The Balance Between Top-Down and Bottom-Up Attention in Misophonia

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Misophonia is a disorder of abnormal emotional reactions to specific sensory stimuli, and in particular auditory stimuli. While mechanisms involving differential reactivity of emotional regulation, learning and auditory processing have been proposed, thus far little is known or understood about the neural basis of misophonia.

Auditory sensitivities are also commonly associated with autism spectrum disorder (ASD). We have previously shown that abnormal sensory processing in ASD has been is linked to abnormally increased bottom-up cortical functional connectivity. At the same time, there is evidence that top-down processes that would typically down-regulate cortical responses to stimuli are reduced in ASD.

Since abnormal sensory reactivity in misophonia is stimuli-specific, rather than generalized, as is the case most often in ASD, we hypothesize that participants with misophonia will show increased top-down functional connectivity from cortical areas that modulate attention into the auditory cortex. We further hypothesize that unlike ASD participants, they will only show this increased functional connectivity during aversive triggering auditory stimuli, indicating that misophonia is top-down regulated.

To test this hypothesis, we are using magnetoencephalography (MEG) to measure bottom-up and top-down functional connectivity during an auditory spatial attention task. Participants are instructed to perform a classic auditory oddball paradigm while attending to only to the cued ear. Attention to the deviant tone in the attended ear is driven by “top-down” attention, which is voluntary and spatially specific. Participants are also instructed to ignore sounds in the non-cued ear, which will involve novel stimuli such as claps or clinks, as well as sounds that are specially selected to trigger misophonia in each individual participant, such as chewing. While all distractor sounds will elicit involuntary “bottom-up” attention, we expect that in misophonia participants, the misophonia-specific sounds will also trigger greater than expected top-down processes.

Data collection is ongoing. Preliminary analyses of our first two participants show that in comparison to the neurotypical participant, the participant with misophonia shows larger differences between evoked responses to misophonic triggers versus non-misophonic triggers. Further analyses with additional participants will examine functional connectivity to test our hypotheses.

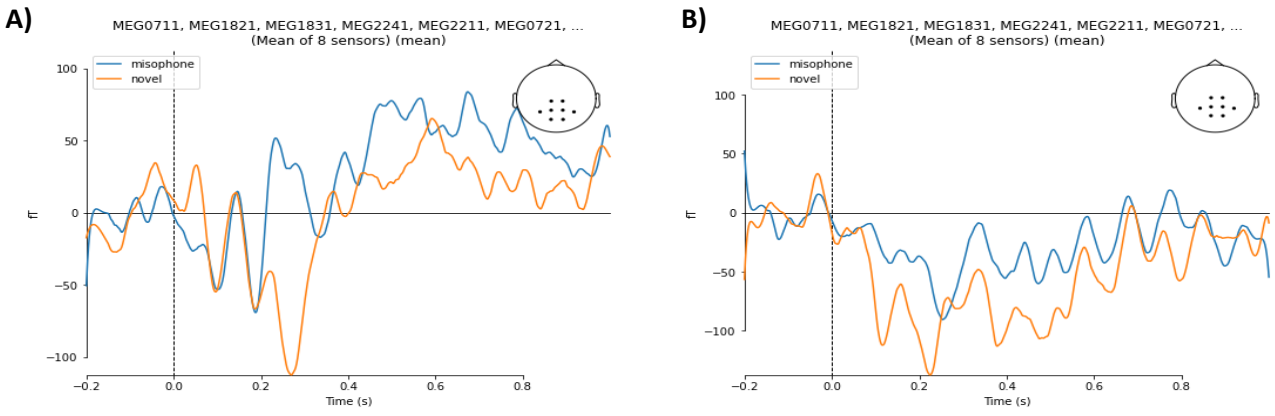

Figure 1 Example evoked responses from central sensors from A) a misophonic participant and B) a neurotypical participant. Between 200ms to 400ms after the sound is presented, the misophonic participant shows a positive peak in the central area to the misophonia-triggering sound but a negative peak to the novel, non-triggering sound. The neurotypical participant shows a similar response to both the misophonia-triggering sound and the non-triggering sound.