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Research report

The serotonin reuptake inhibitor citalopram suppresses activity in the neonatal rat barrel cortex *in vivo*

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ABSTRACT

Inhibition of serotonin uptake, which causes an increase in extracellular serotonin levels, disrupts the development of thalamocortical barrel maps in neonatal rodents. Previous *in vitro* studies have suggested that the disruptive effect of excessive serotonin on barrel map formation involves a depression at thalamocortical synapses. However, the effects of serotonin uptake inhibitors on the early thalamocortical activity patterns in the developing barrel cortex *in vivo* remain largely unknown. Here, using extracellular recordings of the local field potentials and multiple unit activity (MUA) we explored the effects of the selective serotonin reuptake inhibitor (SSRI) citalopram (10–20 mg/kg, intraperitoneally) on sensory evoked activity in the barrel cortex of neonatal (postnatal days P2-5) rats *in vivo*. We show that administration of citalopram suppresses the amplitude and prolongs the delay of the sensory evoked potentials, reduces the power and frequency of the early gamma oscillations, and suppresses sensory evoked and spontaneous neuronal firing. In the adolescent P21-29 animals, citalopram affected neither sensory evoked nor spontaneous activity in barrel cortex. We suggest that suppression of the early thalamocortical activity patterns contributes to the disruption of the barrel map development caused by SSRIs and other conditions elevating extracellular serotonin levels.

1. Introduction

The primary somatosensory cortex in rodents contains a somatotopic map where each whisker is represented in a discrete cytoarchitectural L4 unit, the "barrel" (Woolsey and Van der Loos, 1970; Petersen, 2007; Fox, 2008). During development, the barrel map forms in the first postnatal week, which is also the critical period for barrel map plasticity (for reviews, (O'Leary et al., 1994; Erzurumlu and Kind, 2001; Lopez-Bendito and Molnar, 2003; Feldman and Brecht, 2005; Fox, 2008; Feldman, 2009; Erzurumlu and Gaspar, 2012). During this time, the barrel cortex displays enhanced plasticity at thalamocortical synapses (Isaac et al., 1997; Feldman et al., 1998) and unique spontaneous and sensory-driven activity patterns that are thought to participate in the activity-dependent formation of the topographic thalamocortical barrel maps (Minlebaev et al., 2007, 2009; Yang et al., 2009; Minlebaev et al., 2011; Yang et al., 2013). Among the various signalling mechanisms which are involved in the development of the barrel map, serotonin and glutamate signalling were found to be critical in barrel map formation. An increase in extracellular serotonin levels through a pharmacological blockade of serotonin transporters during the critical period using selective serotonin uptake inhibitors (SSRIs) or genetic deletion of serotonin transporters, as well as through genetic deletion of monoamine oxidase A (MAOA) prevents formation of the whisker-related barrel patterns (Cases et al., 1996; Persico et al., 2001; Salichon et al., 2001; Toda et al., 2013; for review, see van Kleef et al., 2012). Defective barrel phenotypes and/or alteration in the functional organization of cortical columns have also been observed after pharmacological blockade of glutamatergic signalling in the barrel cortex (Fox et al., 1996; Mitrovic et al., 1996) and in mutant mice with genetic blockade of cortical NMDA receptors (Iwasato et al., 2000; Lee et al., 2005) and the metabotropic glutamatergic pathway including the mGluR5

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