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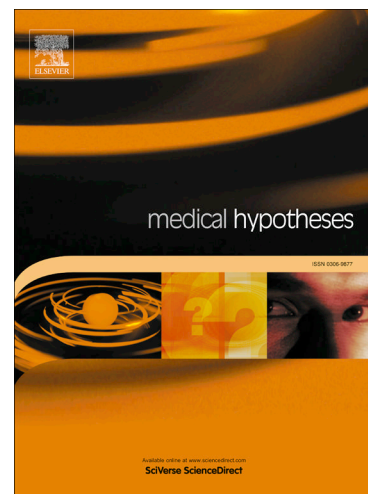
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Cognitive disorders in allergic rhinitis may be induced by decline of respiration entrained rhythm in the brain

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Allergic rhinitis (AR) is a prevalent chronic inflammatory disease of the upper respiratory tract, which is characterized by some nasal symptoms, including itching, nasal blockage, sneezing, and rhinorrhea (1-3). In addition to such classical symptoms, dysfunction of some cognitive behavior for instance concentration, memory, and learning ability lead to reduce quality of life in these patients (2, 3). Although cognitive disorders may be caused by different reasons such as sleep disorders and effects of inflammatory mediators on central nervous system (4-6), the underlying mechanisms of cognitive disorders in these patients remain poorly understood.

As revealed by odor detection ability test and analyzing local field potentials of olfactory bulb, olfactory dysfunction was observed in the animal AR model (7). Ozaki et al. exhibited that a possible cause of impaired olfactory function in AR mice is increased the number and size of glands, as well as cytotoxicity and neurotoxicity of infiltrated eosinophils and macrophages in olfactory mucosa (7). In addition, it has been reported that there is a reduction in the volume and function of olfactory bulb in patients with AR (8). Taken together, these results indicate that olfactory bulb may be structurally and functionally impaired in AR patients.

Mammalian olfactory sensory neurons (OSNs) not only can detect chemical stimuli, but also mechanically receive nasal air flow information, with a shared second-messenger cascade (9). Several studies have started to demonstrate respiration drive neural oscillations in different brain areas. Fontanini and Bower for the first time indicated that slow oscillations in neocortex are in synchrony with nasal breathing (10). During nasal breathing, nasal airflow entrains rhythmic neural activity in olfactory bulb by activating mechanosensitive OSNs in the nasal epithelium. Olfactory bulb activity paces respiration-entrained slow oscillations in neocortical and subcortical regions such as piriform cortex, prefrontal cortex, amygdala and hippocampus (11, 12).

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