responses. PFC dysfunction occur in depression, and serotonin, a neurotransmitter known to be implicated in depression, is able to modulate PFC function. In this area, three important serotonin receptors are expressed, both in glutamatergic and GABAergic neurons: $5HT_{1A}$, $5HT_{2A}$ and $5HT_3$. Besides plasticity being an important factor in depression physiopathology, it is not yet clear how serotonin can modulate plasticity on key brain areas involved with emotion expression and stress responses. Thus, we investigate if serotonin and its 1A receptor expressed in the PFC was able to evoke plastic responses. Using brain slice electrophysiology, we patched PFC layer V pyramidal neurons from rats at 14 and 25 days of age, applying stimulus on the layer I to induce depolarizations. Applying serotonin in these conditions induced a marked LTD in both ages, while a $5HT_{1A}$ agonist induced a less pronounced LTD in 25 days-old animals, in both control and depressed animals. However, coupling *theta burst* stimuli with serotonin application caused LTP expression, and this effect was only seen with 14 days-old animals, suggesting an important role of serotonin on plasticity during neurodevelopment. This effect was absent in depressed animals, indicating differences in serotonergic modulation of the PFC in depression. In conclusion, our data indicates serotonin mostly induce LTD on PFC layer V neurons, this effect is mediated at least in part by $5HT_{1A}$ receptors, and depression can be a result of altered serotonergic modulation in the PFC during development, while the $5HT_{1A}$ receptors do not seem to have a role on this process in depression. Therefore, the other two serotonergic receptors expressed in the PFC must be involved in this changes in serotonergic modulation that occur in depressed animals.

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P16.22

Chemical LTD, but not LTP, induces transient accumulation of gelsolin in dendritic spines

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Both strengthening and weakening of synapses is mediated by Ca^{2+} signaling, but it is still unclear how one signal molecule can trigger two opposite outcomes. Identifying molecules which can distinguish between signaling leading to weakening or strengthening, can improve our understanding of how synaptic plasticity is regulated. We tested gelsolin's response to the induction of chemical long-term potentiation (cLTP) or long-term depression (cLTD) in cultured rat hippocampal neurons at DIV21. We saw that gelsolin relocates from the dendritic shaft to dendritic spines upon cLTD induction while it did not show any relocalization upon cLTP induction. Dendritic spines are small actin-rich protrusions on dendrites, where LTD/LTP-responsive excitatory synapses are located. We propose that the LTD-induced modest but relatively long-lasting elevation of Ca^{2+} concentration increases the affinity of gelsolin to F-actin. As F-actin is enriched in dendritic spines, it is probable that increased affinity to F-actin induces the relocalization of gelsolin.

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Changes in corticomotor excitability of the calf muscles during postural tasks

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It was shown that the corticospinal pathway is the one of important element of human standing feedback control. The excitability this system is flexible during the upright stance. However, it has been unclear how the body position and surface parameters can modulate corticospinal excitability.

Twelve relevantly healthy individuals (age: 23.2 ± 2 years, all female) were assessed using single-pulse transcranial magnetic stimulation of the motor cortex. The motor-evoked amplitude was registrated via surface electromyography. We investigated the corticospinal excitability of the soleus (SOL) and tibialis anterior (TA) muscles which were modulated under three experimental conditions: sitting position, standing on a hard floor surface and standing over foam surface. TA and SOL MEP amplitudes were compared during: (1) sitting versus standing on a hard floor surface; (2) standing on a hard floor surface versus standing over foam surface.

The TA and SOL MEP amplitudes were significantly lower during sitting versus standing ($p < 0.05$). However, no significant difference in the TA- and SOL-MEPs amplitude was observed for standing on a hard floor surface versus standing over foam surface ($p < 0.05$).

These results can provide a part of essential information concerning corticomotor excitability and postural control. Supported by (RFB NO. 18-315-00263).

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Cholinergic modulation of the intrinsic properties of subicular neuron via direct suppression of HCN channel

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Cholinergic inputs to the hippocampus play a pivotal role in maintaining theta oscillations, which is correlated with different behavioral states. Subiculum also receives septal cholinergic inputs and has a role in cognition like spatial navigation and movement. Subiculum forms the main output region of the hippocampus and any change in the intrinsic properties of subicular neurons can change the hippocampal output pattern. The synchronous firing of neurons in theta frequency is tuned by the intrinsic resonance of the neurons which is profoundly mediated by I_h.

Our aim was to study the effect of the exogenous/endogenous cholinergic agent on the intrinsic properties of subiculum neurons which contribute to the local theta rhythms. To isolate the effect of a cholinergic agent apart from changes due to binding to its receptors (nAChR and mAChR) we used nicotine in the presence of nAChR blockers.

Biocytin staining and patch clamp technique on hippocampal rat brain slices was used to validate the morphology and intrinsic properties of the neurons. On bath application of nicotine, we observed decrease in sag amplitude and resonance frequency indicating the modulation of I_h. These effects were persistent in the presence of non-specific nAChR blocker, indicating the changes are not mediated via AChRs. Nicotine treatment also modulated the