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Effects of a preceding sound on medial olivocochlear bundle reflex as a function of the preceding time interval

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ABSTRACT

Our previous study reported that a preceding sound, regardless of whether it induced a medial olivocochlear bundle reflex (MOCR) by itself, expedites MOCR, and a 60-dB-SPL preceding-noise, which also induces an MOCR, that enhances, in addition to expediting, the MOCR. The current study compares the dependency of the enhancing and expediting effects on the preceding time interval. The MOCR is induced by sounds and exerts an inhibitory effect on the outer hair cells. The suppressive effect was assessed non-invasively in terms of the suppression of otoacoustic emissions (OAEs) induced by a contralateral acoustic stimulation, referred to as MOCR elicitor. A 60-dB-SPL noise was used as a preceding sound, and the inter-stimulus interval (ISI) between the preceding sound and the MOCR elicitor was varied from 0.5 to 2 s. The MOCR strength decreased as the ISI became larger and reached the same level as without a preceding sound within 2 s of ISI. In contrast, the onset delay of the MOCR did not depend on the ISI and was still significantly smaller than without a preceding sound even at 2 s of ISI. The difference in the dependency of the preceding time interval implicates that the preceding-sound-induced enhancing and expediting effects are underpinned by different mechanisms.

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Keywords: medial olivocochlear reflex, preceding sound, otoacoustic emissions

1. INTRODUCTION

The medial olivocochlear reflex (MOCR) is a feedback response that controls the input to the auditory system. Outer hair cells (OHCs) are innervated by the medial part of the superior olivary complex via the medial olivocochlear (MOC) bundle. These MOC fibers are activated by acoustic stimulation and exert an inhibitory effect on OHC motility. This suppressive effect has been termed the MOCR, and is thought to play an important role in improving the detection of signals in the presence of background noise (1, 2), in selectively attending to target signals (3), and in protecting the auditory periphery from damage caused by acoustic overexposure (4, 5).

Most previous studies of the MOCR have measured the responses induced by single acoustic stimuli, such as a single short-duration or continuous contralateral white noise (5, 6, 7, 8), and it is not clear whether or how the MOC system changes its behavior based on the history of time-varying acoustic inputs. In contrast, in a previously published study, we reported that a preceding sound, regardless whether it induced an MOCR by itself, expedites MOCR, and a 60-dB-SPL preceding-noise, which itself induces an MOCR, and not only expedites but also enhances the MOCR (9). The result implies that MOCR dynamically changes depending on the history acoustic inputs. However, it is not clear whether the preceding-sound-induced enhancing and expediting effects are underpinned by the same

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mechanism. To clarify this, the present study compares the dependency of the enhancing and expediting effects on the inter-stimulus interval (ISI) between the preceding sound and the MOCR elicitor. Hypothetically, if both effects were caused by the same mechanism, their ISI dependency would be similar.

2. METHODS

2.1 Participants

Twelve volunteers (two males and ten females) aged 21–32 years (mean=23.8, standard deviation=3.7) participated in the study. Ears from all volunteers had normal pure-tone audiometric thresholds (HL< 20dB), ranging from 0.25 to 8kHz. The experiments were approved by the Research Ethics Committee of NTT Communication Science Laboratories and Chiba University.

2.2 Stimuli and procedure

We measured the contralateral MOCR in terms of the suppression of click-evoked otoacoustic emissions (CEOAEs) induced by noise presented to the contralateral ear. Click trains were played into the right ear canal to generate CEOAEs from within the cochlea. Each click had a duration of 100 μs and was presented at a 55-dB peak-equivalent sound pressure level (SPL). Clicks were presented at a rate of 50 times per second. The MOCR was elicited by a noise presented to the left ear, referred to as the MOCR elicitor, during the CEOAE recording. The noise was band-pass filtered between 100 and 10,000 Hz, with a duration of 0.5 s, including a 10-ms raised-cosine ramp. The noise was presented to the left ear at 60-dB SPL. As a preceding sound, the same noise was presented prior the MOCR elicitor. To evaluate the effect of the preceding time interval, the inter-stimulus interval (ISI) between the preceding sound and MOCR elicitor was varied in 0.5, 1, 1.5 and 2 s. A MOCR without any preceding sound was also measured.

2.3 Recording and data analysis

Ear-canal sound pressure was recorded with an Etymotic Research ER-10B low-noise microphone system inserted in each ear. The recorded signals were band-pass filtered between 1 and 4 kHz, for which the largest MOCR-related CEOAE suppression was observed. An epoch with duration of 2.5 s, including 1.5 s pre-onset and 0.5 s post-offset of the MOCR elicitor, was extracted from the filtered signals for every presentation of an MOCR elicitor. The extracted epochs were averaged across the trials for each condition, and a time series composed of 125 CEOAE waveform samples was obtained from the averaged epoch. To smooth fluctuations included in the time series, ten adjacent CEOAE waveform samples were averaged for each time point. The CEOAE level (in dB SPL) was calculated as an RMS value for each waveform sample in the 8–18-ms region of the waveform. Lastly, a time course of MOCR was obtained by subtracting the baseline level from the time series of CEOAE levels. The baseline level was defined as the average CEOAE level in a period of 1 sec prior to the onset of the first stimulus in a series. The strength of the MOCR for each time course was defined as the mean suppression between time points 0.25 and 0.75 s after the onset of the preceding sound. The onset delay was defined as the time at which the OAE level decreased by 0.25 dB from the baseline.

2.4 Equipment

Stimuli were digitally synthesized with sampling rates of 48 kHz and converted to analog signals using an Fireface UCX (16 bits). The analog signals were amplified by a headphone buffer and presented through Etymotic Research ER-3A earphones connected to an ER-10B low-noise microphone system. The two outputs from the ER-2A were calibrated using a DB2012 accessory (external ear simulator) of a Bruel and Kjaer Type 4257 ear simulator (IEC 711). Ear canal sound pressure was recorded using an Etymotic Research ER-10B low-noise microphone system inserted in each ear. All measurements were conducted in a double-wall sound-attenuating room.

3. RESULT AND DISCUSSION

Consistent with the previous study (9), it took several hundred ms for the MOCR to build up and the strength of suppression ranged from 2.5 to 3 dB. The MOCR strength decreased as the ISI became larger and reached the same level as without a preceding sound within 2 s of ISI. In contrast, the onset delay of the MOCR did not depend on the ISI and was still significantly smaller than without a preceding sound even at 2 s of ISI. We compared the strengths and onset delay of the MOCR induced by a preceding sound across the conditions using a repeated-measures analysis of variance (ANOVA), with the ISI as the within-subjects factor. The ANOVA revealed a significant effect of ISI on the strength of the MOCR ($F_{3, 33} = 10.5$, $p<0.001$). A *post hoc* comparison using Ryan's method showed that MOCR with ISI of 2 s was significantly larger than that with ISI of 1 s ($T = 3.0$, $p = 0.0047<$nominal level calculated by Ryan's method; Fig. 1) and 1.5 s ($T = 5.1$, $p<0.0001<$nominal level calculated by Ryan's method; Fig. 1). In contrast, there was no significant effect of ISI on the onset delay of the MOCR ($F_{3, 33} = 0.48$, $p = 0.70$).

Together with the previous results that a 40-dB preceding tone, which itself does not induce MOCR, did not enhance MOCR (Otsuka *et al*. 2018), the enhancement may reflect an adaptation induced by a preceding sound. A possible mechanism is the slow component of the MOCR. Cooper and Guinan reported that repeated electrical shocks to the MOC bundle increase MOCR strength on a time scale of tens of seconds, referring to this as the "slow effect" (10). Guinan and coworkers attributed the slow effect to a gradual decrease in OHC stiffness due to prolonged release of ACh, which is the primary neurotransmitter at MOC terminals (11). The decrease in OHC stiffness should recover with time after the stimulation, and the monotonic decrease of MOCR enhancement as a function of ISI may reflect the recovery process. In contrast, the expediting effect lasts at least two seconds. The difference in the dependency of the preceding time interval implicates that the expediting effect cannot be attributed easily to the adaptation in the cellular processes, and the preceding-sound-induced enhancing and expediting effects are underpinned by different mechanisms.

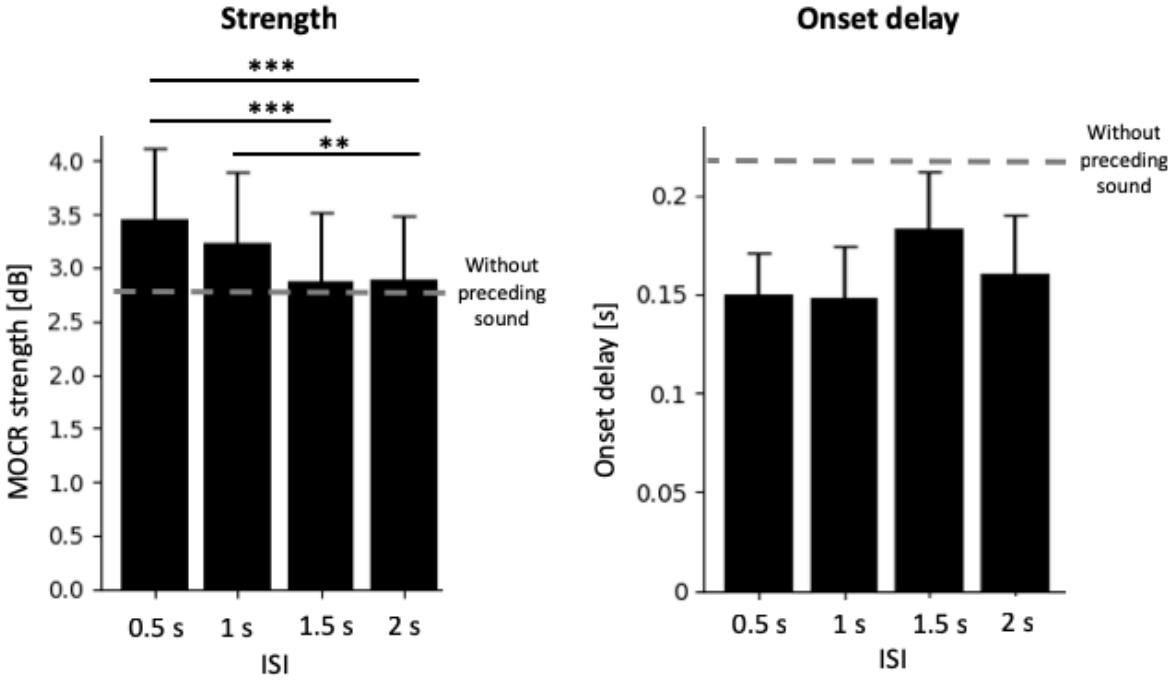

Figure 1 Comparison of the effects of ISI between a preceding noise and MOCR elicitor on the strength and onset delay of MOCR. Error bars represent standard error of the mean. **p<0.01, ***p<0.0001 (Corrected for multiple comparisons with Ryan's method). ISI, inter-stimulus interval; MOCR, medial olivocochlear reflex.

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