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Relationship Between Caudal and Rostral Auditory Efferent Pathways: A Preliminary Investigation

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Abstract

Perception of sounds involves excitatory as well as inhibitory activities. Inhibition occurs throughout the auditory system, from the auditory cortex to the cochlea, and is predominantly mediated by the auditory efferent system. In the present study, we assessed the interactions between two measures of inhibition in neurotypical adults—contralateral inhibition of otoacoustic emissions, which is a subcortical measure, and sensory gating, a cortical measure. We found an inverse relationship between these two functions. The possible reasons for this are discussed with an implication to the auditory efferent system.

Keywords: auditory efferent system; auditory sensory gating; contralateral inhibition of otoacoustic emissions; inhibition

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Introduction

Speech perception often occurs in dynamic and complex acoustic environment. Accurate perception in complex acoustic environment entails the auditory system to extract important cues while ignoring the irrelevant ones. This necessitates an interplay of both afferent and efferent auditory systems. While the afferent auditory system processes and conducts sound from cochlea to cortex, it is proposed that the efferent auditory system modulates the bottom-up afferent processing (Hackney, 1987; Suga et al., 2000). Though the human auditory system has a rich and elaborate descending neural network, the interplay amongst these pathways and its contribution to hearing remains unclear.

Neuroanatomical evidence demonstrates the presence of four main types of descending auditory networks—the corticocortical, corticothalamic, corticocollicular, and the olivocochlear modulations (Delano & Elgoyhen, 2016; Suga, 2020). The cortico-cortical network connects the auditory cortex with other areas of the cerebral cortex, including the prefrontal cortex (Suga, 2020). The corticothalamic network extends from the auditory cortical structures to the medial geniculate body (Tang et al., 2012). The corticocollicular descending auditory pathway extend from the layer V of the auditory cortex to the neurons of inferior colliculus (Ma & Suga, 2001; Yan & Ehret, 2002). The olivocochlear bundle originates from the superior olivary complex and projects towards the cochlea. This descending fiber tract comprises of two subsystems—medial and lateral olivocochlear bundle (Guinan, 2006). The olivocochlear bundle forms the final common pathway from the central nervous system to the cochlear receptor organs (Terreros & Delano, 2015).

Maruthy et al. (2017) divided the auditory efferent systems into caudal and rostral efferent system. The efferents from subcortical level to the lower structures is termed as the caudal efferents, whereas those originating from the cortical level is referred to as the rostral efferents. Both caudal and rostral efferent systems have been investigated for their role in speech in noise perception, auditory

plasticity, and attention (Briggs & Usrey, 2008; Campbell, Nielsen, LaBrec, et al., 2020; de Boer & Thornton, 2007; Garrido et al., 2009; Kumar et al., 2010; Kumar & Vanaja, 2004).

The medial olivocochlear bundle is the extensively studied efferent pathway. This is because functioning of this pathway can be easily studied using quick and noninvasive procedures such as contralateral inhibition of otoacoustic emissions (OAEs; Collet et al., 1990). OAEs are by-products of electromotility of the outer hair cells in the cochlea. OAEs can be recorded by placing a sensitive microphone in the ear canal. The stimulation of the contralateral ear reduces the amplitude of the OAEs, which is thought to be a result of the inhibitory effects of medial olivocochlear efferent system. Several studies have shown that the magnitude of contralateral inhibition of OAE correlates with speech perception in noise, indicating an antimasking role of the olivocochlear bundle (Abdala et al., 2014; Giraud et al., 1997; Kumar & Vanaja, 2004).

The rostral efferent system, on the other hand, is sparsely studied due to its complex nature, multiple connections, and the lack of any standardized procedures. Some indirect measures that have been used to study the rostral efferent system are assessment of (a) context-dependent encoding of speech (Maruthy et al., 2012) and (b) stimulus-specific adaptation (Anderson et al., 2009; Malmierca et al., 2015; Takaura & Fujii, 2016). In context-dependent encoding, the auditory stimuli are presented in two paradigms—a repetitive paradigm and a contextual paradigm. In the repetitive paradigm, the test stimulus is presented multiple times and the auditory cortical response is recorded. On the other hand, the contextual paradigm involves presentation of the target stimulus in context of other stimuli while recording the cortical response. The amplitude of the cortical evoked response is higher in the contextual paradigm when compared to the repetitive paradigm. In stimulus-specific adaptation, the standard and deviant stimuli (termed based on probability of occurrence) are presented in an oddball paradigm. An adaptation (weakening) of evoked response is observed for the high probability stimuli, when compared to the stimuli occurring less frequently (the deviant stimuli). This waning of response (in repetitive paradigm in context-dependent encoding of speech and standard stimulus in stimulus specific adaptation) is thought to reflect the inhibition caused due to redundancy of information and is majorly mediated by the rostral efferent system. However, the use of these techniques is limited as the magnitude of inhibition (caused by the redundant stimuli) are small and highly variable.

Sensory gating refers to the phenomenon in which cortical neural responses to repetitive stimuli are reduced compared to a novel stimulus. The auditory sensory gating is commonly assessed in a conditioning-testing paradigm which involves presenting two identical stimuli in succession. Here, the amplitude of auditory evoked potential to the second stimuli (S2) is reduced compared to that of the first (S1). This is thought to be due to the detection of redundancy of information in the S1 and S2 and the activation of a gating-out process (Boutros & Belger, 1999; Freedman et al., 1987). This phenomenon reflects top-down modulation of sensory stimuli. The sensory gating effect is robust, consistent and has been investigated in a variety of population (Adler et al., 1982; Arciniegas et al., 2000; Campbell et al., 2018; Campbell, Nielsen, Bean, et al., 2020; Lijffijt et al., 2009). We presume that this inhibitory action is likely mediated by the rostral efferent system, particularly, the corticocortical and corticothalamic pathways. In a recent study, Campbell, Nielsen, LaBrec, et al. (2020) reported an association between speech perception in noise and auditory sensory gating in normal hearing individuals with and without speech in noise deficits.

The contralateral inhibition of OAE and sensory gating are functionally similar, facilitating response inhibition in the auditory system to improve the overall efficiency of sensory processing. Therefore, it would be interesting to assess the relationship between the two mechanisms—sensory gating and contralateral inhibition of OAEs. It seems likely that the two mechanisms interact with each other to achieve common goals. Given this background, we performed the current study with the aim of understanding the interplay between the two inhibitory mechanisms in the auditory system in clinically normal hearing adults. The objectives of the study were to assess and correlate the two measures of auditory inhibition: inhibition of transient evoked OAEs (TEOAEs) amplitude brought by contralateral acoustic stimulation and the amplitude ratio between auditory cortical responses to two stimuli presented in a conditioning-testing paradigm.

Materials and Methods

Participants

We recruited 15 young normal hearing volunteers (3 males and 12 females) with a mean age of 24.47 ± 2.9 years. The participants had no known speech, language, or cognitive deficits. This was ascertained by carrying out an informal interview prior to the study. Smokers and individuals with tinnitus were excluded from the study as these factors are known to interfere with the test findings (Campbell et al., 2018; Harkrider & Hedrick, 2005). Individuals with hypertension and diabetes were also excluded from the study. All participants had air-conduction hearing sensitivity within 15 dB HL at octave frequencies between 250–8000 Hz, normal middle ear functioning, and click evoked OAE present with a minimum amplitude of 6 dB SPL. A signed informed consent was obtained from all the participants prior to the study. The study adhered to the biobehavioral ethical guidelines of the All India Institute of Speech and Hearing, Mysore (Venkatesan & Basavaraj, 2009).

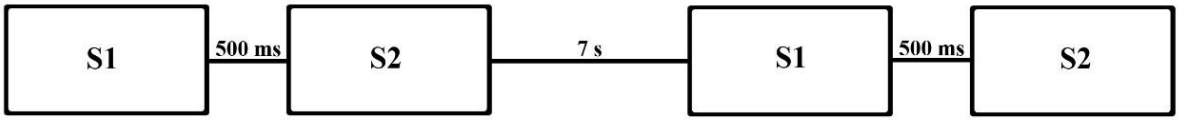
Contralateral Inhibition of TEOAE

We seated the participants comfortably and provided them with a reading material (to ensure passive attention condition) while the test was being carried out. The TEOAE probe was placed in the right ear of the participants, and an insert receiver was placed in the left ear. Following this, we presented 260 pairs of linear clicks at 70 dB peSPL using an Otodynamics ILO-V6 OAE equipment (Otodynamics Ltd., London, UK) and measured the TEOAEs. TEOAEs were recorded again in the presence of a 60 dB SPL of white noise presented to contralateral ear. The noise was delivered via an ER 3A insert receiver connected to a calibrated GSI Audiostar Pro 2-channel clinical audiometer (Grason-Stadler Inc., Eden Prairie, MN). The probe position was unaltered between the two recordings. The amplitudes obtained in these two conditions were then subtracted to obtain the magnitude of inhibition.

Sensory Gating

***Stimuli*.** The sensory gating ability of the participants was measured in a conditioning-testing paradigm. The stimuli consisted of a pair of identical 250 Hz tone bursts with 50 ms plateau and 10 ms rise–fall times (generated in Adobe Audition, version 3.0) with a gap of 500 ms between them. The first stimulus of the pair was designated as S1 and the second stimulus was designated as S2. An interval of 7s was maintained between two consecutive pairs. Figure 1 shows the graphical representation of the stimuli.

Figure 1. *Representation of the Stimulus Paradigm Used in the Sensory Gating Experiment.*



Note. *Two pairs of identical stimuli separated with an inter stimulus interval of 500 ms and an inter-pair interval of 7s.*

Recording of Auditory Evoked Potentials. The calibrated stimuli were presented using the Continuous Acquisition Module of SmartEP equipment (Intelligent Hearing System Corp., Miami, FL). We placed the noninverting electrode at Cz, inverting electrode on the test ear mastoid, and the ground electrode on the nontest ear mastoid. A second ocular channel was used to eliminate the ocular artifacts. The gain was set to 50000 µV. All the electrode impedances were kept below 5 kΩ. Stimuli were presented to the right ear of the participants at 70 dB nHL through electrically shielded ER-3A insert earphones. Participants were sitting comfortably in a reclining chair during the recording. The participants were instructed to stay as still as possible, and to reduce their eye movements during the recording. The raw EEG was recorded with a bandpass filter of 1–100 Hz and converted into a digital signal at a sampling frequency of 1000 Hz. Artifact-free responses were recorded for a total of 150 stimulus pairs. Throughout the recording, we played a close-captioned movie of participants choice to maintain passive attention.

The continuous raw EEG of every participant was subjected to offline analyses using EEGLAB (version 14.1.2; Delorme & Makeig, 2004) implemented in MATLAB (The Mathworks Inc., Natick, MA). The continuous EEG was filtered (to 1–30Hz) and epoched for 570ms. The average event-related potentials for both S1 and S2 were extracted separately. The cortical evoked response peaks P1, N1, and P2 were identified for both S1 and S2, and the amplitudes of the peaks were noted. This was then divided (S2/S1) and multiplied by 100 to obtain the sensory gating ratio. The lower the sensory gating ratio (more difference between the amplitudes of S1 and S2 responses), the better the gating mechanism.

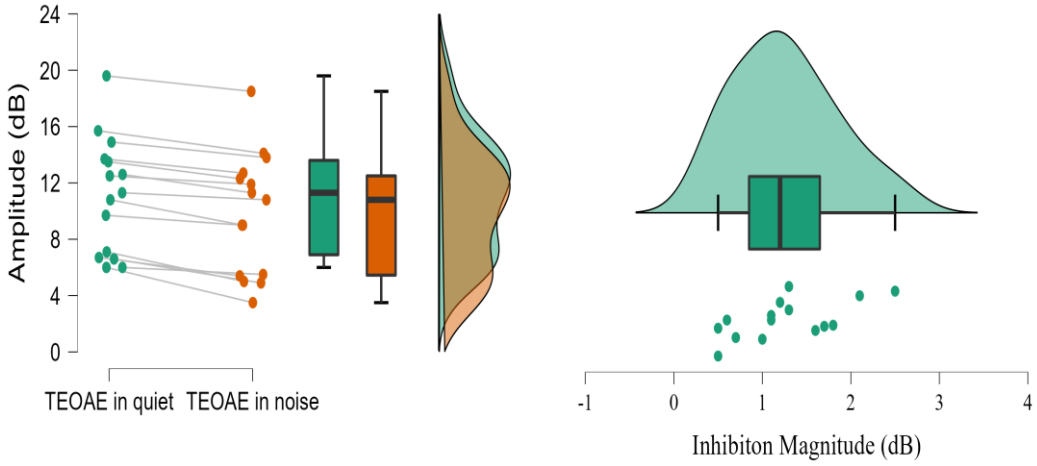
Results

All the statistical analyses were carried out using the JASP (JASP team, 2021, version 0.15.0.0) statistical software.

Contralateral Inhibition of TEOAE

A Wilcoxin Signed Rank test was run on the TEOAE amplitudes with and without contralateral stimulus (see Figure 2a, for individual and group data).

Figure 2. *(a) TEOAE Amplitudes in Quiet (Green) and with Contralateral White Noise (Orange) – Individual Data Points, Box Plots and Data Distribution; (b) The Difference Plot Depicting the Magnitude of Inhibition Brought About by the Contralateral Stimulation.*



(a) (b)

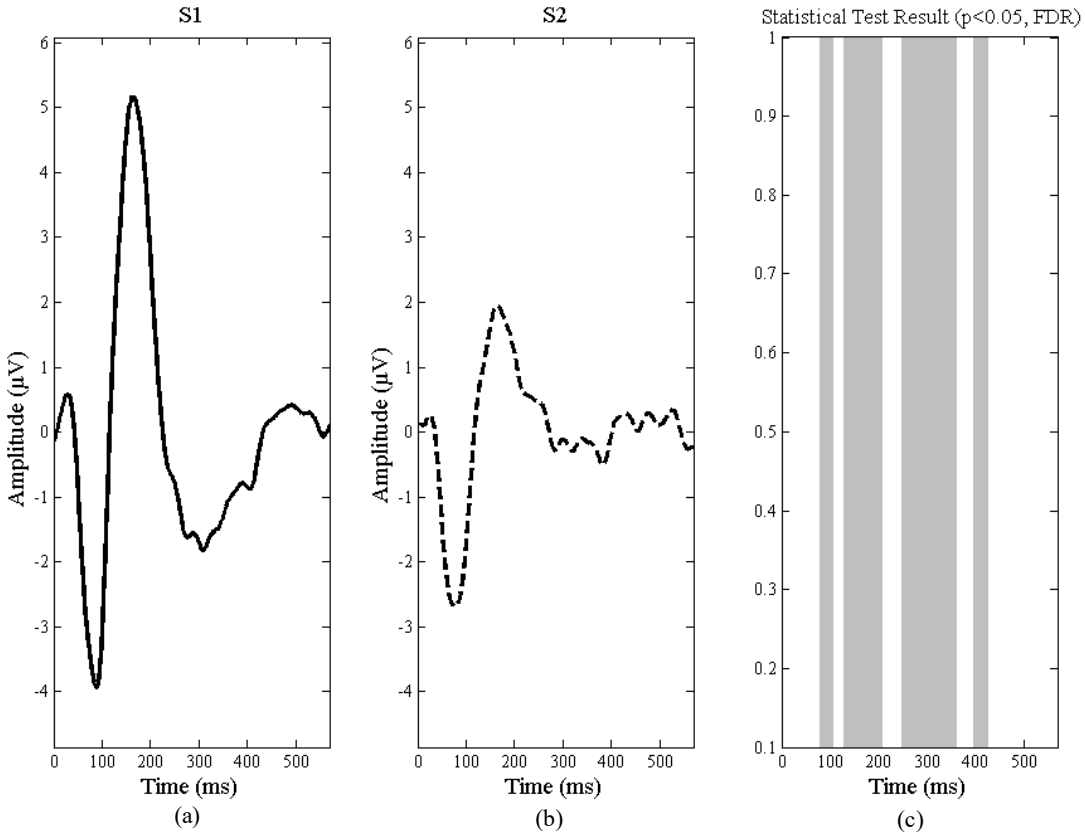
We found a significant reduction of TEOAE amplitudes in the presence of contralateral noise (Z = 3.41, p < .01). A rank-biserial correlation was performed to assess the effect size and an r value of 1 (maximum effect) was obtained (Kerby, 2014). Figure 2b depicts the inhibition magnitude (TEOAE amplitude without noise − TEOAE amplitude with noise) obtained in our study.

Sensory Gating

Figure 3a and 3b depict the grand average waveform obtained for the S1 (solid line) and S2 (dashed line) for our participants. Waveforms of S1 and S2 were analyzed using permutation-based statistics to obtain statistical significances (depicted in Figure 3c). False discovery rate (FDR) corrections were incorporated to account for multiple comparisons.

From Figure 3c it can be seen that the waveforms of S1 and S2 differed from each other between 80–110 ms (which corresponds to the N1 region), 130–210 ms (which corresponds to the P2 region), 250–360 ms, and 400–430 ms. Furthermore, peak amplitudes of P1, N1, and P2 were identified and marked.

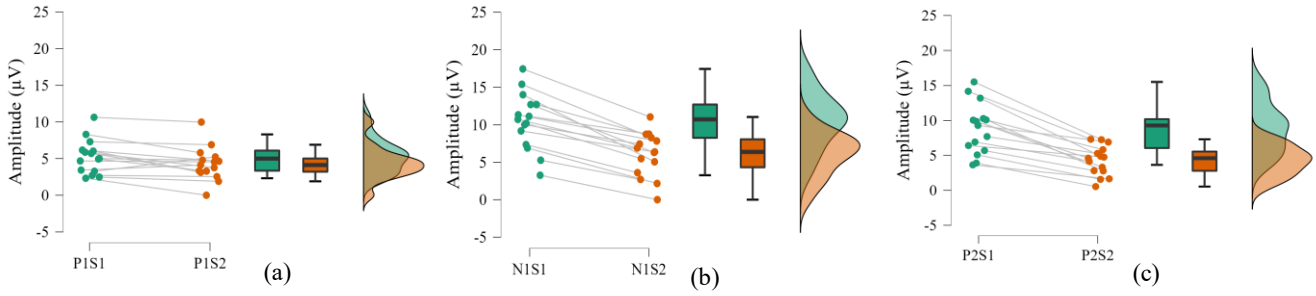
Figure 3. *Comparison of the Grand Averaged Waveform of Response Obtained in a Conditioning-testing Paradigm.*



Note. *(a) S1 – solid line. (b) S2 – dashed line. The panel (c) depicts the statistical test findings of the multiple comparisons with FDR corrections. The shaded regions indicate significant differences at 0.05 level of significance.*

Figure 4 shows the peak amplitudes (in terms of individual data points, box plots, and distribution of the data) of P1, N1, and P2 respectively for both S1 and S2 stimuli. Wilcoxon Signed Rank test showed that the amplitude of S1 was significantly higher than that of S2 for all the three peaks; P1 (Z = 2.442, p = .012, r = 0.7), N1 (Z = 3.408, p < .001, r = 1), and P2 (Z = 3.408, p < .001, r = 1).

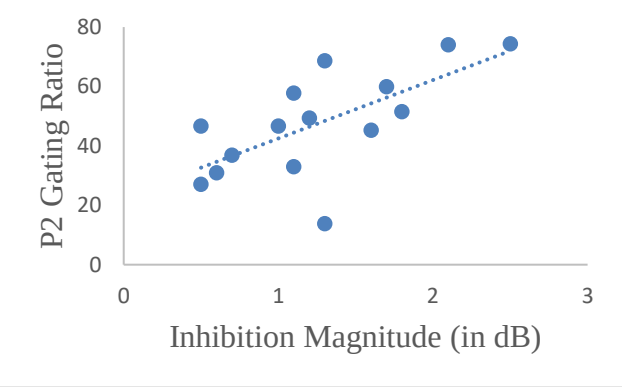
Figure 4. *The Amplitudes of the Auditory Cortical Evoked Potential in a Sensory Gating Paradigm across (a) P1, (b) N1, and (c) P2 for S1 and S2.*



Relationship Between Contralateral Inhibition of TEOAE and Sensory Gating Indices

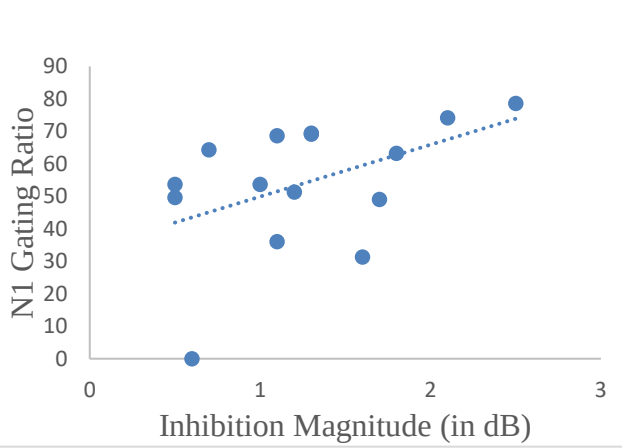
We performed the Spearman's correlation to assess the relationship between contralateral inhibition of TEOAE and sensory gating indices. The Spearman's correlation revealed a significant positive correlation between the inhibition magnitude and P2 sensory gating ratio (r = 0.666, p = .007). This indicates that individuals with higher magnitude of TEOAE inhibition showed lower sensory gating capacities. Figure 5 shows the scatter plot between TEOAE inhibition magnitude and P2 sensory gating ratio.

Figure 5. *Scatter plot depicting the relationship between magnitude of contralateral inhibition of TEOAE and P2 sensory gating ratio.*



Further, we noticed that the relationship between the inhibition magnitude and N1 gating ratio was nearing significance (r = 0.444, p =.09). Figure 6 depicts this relationship in a scatter plot.

Figure 6. *Scatter plot depicting the trend between magnitude of contralateral inhibition of TEOAE and N1 sensory gating ratio.*



Discussion

In the present study, we investigated the relationship between sensory-gating indices and magnitude of contralateral inhibition of OAEs. The results revealed a significant positive correlation between P2 sensory gating ratio and contralateral inhibition of OAEs. The contralateral inhibition of OAEs is a proven method to assess the functioning of medial olivocochlear bundle in humans (Collet et al., 1990; Guinan, 2006). The magnitude of contralateral inhibition observed in this study are comparable to those reported in literature (Maruthy et al., 2017; Stuart & Kerls, 2018). In the sensory gating experiment, we observed that amplitudes of auditory P1, N1, and P2 were significantly smaller for the second stimuli of the pair compared to the first (Figures 3, 4). The magnitude of sensory gating—as indicated by the ratios between the amplitudes of auditory evoked potential for S2 and S1—observed in the current study are also comparable to those in previous studies (Fuerst et al., 2007; Patterson et al., 2008; Rentzsch, Gomez-Carrillo de Castro, et al., 2008).

Sensory gating involves detection of redundancy of information in ongoing streams (S1 and S2) and the subsequent activation of a gating-out process (Boutros & Belger, 1999; Freedman et al., 1987). Previous studies have shown that gating response is primarily mediated by the auditory and the prefrontal cortex, with contributions from thalamic network. It is proposed that auditory cortices process the basic stimulus related information, whereas, the prefrontal cortex along with the thalamic connections inhibits further flow of sensory information in the cortex (Mayer et al., 2009). These networks are also implicated in the corticocortical and the corticothalamic auditory descending pathway (Antunes & Malmierca, 2021; Delano & Elgoyhen, 2016; Suga, 2020). Therefore, the gating response observed for auditory evoked potential may reflect the strength of the rostral efferent system. This is also in par with the predictive coding model put forth by Friston (2005). According to Friston, the cortical structures continuously monitor the incoming stimuli and generate predictions about the next stimuli. These predictions, when met, lead to an inhibition of responses following the redundant information. However, a deviation in the stimuli causes a "prediction error" and brings about facilitatory responses. The presence of such predictive mechanisms in the auditory system are supported by other studies as well (O'Reilly, 2021; Todorovic & de Lange, 2012). We propose that the presentation of S1 (in a conditioning-testing paradigm) leads to the prediction of S2, thereby causing response

inhibition. Though the sensory gating has previously been extensively evaluated in individuals with schizophrenia (Adler et al., 1985; Hirano et al., 2010), it may additionally prove to be a measure of the rostral efferents. Previous studies confirm that sensory gating can be reliably measured in healthy participants as well as in individuals with various disorders (Campbell et al., 2018; Fuerst et al., 2007; Rentzsch, Jockers-Scherübl, et al., 2008).

There is some evidence to suggest that both the rostral and caudal efferent systems aid in speech perception in noise. Campbell, Nielsen, LaBrec, et al. (2020) reported that individuals with better sensory gating abilities performed better in tasks involving perception of speech in adverse listening situations. Similarly, several studies have shown that individuals with higher magnitude of contralateral inhibition of OAEs had better speech perception in noise scores (Kumar & Vanaja, 2004; Mertes et al., 2019). These studies in combination suggest that the two subsystems of efferent auditory pathway (rostral and caudal) may function together to achieve this goal. However, as we did not assess the speech perception in noise abilities of our participants, the contribution of the descending pathways to such functions cannot be commented on.

Disruptions of these inhibitory mechanisms (both caudal and rostral) have been associated with tinnitus and schizophrenia (Adler et al., 1985; Campbell et al., 2018; Riga et al., 2007; Wahab et al., 2016). In tinnitus, the magnitude of inhibition measured using contralateral suppression of OAEs and sensory gating are reduced (Campbell et al., 2018; Riga et al., 2007). Additionally, Campbell et al. (2018) directly associated the severity of tinnitus with reduced rostral efferent activity. In schizophrenia, Wahab et al. (2016) reported increased inhibition of OAEs and Adler et al. (1985) showed reduced sensory gating. The reduced sensory gating in schizophrenia has been linked positively to increased auditory hallucinations (Smith et al., 2013).

In the present study, we observed a significant positive correlation between P2 sensory gating ratio—presumed to reflect the strength of rostral efferent system—and the magnitude of contralateral inhibition of OAEs—presumed to reflect the strength of caudal (olivocochlear) efferent system. Positive correlation indicates that individuals with higher magnitude of OAE inhibition showed lower sensory gating capacities, and vice versa. We think that the reduced activity at one level in the efferent auditory system is compensated at the other level. The precise nature and consequences of this reciprocal relationship is unclear at present. To the best of our knowledge, this is the first study to investigate the relationship between the two inhibitory effects in the auditory system—one central and other peripheral. It is possible that these two mechanisms work in tandem to balance the overall inhibitory effect on the stimuli. This reciprocal relationship between the two subsystems of the efferent system might aid in adequate inhibition of the undesired stimuli, while facilitating the processing of the desired target. The balanced/differential inhibition mediated by the auditory efferent system may play a crucial role in many auditory processes such as auditory attention, listening in the presence of competing signals, binaural hearing, and auditory plasticity. Therefore, investigating the interactions within the auditory inhibitory networks is crucial in understanding the normal auditory perception and pathophysiology of various auditory related disorders. However, the results of the present study should be generalized with caution and requires further augmentation.

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