

XXVI Congreso Anual de la Sociedad Argentina de Investigación en Neurociencia

> Huerta Grande, Córdoba 18-22 Octubre 2011.

## SAN2011 | Organizing Committee

Guillermo Lanuza, Instituto Leloir - Buenos Aires Pablo López, Instituto Mercedes y Martín Ferreyra - Córdoba María Eugenia Pedreira, Facultad de Ciencias Exactas y Naturales Universidad de Buenos Aires - Buenos Aires Estela Muñoz, Universidad Nacional de Cuyo - Mendoza Cecilia Inés Calero, Facultad de Ciencias Exactas y Naturales, Universidad de Buenos Aires - Buenos Aires Franco Mir, Instituto Mercedes y Martín Ferreyra - Córdoba Ricardo Pautassi, Facultad de Psicología, Universidad Nacional de Córdoba - Córdoba

## Course | Organizing Committee

**Germán Szapiro**, *Neuroscience Section*, *Ecole Normale Supérieure*, *Paris*, *France*. **Juan Goutman**, *INGEBI-CONICET*, *Buenos Aires*, *Argentina*.

Logistic Organization: Silvina Andrea Ceriani

outposts into dendrites. In addition, using a plasma membrane (PM) protein (e.g.transferrin receptor [TfR] fused to GFP) engineered with reversible/removable aggregation domains we observed that suppression or expression of DN-BARS delay the exit of TfR from the Golgi apparatus. Taken together, these data provide the first set of evidence suggesting a role for BARS in neuronal polarization by regulating membrane trafficking and organelle positioning.

Supported by ANPCyT y Agencia Córdoba Ciencia.

## Cellular and Molecular Neurobiology Poster Number 37 | Session 1

## "Activation of a retinoid orphan receptor is required for docosahexaenoic acid protection of photoreceptors"

Olga Lorena German, Sandra Mónaco, Daniela Agnolazza, Nora Patricia Rotstein, Luis Enrique Politi

Instituto de Investigaciones Bioquímicas de Bahía Blanca (INIBIBB), Universidad Nacional del Sur (UNS)-CONICET. 8000 Bahía Blanca, Argentina.

olgerman@criba.edu.ar

Docosahexaenoic acid (DHA), the major omega-3 polyunsaturated fatty acid in the retinal promotes the survival of rat retina photoreceptors (PR) during early development in vitro and upon oxidative stress by activating the ERK/MAPK signaling pathway. We investigated if DHA activates this pathway by direct activation of tyrosine kinase receptors (TRK) or of retinoid nuclear receptors (RXR). Using retinal neuronal cultures we determined that DHA prevented PR apoptosis at early culture times in spite of the presence of a TRK inhibitor (K252a), implying TRK are not involved in its effects. On the contrary, RXR antagonists (HX531 or PA452) inhibited DHA protection during early development in vitro and upon paraguat and H202-induced apoptosis. Moreover, RXR agonists (HX630 or PA024) decreased ROS production in H2O2-treated neuronal cultures, as we previously showed for DHA. To evaluate whether DHA has to be released from phospholipids to exert its protective effect, DHA-supplemented cultures were treated with a phospholipase A2 inhibitor (BEL) prior to H2O2 treatment; BEL addition blocked DHA protection on PR upon oxidative stress. These results suggest a new pathway for DHA actions in PR: it is first released from phospholipids and then activates RXR to promote PR survival