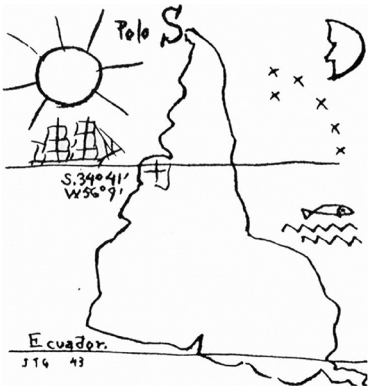
América invertida, Joaquín Torres García, 1943



FIRST MEETING
GLIA CLUB SOUTHERN CONE

The good, the bad, the nice, and the ugly of glial cells

Hybrid format
University of Buenos Aires
School of Pharmacy and Biochemistry
Buenos Aires, Argentina
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Organizing Committee

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The central nervous system (CNS) development begins soon in embryonic development and is a highly conserved process among vertebrate species. The origin of the CNS is driven by the ectodermal neurulation and posterior vesiculation stages. The neural tube closure defines a virtual symmetrical midline in its dorsal and ventral part, through which the commissural neuron growth cone axons must decide whether to cross or not, guided mostly by attraction/repulsion signals. Located at the most caudal prosomere 1, the posterior commissure (PC) is the first transversal commissure to form and defines the dorsal boundary between the diencephalic and mesencephalic vesicles. Likewise, the radial glial cells located underneath this commissure shape the first secretory structure of the brain to differentiate, the so-called subcommisural organ (SCO). The extracellular matrix components forming the path to the pioneer axons to reach the PC midline have been characterized, however, few individual genes that specifically affect the SCO proliferation and differentiation, and thus, the PC development, have been discovered. In the present work, we used PacBio and Illumina RNAseq analysis to identify a wide spectrum of SCO genes that are significantly up and downregulated in E4 compared to E7 chick embryos (HH23-HH30, respectively). Moreover, our data was corroborated through quantitative RT-PCR analysis. The data presented here provide a transcriptional panel to identify genes involved in the key proliferative (HH23) and differentiative (HH30) steps of the SCO and its related structure, the PC. We demonstrated the efficiency of this approach by correctly assembling and annotating all exons from the chicken SCO-spondin gene, including the identification of missing genes in the chicken reference annotations by homology assignments. Importantly, the presented data provide the first transcriptional landscapes of SCO in chicken development and the identification of novel key genes and long noncoding RNAs (lncRNAs) for SCO and brain development. We developed an easy-to-use genome-guided transcriptome annotation pipeline that uses assembled transcripts from hybrid sequencing data as input and distinguishes between coding and long non-coding RNAs by integration of several bioinformatic approaches.

SG7: Damaging Effects Induced by BMAA on Müller Glial Cells

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β-Methylamine-L-alanine (BMAA) is a non-proteinogenic aminoacidic cyanotoxin produced by several cyanobacteria. This cyanotoxin has been linked with the development of neurodegenerative diseases, like Amyotrophic Lateral Sclerosis, Alzheimer, and of some retinal pathologies. In the retina, it induces damaging effects on neurons and in Müller glial cells (MGCs), the major glial cell type, which have crucial roles in preserving normal retinal functionality. We have previously demonstrated that BMAA promotes neuronal degeneration with no protective effect observed by MGCs. In this work, we studied the direct effects of BMAA on MGCs in pure retinal glial cultures. For that purpose, we treated these cultures, obtained from newborn rat retinas, after cells were reseeded, with BMAA (0.4, 1, and 10 μM) at day 1 or at days 1 and 4. Cells were analyzed in either, in a short and in a long-term BMAA exposure of 3 and 9 days, respectively. We evaluated cell viability by DAPI staining and Trypan Blue assays; cellular metabolic activity by MTT assay, and cytoskeleton integrity by staining actin filaments with phalloidin. Our preliminary results showed that in both, short-term and long-term exposure of cultures to BMAA, MGCs displayed nuclear alterations without affecting the viability of these cells. Additionally, BMAA (1 and 10 μM) promoted an increase in the cellular metabolic activity in short-term, but not in long-term studies. Moreover, our results showed that BMAA induced actin network disorganization. Interestingly, in long-term exposure of cultures to BMAA, this toxin-induced an abnormal cytoplasmic growth. In conclusion, these results imply that BMAA induces several alterations in MGCs at the subcellular level without affecting their viability. Hence, these damages may help to understand neurodegenerative damages elicited by BMAA, which affect human health. Our knowledge of the molecular mechanisms involved in BMAA-induced cell damage could help to develop new therapeutic strategies.

SG8: Characterization of Cellular Inflammatory Component in a Mice Choroidal Neovascularization Model

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Age-related macular degeneration (AMD) in its choroidal neovascularization (CNV) stage, where growing neovessels invade the retina inducing photoreceptor degeneration, is the leading cause of vision loss among adults. Mononuclear phagocytic cells (MPCs), such as resident microglia and monocyte-derived macrophages, collaborate in establish a