ABSTRACT

The *Dexras1* gene was identified after induction by glucocorticoid dexamethasone in pituitary tumor cells. *Dexras1* has also been found in other brain regions and in the peripheral organs but its expression is rhythmic only in the suprachiasmatic nuclei of the hypothalamus (SCN), where the mammalian main circadian pacemaker is located. *Dexras1 e*xpression was also affected by stress, amphetamine or prenatal alcohol exposure. Its role in cells has not yet been explained. Dexras1 GTPase activity has been determined to be dependent on the NMDA receptor stimulation. Dexras1 acts as an activator of G protein signaling in cells. Its role has been detected in neuronal iron homeostasis or in the regulation of main circadian pacemaker sensitivity to photic and nonphotic synchronization cues during the day. The aim of our study was to describe the *Dexras1* mRNA expression in the rat brain during ontogeny and during development after visual sensory deprivation by *in situ* hybridization. The earliest *Dexras1* expression was detected on embryonic day 20, in the rat SCN and the ventral posteromedial thalamic nucleus. Postnatally, its expression also appeared in other sensory areas, motor thalamic areas, hypothalamic areas involved in the regulation of water homeostasis, or in limbic system. Our results further show that the sensory deprivation suppress *Dexras1* expression in several brain areas involved in visual processing and enhance its expression in olfactory structures. It suggests that Dexras1 could play the role in the process by which the neuronal system compensates the deprived sensoric system by strengthening of the other.

Key words: Dexras1, G protein, brain, ontogeny, circadian system, visual sensory deprivation