, biomedicine, neuropharmacology, biophysics, or related disciplines is required, as well as documented previous research work experience.The candidate should have strong ability to work independently, independence in planning, performing experiments and data analysis, full command of English, be self-motivated, goal-oriented and have a positive attitude.

It will be highly considered to have experience in establishing and/or maintenance of cell cultures; molecular biology techniques (in situ hybridization, RNAseq); work with laboratory and transgenic animals (certified title).

It will be desirable to have some experience in electrophysiological techniques (patch-clamp) and molecular imaging techniques (functional imaging, fluorescence techniques).

HOST LABORATORY: The Neurophysiology group is located in the University of Barcelona Medical School and belongs to the IDIBAPS Biomedical Institute (biomedical institute of the

Barcelona Clinic Hospital/Medical School) as well as the UB Institute of Neurosciences. It works on the physiopathology of the sensory nervous system (ion channels in sensory neurons, acute and chronic pain and itch) and has been funded by national/international public grants as well as private foundations. It has collaborative projects with international groups or with biotechnological and pharmaceutical companies, and extensive experience in electrophysiological, biochemical, molecular biology and microscopy techniques and animal behavioral tests.

DURATION AND SALARY: 2 years, gross salary € 35-40.000, based on experience.

APPLY: Potential candidates are requested before 22nd January 2023 to send inquiries a/o a letter of interest explaining how they would fit, a CV, a record of their academic results and the names of two references to: Dr. Xavier Gasull, xgasull@ub.edu. The final recruitment will follow the formal procedures stablished by FCRB/IDIBAPS according to spanish law. Recruitment should be concluded by the end of January 2023/beginning of February.

Lab recent selected references:

Migraine-Associated TRESK Mutations Increase Neuronal Excitability through Alternative Translation Initiation and Inhibition of TREK. Royal P, Andres-Bilbe A, Ávalos Prado P, Verkest C, Wdziekonski B, Schaub S, Baron A, Lesage F, Gasull X, Levitz J, Sandoz G. ***Neuron***. 2019 Jan 16;101(2):232-245.e6.
TRESK background K+ channel deletion selectively uncovers enhanced mechanical and cold sensitivity. Castellanos A, Pujol-Coma A, Andres-Bilbe A, Negm A, Callejo G, Soto D, Noël J, Comes N, Gasull X. ***J Physiol.*** 2020 Mar;598(5):1017-1038.
Pyrethroids inhibit K2P channels and activate sensory neurons: basis of insecticide-induced paraesthesias. Castellanos A, Andres A, Bernal L, Callejo G, Comes N, Gual A, Giblin JP, Roza C, Gasull X. ***Pain.*** 2018 Jan;159(1):92-105.
Abnormal activity of corneal cold thermoreceptors underlies the unpleasant sensations in dry eye disease. Kovács I, Luna C, Quirce S, Mizerska K, Callejo G, Riestra A, Fernández-Sánchez L, Meseguer VM, Cuenca N, Merayo-Lloves J, Acosta MC, Gasull X*, Belmonte C*, Gallar J.* ***Pain.*** 2016 Feb;157(2):399-417. *equally contributed.
Acid-sensing ion channels detect moderate acidifications to induce ocular pain. Callejo G, Castellanos A, Castany M, Gual A, Luna C, Acosta MC, Gallar J, Giblin JP, Gasull X. ***Pain.*** 2015 Mar;156(3):483-495.