9e Colloque des Neurosciences - Bordeaux May 26-29, 2009

For posters, authors are expected to be in attendance at the time indicated.
For other sessions, time indicates the beginning of the sessions.

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Poster board 93 - Wed 27/05/2009, 14:00 - Espace Pluriel
Session 009 - Cellular and molecular neurobiology 1
Abstract n° 009.62
Publication ref.: *9e Colloque de la Société des Neurosciences, 009.62 ,2009*

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Title Role of ASIC3 in acidic and inflammatory pain.

Text Acid-sensing ion channels (ASICs) are cationic channels activated by extracellular acidosis that are expressed in both central and peripheral nervous systems. Although peripheral ASICs seem to be natural sensors of acidic pain (e. g., in inflammation, ischaemia, lesions or tumours), a direct demonstration was still lacking. Here, we show that about 60% of rat cutaneous sensory neurons express ASIC1a- and/or ASIC3-type currents. Nociceptors are sensitive to moderate acidification (in the activation range of ASIC1a and ASIC3) and this activity is inhibited by the toxin APETx2, a specific blocker of ASIC3 extracted from a sea anemone. Native ASIC currents, recorded from cutaneous sensory neurons, as well as recombinant ASIC3 current, recorded from F11 transfected cells, respond synergistically to three different inflammatory signals that are slight acidifications (around pH 7.0), hypertonicity and arachidonic acid (AA). ASIC3 thus appears as a molecular integrator of different inflammatory factors since, moderate pH, alone or in combination with hypertonicity and AA, increases nociceptors excitability and produces pain suppressed by the toxin APETx2. Furthermore, both APETx2 and the in vivo knockdown of ASIC3 with a specific siRNA have potent analgesic effects against primary inflammation-induced hyperalgesia in rat. Peripheral ASIC3 channels are thus essential sensors of acidic pain and integrators of molecular signals produced during inflammation where they contribute to primary hyperalgesia.

Theme Cellular and molecular neurobiology