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the chicken inner retina (Verra et al. 2011). Although Opn4 has been extensively characterized, there is still an open question regarding the mechanisms involved in its chromophore regeneration. To this end, we have applied in vivo and ex vivo strategies to investigate retinoids in the chicken inner retina. In isolated retinal layers from chickens exposed to light or maintained in the dark we found that levels of 11 cis-retinal (11cRal) decreased in the photoreceptor cell layer after light exposure, however 11cRal levels were maintained constant or elevated in the retinal ganglion cell (RGC) layer under the same light condition. Besides, cultures of immunopurifided RGCs supplied with all trans-retinal (atRal) displayed the capacity to isomerize atRal into 11cRal after light stimulation. These results strongly support the idea of a novel light-dependent mechanism of chromophore regeneration in the chicken inner retina.

NS-P03
BIOLOGICAL EFFECTS OF GDNF/GFRα1 ON NEURAL CORTICAL PROGENITORS.

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Neurons generation from proliferating progenitor cells is a complex process involving an interplay between intrinsic cellular programs and extrinsic cues such as growth factors. One factor that might play a role in regulating progenitor cell biology is the neurotrophic factor GDNF. Reverse transcription analysis shows that GDNF receptor, GFR1, is expressed at early developmental stages in the forebrain suggesting that GDNF/GFRα1 might play a role in cortical precursor development. Here we show that cultured cortical progenitors maintained in proliferating conditions show lower levels of GFRα1 expression than those maintained in differentiating conditions. Furthermore, cell precursors growth in presence of GDNF result in a significant increase in the morphological complexity of the differentiated neurons. Notably, Ki67 positive proliferative progenitors decreased significantly upon GDNF treatment. Addition of GDNF to proliferating progenitors forming neurospheres resulted in the downregulation of cyclin D and E, required for cell cycle progression, and in the upregulation of p21, a potent cyclin-dependent kinase inhibitor, showing that GDNF induces an arrest of cell cycle of cortical precursors. Thus, our results indicate that GDNF/GFRα1 signaling may play an essential role controlling the transition of neuronal progenitors from a proliferative condition towards neuronal differentiation.

NS-P04
ANANDAMIDE HYDROLYSIS IS MODULATED BY CANNABINOID RECEPTORS IN AGED RAT CEREBRAL CORTEX.

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Fatty acid amide hydrolase (FAAH) has been reported as the main enzyme involved in anandamide (AEA) hydrolysis. Among the multiple functions of AEA we can highlight the regulation of synaptic plasticity and its antiinflammatory role. The aim of this study was to analyze AEA hydrolysis in cerebral cortex (CC) subcellular fractions during physiological aging and its regulation by cannabinoid receptors (CBR). CC membrane and synaptosomal fractions from adult (3 mo) and aged (28 mo) rats were isolated by differential centrifugation and the synaptosomal fraction was purified in ficoll gradients. AEA hydrolysis was assayed using [3H]AEA and its product was quantified from the aqueous phase. Aging differently modulated FAAH by increasing and decreasing its activity in membranes and synaptosomes, respectively. In the presence of FAAH specific inhibitor URB-597 AEA hydrolysis activity assay corroborates that AEA is the main enzyme involved in CC AEA degradation. CBR agonists decreased FAAH activity, mainly by CB2R, thus increasing CC AEA availability. Our results show that while aged CC membrane AEA availability decreases, possibly compromising its antiinflammatory functions, the endocannaboid level in synaptosomes increases, protecting against the synaptic dysfunction inflicted by aging. AEA availability could be increased by targeting CB2R, thus improving brain damage caused by aging.

NS-P05
DISSECTING THE TRANSCRIPTIONAL CODE OF THE DOPAMINE D2 RECEPTOR GENE.

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The dopamine D2 receptor (D2R) plays a major role in the central control of locomotor, appetitive, emotional and cognitive functions. Despite its significance, the molecular mechanisms that control the expression of the D2R gene