Retinoic acid

Retinoic acid (RA) signaling plays a key role in the development and function of several systems in mammals.

Retinoic acid (RA) and nerve growth factor (betaNGF) were found to be potent enhancers of neuronal differentiation, eliciting extensive outgrowth of processes and the expression of neuron-specific molecules.

Retinoic acid serves as a modulator of neurofilament expression in this in vitro model of nerve regeneration [1].

Retinoic acid (RA), a developmental morphogen, has emerged in recent studies as a novel synaptic signaling molecule that acts in mature hippocampal neurons to modulate excitatory and inhibitory synaptic transmission in the context of homeostatic synaptic plasticity. However, it is unclear whether RA is capable of modulating neural circuits outside of the hippocampus, and if so, whether the mode of RA's action at synapses is similar to that within the hippocampal network.

Yee and Chen explore for the first time RA's synaptic function outside the hippocampus and uncover a novel function of all-trans retinoic acid at inhibitory synapses. Acute RA treatment increases spontaneous inhibitory synaptic transmission in L2/3 pyramidal neurons of the somatosensory cortex, and this effect requires expression of RA's receptor RARα both pre- and post-synaptically. Intriguingly, RA does not seem to affect evoked inhibitory transmission assayed with either extracellular stimulation or direct activation of action potentials in presynaptic interneurons of connected pairs of interneuron and pyramidal neurons. Taken together, these results suggest that RA's action at synapses is not monotonous, but is diverse depending on the type of synaptic connection (excitatory versus inhibitory) and circuit (hippocampal versus cortical). Thus, synaptic signaling of RA may mediate multi-faceted regulation of synaptic plasticity [2].

Carbenoxolone, quinine, mefloquine, quinidine, anandamide, oleamide, heptanol, octanol, meclofenamic acid, niflumic acid, flufenamic acid, glycyrrhetinic acid and retinoic acid have all been evaluated on animal seizure models.

There is a possible relationship of the myelocystocele to teratogens such as loperamide HCl and retinoic acid, although the exact etiology of this entity is not known [3].

NTera-2 (NT2) cells are a human embryonal carcinoma (EC) cell line derived from a teratocarcinoma that differentiate exclusively into postmitotic neurons in vitro following retinoic acid (RA) treatment.

1)
Hall CM, Else C, Schechter N. Neuronal intermediate filament expression during neurite outgrowth from explanted goldfish retina: effect of retinoic acid. J Neurochem. 1990 Nov;55(5):1671-82. PubMed PMID: 2213018.
2)

Yee A, Chen L. Differential regulation of spontaneous and evoked inhibitory synaptic transmission in somatosensory cortex by retinoic acid. Synapse. 2016 Jun 27. doi: 10.1002/syn.21921. [Epub ahead of print] PubMed PMID: 27348405.

3)

Peacock WJ, Murovic JA. Magnetic resonance imaging in myelocystoceles. Report of two cases. J Neurosurg. 1989 May;70(5):804-7. PubMed PMID: 2709123.