Odorant Mediated vs. Sniffing Mediated BOLD Activation in Human Primary Olfactory Cortex

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INTRODUCTION

The odor perception is intimately coupled with sniffing, and both functions are processed in the primary olfactory cortex (POC). A variety of studies on mammalian olfactory systems have shown the spatial distribution of the olfactory components in the olfactory system. However, it is difficult to differentiate odorant perception from sniffing in human primary olfactory cortex using fMRI. In this study, we demonstrated that the BOLD signals in the POC from odorant stimulation can be separated and quantified from those by sniffing, which opens up opportunities for investigating mechanisms of olfactory deficits in several neurodegenerative diseases such as Parkinson's disease.

METHODS

The sniffing-odor stimulation paradigm contained 6 s of visual prompted sniffing of either odorized air (0.10% of lavender oil in 1,2-propanediol) or odorless air, with each condition repeated twelve times and interleaved with 22-38 s odorless air at a constant air flow of 6 L/min. The intervals between sniffings were pseudo-randomized to reduce anticipatory effect. Twenty-seven healthy volunteers (27.7±5.8 years, 11 male, 2 left-handed) with normal olfactory functions (UPSIT score 36.5±1.7) completed the fMRI study. The fMRI data were collected on a 3 T system with an 8-channel head coil and a T2*-weighted EPI sequence. The subjects were instructed to sniff whenever they saw the word 'SNIFF' on the screen, but not required to provide any response during the execution of the scan protocol. The subjects' sniffing and respiration patterns were monitored via a pneumatic respiration sensor and recorded together with the odor delivery and imaging data.

These data sets were processed with gMRI (http://www.pennstatehershey.org/web/nmrlab/resources/software/gmri) for the stimulation onset vectors and sniffing volume calculation. The fMRI data were processed with SPM5 (University College London, UK) following the standard procedures. Statistical parametric maps were generated separately for the sniffing events with odorant or odorless air at the individual level by fitting the stimulation paradigm to the functional data with a default 128sec high-pass filter, convolved with the canonical hemodynamic response function (FWE corrected, p < .05, extent threshold = 10). Activation maps at the group level were generated for each stimulation condition (one-sample t-test, FWE corrected, p < .01, extent threshold = 10). The difference between the sniffing functions responding to odorant or odorless air was generated at the individual level and at the group level (paired t-test, uncorrected, p < .005, extent threshold = 10). Major activation clusters from the group level analysis were saved as region-of-interests (ROI) to measure the BOLD signals at the individual level using MarsBaR. The BOLD signals responding to the two stimulation conditions were compared at the group level.

RESULTS

The sniffing function with or without concurrent odorant administration triggered significant activation in the bilateral POC and secondary olfactory structures, e.g., insular cortex, prefrontal cortex, anterior and posterior cingulate cortex, striatum, thalamus, cerebellum, and other areas in temporal and occipital cortices. In general, sniffing odorant triggered the same anatomical structures to function as sniffing odorless air. While there was no significant difference in sniff volume between odorant sniffing and odorless sniffing at either individual level or at group level (paired t-test, p > .10), significantly stronger activation was observed in the bilateral POC during odorant sniffing (Fig. 1). In addition, responding to the odorant sniffing, the BOLD signals in the bilateral POC were significantly stronger than those triggered by odorless sniffing (paired t-test, p < .01) (Fig. 2).

DISCUSSION

Our results showed that the sniffing of odorant triggered significantly stronger activation in the POC than the sniffing of odorless air. This is consistent with the result from a previous PET study [1]. The difference of the activation in the POC is due to neural activities associated with odor perception, i.e., short term odorant memory or other odorant information processing in the POC, apart from the sniffing response and the somatosensory stimulation by the air flow [2]. Thus, when the sniffing function is carefully monitored, our simple sniffing task can be used for the studies of Parkinson's disease involving both olfactory and motor functional changes.

Fig. 1. Odorant triggered activation in the bilateral POC (paired t-test, uncorrected, p < .005, extent threshold = 10).

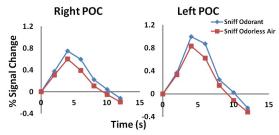


Fig. 2. The BOLD signal in the bilateral POC responding to the sniffing of odorized or odorless air.

References:

- 1. Kareken, D.A., et al. Neuroimage, 2004. 22(1): p. 456-65.
- 2. Sobel, N., et al. Nature, 1998. 392(6673): p. 282-6.

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