LETTER TO THE EDITOR

Reply: Increased olfactory bulb volume due to treatment of chronic rhinosinusitis: neuroinflammation and adult neurogenesis

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Sir, Dr Jian Li and Dr Ti-Fei Yuan emphasize in their Letter to the Editor two additional points that could contribute to the measured increase of olfactory bulb volume following treatment of chronic rhinosinusitis (Gudziol et al., 2009). Treatment of chronic rhinosinusitis would decrease nasal inflammation, possibly also resulting in a decrease of inflammatory mediators that may impact on cell proliferation in the subventricular zone and on cell survival in the olfactory bulb. Additionally, the post-operatively administered retinol could contribute to neurogenesis in the brain. Therefore, both decreased nasal inflammation and retinol could impact on changes of the volume of the olfactory bulb.

We would like to thank both authors for their thoughtful comments. However, we think that it is not the main cause of the observed increase of olfactory bulb volume following treatment of chronic rhinosinusitis. It would also imply that the smaller olfactory bulbs at baseline in the patient group compared with healthy subjects are a result of increased nasal inflammation. Olfactory bulb volume is also reduced in non-inflammatory causes of smell loss, indicating that inflammation is not the sole cause of the reduced olfactory bulb volumes (Yousem et al., 1999; Mueller et al., 2005b; Rombaux et al., 2006). Additionally, no major difference in olfactory bulb size was found in hyposmic patients with idiopathic Parkinson’s disease compared with normosmic controls (Mueller et al., 2005a). In contrast with the patients with smell loss due to sinunasal disease or head trauma, the peripheral olfactory system in patients with Parkinson’s disease does not seem to be disrupted (Witt et al., 2009; Yee et al., 2009). Further longitudinal studies in patients with non-inflammatory smell loss are needed to confirm these ideas.

During post-operative care, patients self-administered Retinol containing oil to the nasal cavity. Although the effect of topically applied retinoid acid on olfactory function has not been shown yet in humans, retinoic acid has been reported to improve recovery of olfactory receptor neurons in animals (Yee and Rawson, 2000). It appears unlikely that large quantities of retinoic acid diffused via the olfactory nerves into the central nervous system and impacted on central nervous system neurogenesis in the presented study. Oil containing retinoic acid administrated by the used device stays largely in the inferior nasal meatus (Scheibe et al., 2008), where rarely olfactory epithelium is found (Leopold et al., 2000). In case nasally applied retinoic acid should be absorbed systemically, an impact on central nervous system neurogenesis could be possible. An argument against this are the negative findings of a double-blind study where oral application of retinoic acid could not be demonstrated to improve olfactory function in patients with smell loss (Lill et al., 2006). Although an influence of retinoic acid on the change of olfactory bulb volume cannot be entirely excluded, in the presently investigated paradigm, it seems that a bottom-up effect is mostly responsible for an increase of olfactory bulb volume. In other words, the observed increase of the olfactory bulb volume three months post-operatively appears to be largely due to increased peripheral olfactory function.

References


