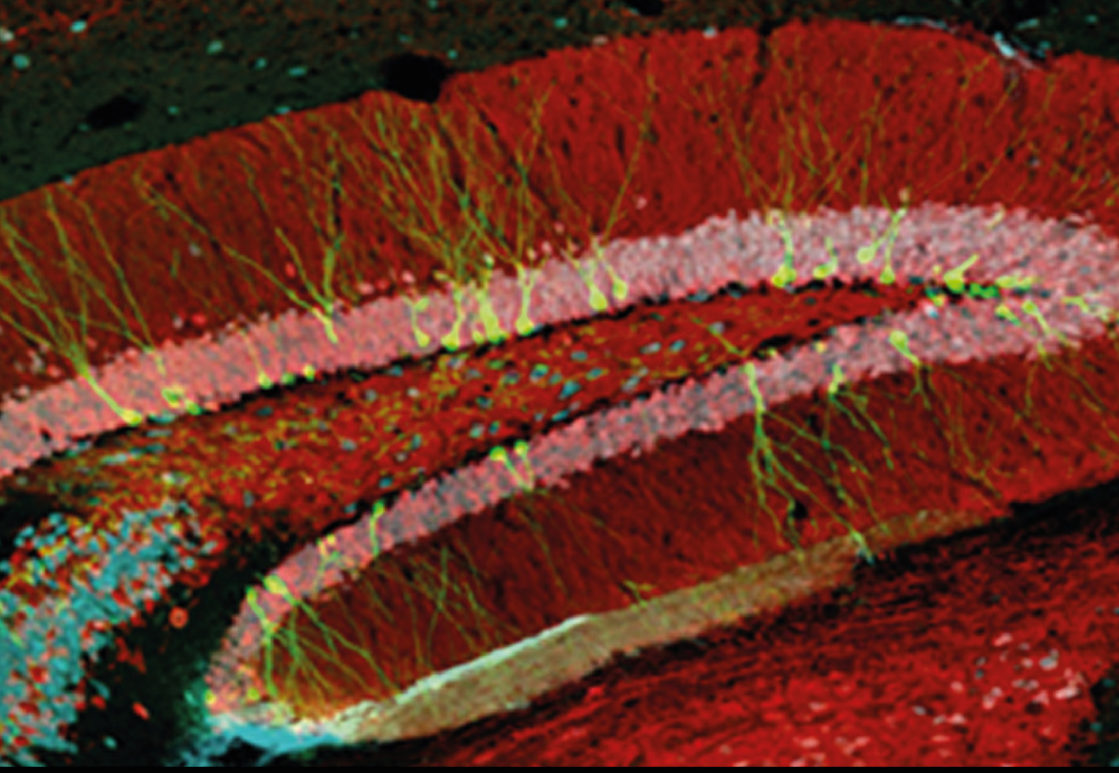

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SOCIEDAD ARGENTINA DE INVESTIGACIÓN EN NEUROCIENCIAS

XXIX ANNUAL MEETING
AND SAN-ISN SMALL CONFERENCE AND COURSE

"New mechanisms of neuro-glial interaction: Their contribution to nervous system development and repair"

September 29 | October 3, 2014
Huerta Grande, Córdoba, Argentina

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P137.-ATP and adenosine modulate acetylcholine release through P2Y and P1 receptors at the efferent-inner hair cell synapse in the developing inner ear

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Before the onset of hearing (postnatal day 12 in mice) inner hair cells (IHCs) are transiently innervated by medial olivocochlear (MOC) efferent fibers. Acetylcholine (ACh) released by these fibers activates $\alpha9\alpha10$ nicotinic receptors coupled to SK2 calcium-activated potassium channels, leading to inhibitory post synaptic currents (IPSCs). During this period, IHCs fire spontaneous sensory-independent action potentials that are required for normal development of the auditory pathway. This activity is driven and/or modulated by ATP released from cochlear supporting cells. ACh release from efferent fibers also contributes to this modulation. By electrically stimulating MOC fibers and recording IPSCs, we showed that ATP decreases ACh release in a reversible and concentration-dependent manner. In this work, we demonstrate that this effect is through P2Y receptor activation, as the specific P2Y agonist 2-MeSADP mimicked the effect of ATP. Moreover, the non-hydrolyzable ATP analog ATPγS decreased ACh release as well, indicating that this modulation is driven by ATP itself. We further tested if adenosine can also modulate ACh release. Adenosine reversibly decreased the quantal content. CGS15943, a specific P1 receptor antagonist, abolished the effect of adenosine. On the other hand NECA, a specific P1 agonist, decreased ACh release. Our results suggest that both ATP and adenosine inhibit ACh release at the MOC-IHC synapse through the activation of
P2Y and P1 receptors, respectively.