

## Olivocochlear efferent contributions to speech-in-noise recognition across signal-to-noise ratios

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# Olivocochlear efferent contributions to speech-in-noise recognition across signal-to-noise ratios<sup>a)</sup>

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The medial olivocochlear (MOC) efferent system modifies cochlear output to aid signal detection in noise, but the precise role of efferents in speech-in-noise understanding remains unclear. The current study examined the contribution of the MOC reflex for speech recognition in noise in 30 normal-hearing young adults (27 females, mean age = 22.7 yr). The MOC reflex was assessed using contralateral inhibition of transient-evoked otoacoustic emissions. Speech-in-noise perception was evaluated using the coordinate response measure presented in ipsilateral speech-shaped noise at signal-to-noise ratios (SNRs) ranging from  $-12$  to  $0$  dB. Performance was assessed without and with the presence of contralateral noise to activate the MOC reflex. Performance was significantly better with contralateral noise only at the lowest SNR. There was a trend of better performance with increasing contralateral inhibition at the lowest SNR. Threshold of the psychometric function was significantly correlated with contralateral inhibition. Response time on the speech task was not significantly correlated with contralateral inhibition. Results suggest that the MOC reflex contributes to listening in low SNRs and the relationship between the MOC reflex and perception is highly dependent upon the task characteristics. © 2019 Acoustical Society of America.

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## I. INTRODUCTION

The human auditory system contains a rich efferent network wherein the cortex modulates brainstem activity, and the brainstem influences cochlear activity. The auditory efferent system improves detection of sounds in background noise, reduces auditory damage due to high-intensity sound exposure, and may be involved in selective attention (for recent reviews, see [Guinan, 2018](#); [Lopez-Poveda, 2018](#)). The medial olivocochlear (MOC) branch of the auditory efferent system innervates the outer hair cells (OHCs), which are responsible for cochlear amplification (for a review of cochlear amplification, see [Ashmore, 2008](#)). Stimulation of the MOC reflexively alters cochlear amplifier function and is therefore termed the medial olivocochlear reflex (MOCR). Activation of the MOCR causes hyperpolarization of OHCs and an increase in OHC stiffness ([Housley and Ashmore, 1991](#); [Dallos et al., 1997](#); [Cooper and Guinan, 2003](#)). The result is that OHCs provide less cochlear amplification of basilar membrane motion ([Murugasu and Russell, 1996](#); [Guinan and Cooper, 2008](#)) due to a reduction in OHC electromotility. MOCR activation increases firing rates and increases the dynamic range of auditory nerve fibers in response to brief stimuli presented in a background of continuous noise ([Winslow and Sachs, 1987, 1988](#); [Kawase et al., 1993](#)). This process is often referred to as *antimasking*.

Such continuous noise is a strong activator of the MOCR, decreasing neural adaptation in response to the noise and allowing for transient stimuli to be encoded neurally ([Lieberman and Guinan, 1998](#)).

These physiologic effects of the MOCR have implications for sound perception. Antimasking can improve the signal-to-noise ratio (SNR) at the level of the cochlea, and it is well known that increases in SNR result in better recognition of speech materials in noise (e.g., [Festen and Plomp, 1990](#)). Animal work has demonstrated that surgical lesions of MOC fibers impair psychophysical performance, including discrimination of high-frequency pure tone intensity ([May and McQuone, 1995](#)), vowel formants presented in noise ([Hienz et al., 1998](#)), and phonemes presented in noise ([Dewson, 1968](#)). These processes are important for speech understanding. For example, intensity discrimination is involved in the perception of stop consonants and vowel formants ([Sinnott et al., 1985](#)). Additionally, the MOCR may aid in encoding amplitude modulation at the level of the basilar membrane ([Marrufo-Pérez et al., 2018a](#)), which can benefit perception of the different temporal components of the speech waveform ([Rosen, 1992](#)).

The contribution of the MOCR to auditory perception in humans has been examined indirectly through correlational studies of MOCR function and performance on behavioral tasks. In such studies, MOCR strength is assessed through measurements of otoacoustic emissions (OAEs), which are sounds generated as a byproduct of cochlear amplification that can be measured with a probe microphone placed in the ear canal (reviewed in [Probst et al., 1991](#); [Kemp, 2002](#)). Because MOCR activation decreases cochlear amplifier

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gain, it also decreases the amplitude of OAEs. Typically, OAEs are measured in the ipsilateral ear and the MOCR is activated by presenting broadband noise to the contralateral ear (Collet *et al.*, 1990). This contralateral measurement is methodologically convenient, but it must be noted that it only assesses the contralateral, but not the ipsilateral, MOC pathway. The decrease in OAE amplitude is referred to as contralateral inhibition, where larger inhibition values are interpreted as stronger MOCs (De Ceulaer *et al.*, 2001; Backus and Guinan, 2007).

Human studies have demonstrated a link between MOCR activity and sound perception. This work has shown that participants with stronger MOCR activity had better performance on a number of behavioral tasks relative to participants with weaker activity, including difference limens for pure-tone intensity (Micheyl *et al.*, 1997), phoneme discrimination in noise (de Boer and Thornton, 2008; Abdala *et al.*, 2014), monosyllabic word recognition in noise (Giraud *et al.*, 1997; Kumar and Vanaja, 2004; Mishra and Lutman, 2014), and sentence identification in noise (Bidelman and Bhagat, 2015; Maruthy *et al.*, 2017; Mertes *et al.*, 2018). These results are consistent with the hypothesis that the MOCR unmasks target signals (i.e., speech) in the presence of background noise.

However, conflicting evidence from other studies has called into question the nature and extent of the MOCR's involvement for speech in noise. Two studies have found no significant association between speech-in-noise abilities and the MOCR (Mukari and Mamat, 2008; Wagner *et al.*, 2008), and two studies found an inverse correlation (de Boer *et al.*, 2012; Milvae *et al.*, 2015). These discrepancies may be explained in part by differences in a number of factors, which may include participant characteristics, the specific speech-in-noise task, the MOCR measurement and analysis, and the stimulus levels. Additionally, our recent work (Mertes *et al.*, 2018) found that the strength of the MOCR was correlated with the slope of the psychometric function on speech-in-noise tasks measured across two SNRs but not at a single SNR. This suggests that the relationship between the MOCR and speech-in-noise ability may only be apparent when measured across listening conditions such as multiple SNRs. The current study examined the relationship between MOCR function and speech-in-noise performance in normal-hearing young adults. Performance was assessed across a range of SNRs in the presence of both ipsilateral and bilateral noise.

## II. METHODS

### A. Participants

Participants were recruited from the University of Illinois at Urbana-Champaign campus. The study protocol was approved by the University of Illinois at Urbana-Champaign Institutional Review Board, and written informed consent was obtained from all participants. All participants received monetary compensation. Interested individuals first completed a brief electronic mail questionnaire to determine initial eligibility. Individuals were invited for a laboratory visit if their responses indicated that they were

between 18 and 40 yr old and had a healthy hearing history, defined as a negative history of: hearing difficulties; noise exposure within the past six months that caused tinnitus, aural fullness, and/or muffled hearing; tinnitus of a severe and/or bothersome nature; use of ototoxic medication; vertigo; and chronic middle ear pathology. Additionally, participants were invited only if they reported speaking English as their first language to avoid potential confounds of native language on speech-in-noise perception (Mayo *et al.*, 1997) and if they reported being right-hand dominant to avoid potential handedness effects on contralateral inhibition (Khalfa *et al.*, 1997).

At the initial laboratory visit, informed consent was obtained and participants underwent an audiologic screening in a sound-treated booth. Audiologic inclusion criteria consisted of the following: an unremarkable otoscopic examination, 226-Hz tympanograms within normal clinical limits (tympanometric peak pressure:  $-100$  to  $+50$  daPa; static acoustic admittance: 0.2 to 1.8 mmho; equivalent ear canal volume: 0.6 to 2.5 cc), ability to perceive pure-tone air-conduction stimuli presented at 20 dB hearing level (HL) for octave frequencies from 250 to 8000 Hz, and measurable transient-evoked otoacoustic emissions (TEOAEs) in the right ear in response to 65 dB peak sound pressure level (pSPL) clicks presented at 19.5/s after obtaining 1250 sweeps. TEOAEs were deemed present when the time-domain SNR was  $>6$  dB and the reproducibility was  $>70\%$  when analyzed from 1000 to 2000 Hz (Mertes, 2018).

A total of 30 participants (27 females) completed the experiment. The mean age was 22.7 yr [standard deviation (SD) = 5.0] and the age range was 18 to 36 yr. It should be noted that we did not attempt to include an equal number of males and females. However, recent work suggests that there is not a significant effect of sex on contralateral inhibition of TEOAEs (Stuart and Kerls, 2018).

### B. Equipment

All study procedures were conducted in a single-walled sound-treated booth (Tracoustics, Inc., Austin, TX). The ambient noise levels in the booth met ANSI S3.1-1999 standards (ANSI, 1999) for testing with ears covered and uncovered. For contralateral inhibition measurements, stimulus delivery and response acquisition were achieved with an RZ6 auditory processor [Tucker-Davis Technologies (TDT), Alachua, FL] and a WS4 workstation (TDT). Acoustic stimuli were delivered via ER-2 insert earphones (Etymotic Research, Elk Grove Village, IL). TEOAEs were recorded using an ER-10B+ probe microphone (Etymotic Research) with 40 dB of preamplifier gain. The microphone signal was routed to the RZ6 processor and sampled at the default processor rate of 24 414.1 Hz. Contralateral inhibition testing and analysis were conducted using custom code written in MATLAB (version R2017A, The MathWorks, Inc., Natick, MA) and RpvdsEx (TDT). For speech-in-noise testing, stimuli were routed from the RZ6 processor to a Babyface Pro audio interface (RME, Haimhausen, Bavaria, Germany) and then to a pair of ER-2 insert earphones. Participants made responses to the speech stimuli on a touch screen monitor.

Speech-in-noise testing was conducted with custom code written in MATLAB (provided by Dr. F. J. Gallun).

### C. Contralateral inhibition

Below, a brief description of the contralateral inhibition methodology is provided and described more fully in [Mertes \(2018\)](#). Participants were seated upright in a recliner during testing. They were instructed to relax, sit quietly, and refrain from moving, coughing, or swallowing as much as possible during testing. The experimenters were seated in an adjacent room and monitored participants in the booth using a video camera and intercom. Because sleep and changes in attention can introduce variability in measurements of contralateral inhibition ([Froehlich et al., 1993](#); [de Boer and Thornton, 2007](#)), we attempted to reduce this variability by having participants watch a closed-captioned, silent video of their choice on a tablet computer during testing to keep them awake, alert, and still.

Contralateral inhibition was measured by recording TEOAEs in the right ear with and without contralateral acoustic stimulation (CAS) consisting of broadband Gaussian noise presented to the left ear at 60 dB(A). Clicks were presented at 65 dB pSPL and at a rate of 19.5/s. A total of 1250 sweeps were recorded each without and with CAS (referred to hereafter as “CAS-” and “CAS+”, respectively). Responses were band pass filtered from 1000 to 2000 Hz, and high-amplitude artifacts were rejected *post hoc*. Contralateral inhibition was computed as the difference in root-mean-square (RMS) TEOAE waveform amplitude in CAS- minus CAS+ in the time window from 8 to 18 ms, where 0 ms was the time location of the peak stimulus amplitude. This time window was chosen because previous work has shown that the largest contralateral inhibition occurs within this window ([Berlin et al., 1993](#)). Positive difference values denoted inhibition and larger values were interpreted as stronger MOCRs ([Backus and Guinan, 2007](#)). Middle ear muscle reflex (MEMR) activation was assessed because it can alter TEOAE amplitudes and thus confound the interpretation of the MOCR results ([Guinan, 2006](#)). To assess MEMR activation, the average pSPL of the recorded click stimuli in the CAS+ and CAS- conditions was obtained and the amplitude difference was computed in dB. If this difference exceeded 0.12 dB, MEMR activation was considered present ([Abdala et al., 2013](#)). However, no participants exhibited MEMR activation using this criterion.

MOCR inhibition data were included if the TEOAE SNR was >6 dB. Additionally, the mean TEOAE difference waveform (CAS+ minus CAS-) was required to have an SNR >6 dB based on the recommendations of [Guinan \(2011\)](#). Such criterion would help to exclude contralateral inhibition measurements that are contaminated by background noise and do not contain appreciable changes in TEOAE amplitude.

### D. Speech-in-noise perception

Speech-in-noise testing was conducted at a separate visit after contralateral inhibition testing. The coordinate response measure (CRM; [Bolia et al., 2000](#)) was used to

assess speech-in-noise ability across a range of SNRs. The CRM is a recorded corpus of speech materials consisting of a carrier phrase followed by 1 of 32 color-number combinations (numbers 1 through 8 and the colors red, blue, green, and white). An example sentence is “Ready Charlie go to *blue one* now.” We chose the CRM for this study because there are limited practice effects, low chance performance, and lack of contextual cues. The same male talker and carrier phrase “Ready Charlie go to” was used each time.

Speech stimuli were always presented in ipsilateral speech-shaped noise. The speech-shaped noise was developed from the CRM corpus by concatenating all CRM speech waveforms for the male talker, computing a fast Fourier transform (FFT), randomizing the phases, and then computing an inverse FFT. To assess the contribution of the contralateral MOCR to speech-in-noise perception, the task was completed at each SNR without and with CAS (CAS- and CAS+, respectively). Figure 1 shows a schematic of the two CAS conditions. Because the bandwidth and amplitude of CAS impacts the magnitude of the MOCR ([Veuille et al., 1991](#); [Velenovsky and Glatke, 2002](#); [Lilaonitkul and Guinan, 2009](#)), the CAS for the speech-in-noise task was identical to that of the contralateral inhibition testing to eliminate confounds of different CAS bandwidth and amplitude between tasks. Ipsilateral and contralateral noises were presented alone for 500 ms before and after the speech stimuli to allow for the onset and offset, respectively, of the MOCR

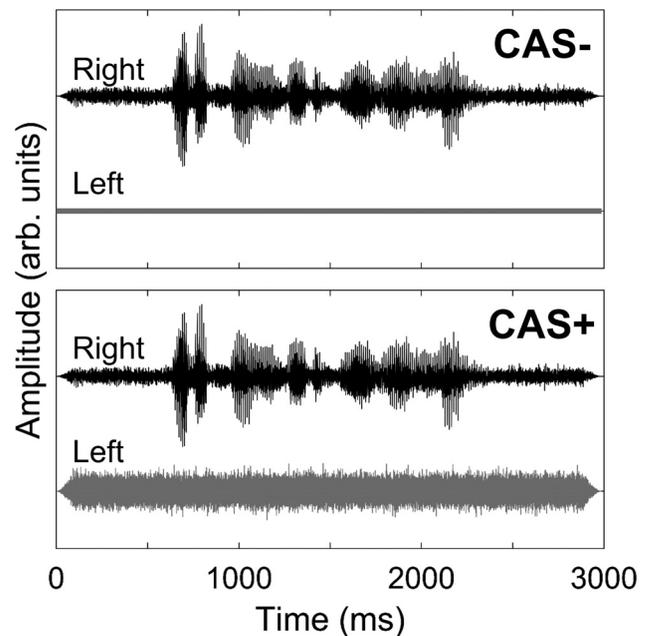


FIG. 1. Schematic of stimulus waveforms for a single trial of the CRM task. The top and bottom panels display the CAS- and CAS+ conditions, respectively. Presentations of a single CRM speech stimulus (top waveform) and CAS (bottom waveform) are shown in each panel. In the CAS- condition, speech and ipsilateral speech-shaped noise were presented to the right ear and no contralateral noise was presented (represented by the flat horizontal line). In the CAS+ condition, speech and ipsilateral speech-shaped noise were presented to the right ear, and broadband Gaussian noise was presented to the left ear. Waveform amplitudes shown in the figure were modified for illustration purposes and do not represent the relative amplitudes of the actual stimuli.

(Backus and Guinan, 2006). It must be noted that the ipsilateral speech-shaped noise presumably activated the ipsilateral MOCR pathway, while the introduction of CAS activated the contralateral MOCR pathway. However, our contralateral inhibition measurements did not allow for a quantification of participants' ipsilateral MOCR functioning. Our metric of interest was how speech-in-noise perception changed with only ipsilateral MOCR activation (ipsilateral speech-shaped noise only) compared to bilateral MOCR activation (ipsilateral speech-shaped noise and CAS). This approach has also been examined in previous studies (Giraud *et al.*, 1997; Kumar and Vanaja, 2004; Mishra and Lutman, 2014), but these studies did not analyze the psychometric function across SNRs, which may be a more sensitive index of MOCR contributions to speech-in-noise perception than score at a single SNR (Mertes *et al.*, 2018).

Based on preliminary testing conducted on the authors (normal-hearing adults falling within the allowable age range of the study participants), five SNRs of  $-12$  to  $0$  dB (in 3-dB steps) were used to provide a psychometric function spanning from approximately 0% to 100% correct. Speech, speech-shaped noise, and CAS were presented through ER-2 insert earphones. All stimuli were calibrated in a 2-cc coupler. The speech waveforms were calibrated to have an overall RMS amplitude of 50 dB(A). The speech-shaped noise (presented ipsilateral to the speech in all conditions) and CAS were calibrated to have an overall RMS amplitude of 60 dB(A). The speech and CAS waveforms were fixed in amplitude while the speech-shaped noise waveform was scaled digitally to yield the five different SNRs.

Participants were seated at a small desk inside the sound booth for speech perception testing. The experimenters were seated in an adjacent room and monitored participants using an intercom. Additionally, the experimenters monitored each participants' progress on the experiment using a graphical user interface displayed in MATLAB. A grid of the 32 color-number combinations was displayed to participants on a touch screen monitor. Participants were instructed to press the square in the grid corresponding to the color-number they heard and guess whenever they were unsure what the correct answer was.

After verbal instructions, participants began with a brief practice session. The practice consisted of 15 trials (individual stimulus presentations) with 5 trials in quiet, 5 trials with ipsilateral speech-shaped noise presented at 0 dB SNR, and 5 trials with ipsilateral speech-shaped noise at 0 dB SNR along with CAS. In order to proceed to the test conditions, participants were required to perform correctly on  $\geq 14$  practice trials, otherwise, the practice was repeated until they scored  $\geq 14$  correct. One participant required the practice to be repeated once before achieving criterion performance.

There were ten test conditions (5 SNRs  $\times$  2 CAS conditions) presented in an order that was randomized for each participant.<sup>1</sup> For each condition, a total of 50 trials were presented. The specific color-number combination presented at each trial was randomized. Participants were required to correctly identify both the color and number to be correct. After each trial, on-screen feedback ("correct" or "incorrect") was displayed. Two mandatory breaks were provided after

conditions 3 and 6 (2.5-min and 5-min in duration, respectively). Participants were encouraged to take additional breaks if needed. The laboratory visit for speech-in-noise testing lasted a total of approximately 75 min.

The outcomes of interest were scores at each individual SNR, and the slope and threshold of the psychometric function. Psychometric functions were computed on each participant's results using a logistic function of the form

$$p = 100 \left( \frac{1}{1 + e^{-(a+bx)}} \right), \quad (1)$$

where  $p$  is the percent correct,  $x$  is the SNR, and  $a$  and  $b$  are coefficient estimates for constructing the psychometric function that were obtained from MATLAB function "glmfit.m." Slope was computed as the gradient of the function when  $p = 50\%$ . Threshold of the psychometric function (in dB SNR) was computed as the point along the  $x$  axis of the function corresponding to  $p = 50\%$ . However, because floor and ceiling effects were present at the lowest and highest SNRs, respectively, all fitted values in percent correct were transformed to rationalized arcsine units (RAU; Studebaker, 1985) to minimize the impact of these effects. The minimum and maximum possible scores were  $-16.5$  and  $116.5$  RAU, respectively (Studebaker, 1985).

## E. Response time as a metric of listening effort

Response time has been used to assess listening effort during speech-in-noise tasks (e.g., Sarampalis *et al.*, 2009; Houben *et al.*, 2013), where faster response times are interpreted as reduced listening effort. In addition to increasing speech intelligibility, the antimasking effects provided by the MOCR may decrease the cognitive effort required to understand speech, perhaps in an analogous way to hearing aid noise reduction algorithms that reduce response time during speech understanding in noise (Sarampalis *et al.*, 2009). Although not the primary focus of this study, we also measured participant response time during the CRM task as an exploratory investigation into listening effort. During each trial of the CRM task, we measured response time as the difference in time between when the speech stimulus was presented in its entirety (participants were not able to press a response button until the stimulus was completed) and when the participant pressed the corresponding button. It must be noted that we did not provide instructions regarding how quickly participants should respond.

## F. Predictions

We hypothesized that activation of the MOCR aids with performance on speech-in-noise perception. This hypothesis is based on physiologic data obtained in animals, including increased auditory nerve fiber firing rates to transients presented in noise (Winslow and Sachs, 1987; Kawase *et al.*, 1993) and the negative impact of olivocochlear lesions on vowel discrimination (Dewson, 1968; Hienz *et al.*, 1998) and intensity discrimination (May and McQuone, 1995). To test this hypothesis, we predicted that scores on the CRM would significantly improve with the introduction of CAS.

Furthermore, based on previous findings that CAS improves scores at moderately challenging SNRs but not at the most or least challenging SNRs (Giraud *et al.*, 1997; Kumar and Vanaja, 2004), we predicted that the improvement in score would be most pronounced at SNRs of  $-9$  to  $-3$  dB but not at  $-12$  dB and  $0$  dB. Additionally, it was predicted that contralateral inhibition would be significantly positively correlated with score, both at individual SNRs, and as the slope and threshold of the psychometric function computed across SNRs (Mertes *et al.*, 2018). Finally, it was predicted that response time would be significantly lower in CAS+ than CAS-, and that response time would be significantly correlated with contralateral inhibition.

### G. Statistical analysis

Analyses were conducted using SPSS Statistics (version 24.0.0.0, IBM Corp., Armonk, NY) and the MATLAB Statistics and Machine Learning Toolbox (version 11.1, The MathWorks, Inc., Natick, MA). A significance level of  $\alpha = 0.05$  was used. When multiple comparisons were conducted,  $p$ -values were adjusted using the false discovery rate adjustment procedure (Benjamini and Hochberg, 1995) using the “mafdr.m” function of the MATLAB Bioinformatics Toolbox (version 4.8, The MathWorks, Inc.).

## III. RESULTS

### A. Contralateral inhibition

TEOAE signal amplitudes and noise floor amplitudes [both expressed in dB sound pressure level (SPL)] were first compared between CAS- and CAS+ using Wilcoxon signed-rank tests [an outlier was present in the noise floor amplitude data as indicated by exceeding 1.5 times the interquartile range (IQR), violating the assumptions of a paired  $t$ -test]. Signal amplitude was significantly higher in CAS- (median = 8.31 dB SPL) than CAS+ (median = 5.27 dB SPL),  $z = 4.4325$ ,  $p < 0.0001$ , effect size ( $z/\sqrt{n}$ ) = 0.8093. There was no significant difference in noise floor amplitude between CAS- (median =  $-11.71$  dB SPL) and CAS+ (median =  $-11.27$  dB SPL),  $z = -0.8947$ ,  $p = 0.3709$ , effect size =  $-0.1633$ . These results suggest that the introduction of CAS significantly reduced TEOAE signal amplitude but did not significantly alter the recording noise floor, as expected.

Contralateral inhibition (expressed in dB) was normally distributed as assessed using a Shapiro-Wilk test of normality,  $W(30) = 0.972$ ,  $p = 0.597$ . This result is consistent with the findings of Backus and Guinan (2007). Additionally, no outliers were present as indicated by all data points falling within  $\pm 1.5$  times the IQR. Mean contralateral inhibition was 3.48 dB (SD = 2.93). The median was 2.93 dB (IQR = 1.42–5.86) and the range was  $-1.71$  to 10.09 dB. It should be noted that negative values indicated that TEOAE amplitude increased rather than decreased in the presence of CAS. Three participants had negative contralateral inhibition values, consistent with previous reports showing a minority of participants with negative inhibition (Hood *et al.*, 1996; De Ceulaer *et al.*, 2001; Mertes, 2018). Results suggested that there was an adequate

distribution of contralateral inhibition values across participants for correlating with the speech-in-noise results.

### B. Speech-in-noise testing

At each SNR, scores on the CRM were quantified for both the CAS+ and CAS- conditions. For each participant, a psychometric function was also computed for the CAS+ and CAS- conditions, allowing us to compare the threshold [SNR yielding a score of 50 RAU, which in this case is equivalent to 50% correct (see Table III of Studebaker, 1985)] and slope of the psychometric function between CAS+ and CAS- for each participant. Additionally, the correlations between scores and the magnitude of contralateral inhibition were examined.

Group data for scores at each SNR are displayed as box and whisker plots in Fig. 2. Scores improved with increasing SNR in a predictable manner. Results also show that floor effects (RAU score of  $-16.5$ ) were not reached at the lowest SNR, but ceiling effects (RAU score of 116.5) were present at the two highest SNRs. A two-way repeated measures analysis of variance (ANOVA) was considered to examine the effect of the factors SNR ( $-12$ ,  $-9$ ,  $-6$ ,  $-3$ , and  $0$  dB) and CAS condition (CAS+ and CAS-) on score. However, the assumptions of a repeated measures ANOVA were not met because there were multiple outliers present, and the studentized residuals were not normally distributed as assessed with a Shapiro-Wilk test of normality ( $p < 0.05$ ). Therefore, we analyzed the effect of CAS condition on score at each individual SNR using Wilcoxon signed-rank tests. This analysis is the nonparametric equivalent of the analyses run by Giraud *et al.* (1997), who computed paired  $t$ -tests between scores in CAS+ and CAS- at each individual SNR. Additionally, the primary outcome of interest in our analysis was the difference in score between CAS+ and CAS- at each individual SNR, since it was expected that score would improve with increasing SNR. The results of our comparisons at each SNR are shown in Table I. Results indicated that the median score was significantly higher in CAS+ than CAS- only at the lowest SNR of  $-12$  dB. These results were contrary to our predictions that the difference in scores

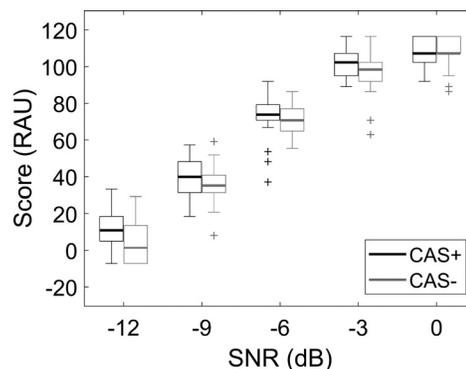


FIG. 2. Box and whisker plots of score across SNR for CAS+ and CAS- (left and right plots at each SNR, respectively). The thick horizontal lines represent the medians. Boxes encompass the 25th and 75th percentiles. Crosses represent outliers, defined as values that exceeded 1.5 times the IQR. Whiskers extend to the largest and smallest values not considered outliers.

TABLE I. Comparison of scores in CAS- versus CAS+ at each SNR. IQRs are shown in parentheses following the median scores. Results of Wilcoxon signed-rank tests ( $z$ ) are shown. The  $p$ -values were adjusted for the false discovery rate. Effect size was computed as  $(z/\sqrt{n})$ , where  $n = 30$ .

SNR (dB)	Median score in CAS- (RAU)	Median score in CAS+ (RAU)	$z$	$p$	Effect size
-12	1.58 (-7.21-13.56)	10.91 (4.99-18.47)	4.0240	0.0003	0.7347
-9	35.17 (31.27-40.83)	39.89 (31.27-48.18)	2.1863	0.0720	0.3992
-6	70.73 (64.83-77.02)	73.82 (70.73-79.24)	1.5372	0.2071	0.2807
-3	98.42 (91.93-102.35)	102.35 (95.01-107.21)	0.8213	0.5143	0.1499
0	107.21 (107.21-116.47)	107.21 (102.35-116.47)	0.4256	0.6704	0.0777

between CAS+ and CAS- would be significant at SNRs of -9 through -3 dB but not at -12 or 0 dB.

We next examined the association between score and the magnitude of contralateral inhibition. We examined the correlation between contralateral inhibition and the score in CAS-, as well as the correlation between contralateral inhibition and the difference in score (CAS+ minus CAS-), where positive values indicated improvement in score with the introduction of CAS. It is of note that previous studies have found significant correlations between contralateral inhibition and scores in CAS- (Abdala *et al.*, 2014; Bidelman and Bhagat, 2015; Mertes *et al.*, 2018) and contralateral inhibition and the difference in score (Giraud *et al.*, 1997; Kumar and Vanaja, 2004; Mishra and Lutman, 2014). Because scores were not normally distributed, Spearman rank correlation coefficients ( $r_s$ ) were computed. Statistical results of the correlations are shown in Table II, and scatter plots of score as a function of contralateral inhibition are shown in Fig. 3.

Results revealed that none of the correlations between score and contralateral inhibition were statistically significant, contrary to our predictions and a number of previous studies. The correlation between score and contralateral inhibition at -12 dB SNR approached significance (the unadjusted  $p$ -value was 0.0069 but was no longer significant after correcting for multiple comparisons). Several findings are apparent from the scatter plots. First, a trend of increasing score with increasing contralateral inhibition at -12 dB SNR can be seen. Second, the number of participants who experienced an improvement in score with CAS+ (points falling

TABLE II. Correlations between score and the magnitude of contralateral inhibition at each SNR. In the second column, the CAS condition “difference” refers to the difference in score (CAS+ minus CAS-), as analyzed in Kumar and Vanaja (2004) and Mishra and Lutman (2014). The third column displays Spearman rank correlation coefficients ( $r_s$ ) with the degrees of freedom shown in parentheses. The  $p$ -values were adjusted for the false discovery rate.

SNR (dB)	CAS condition	$r_s(28)$	$p$
-12	CAS-	0.4827	0.0690
	Difference	-0.0980	0.9177
-9	CAS-	-0.0051	0.9785
	Difference	0.0156	0.9785
-6	CAS-	0.1878	0.9177
	Difference	0.0979	0.9177
-3	CAS-	-0.0180	0.9785
	Difference	0.1689	0.9177
0	CAS-	0.0884	0.9177
	Difference	-0.0981	0.9177

above the dashed line in the bottom panels of Fig. 3) tended to decrease with increasing SNR. At an SNR of -12 dB, 22 participants improved their score with CAS+. However, at an SNR of 0 dB, only nine participants improved their score with CAS+. Additionally, there was a wide range of changes in score. Some participants’ scores decreased in the CAS+ condition, which is consistent with Giraud *et al.* (1997). It must be noted that we did not establish the test-retest reliability of the task, so it is not known how many changes in score were within test-retest variability. Finally, some participants reached ceiling scores (RAU = 116.5) at 0 dB SNR despite the RAU transformation of the data. However, no participants reached floor scores (RAU = -16.5).

It was also of interest to compare the slope and threshold of the psychometric function obtained in CAS+ and CAS-. Group mean psychometric functions are plotted in Fig. 4. The two functions had a similar shape with the function for CAS+ shifted slightly to the left. This suggested that the threshold (SNR yielding a score of 50 RAU) was lower in CAS+ than CAS- but that the two functions had similar slopes. We compared the threshold and slope values in CAS+ versus CAS-. A Shapiro-Wilk test of normality revealed that threshold in CAS+ and CAS- were not normally distributed ( $p < 0.05$ ). Additionally, there were multiple outliers present. Therefore, thresholds in the two conditions were compared using a Wilcoxon signed-rank test. The threshold in dB SNR was significantly higher in CAS- (median = -7.80) than CAS+ (median = -8.30),  $z = 3.2349$ ,  $p = 0.0012$ , effect size = 0.5906. The results were consistent with our prediction. A Shapiro-Wilk test of normality revealed that the slopes for CAS+ and CAS- were normally distributed ( $p > 0.05$ ). Additionally, no outliers were present. Therefore, slopes in the two conditions were compared using a paired  $t$ -test. The slope in RAU-per-dB was not significantly different between CAS- [mean = 12.57, SD = 2.35] and CAS+ (mean = 12.08, SD = 1.96),  $t(29) = 1.0422$ ,  $p = 0.3060$ , effect size (Cohen’s  $d$ ) = 0.1903.

We next examined the association between contralateral inhibition with the slope and with the threshold of the psychometric function, following the recommendations of Mertes *et al.* (2018). Scatter plots of these associations are shown in Fig. 5. As described above, contralateral inhibition was normally distributed and did not contain outliers. However, thresholds in CAS- and CAS+ were not normally distributed and contained outliers, so we computed Spearman rank correlation coefficients. Contralateral inhibition magnitude was significantly negatively correlated with

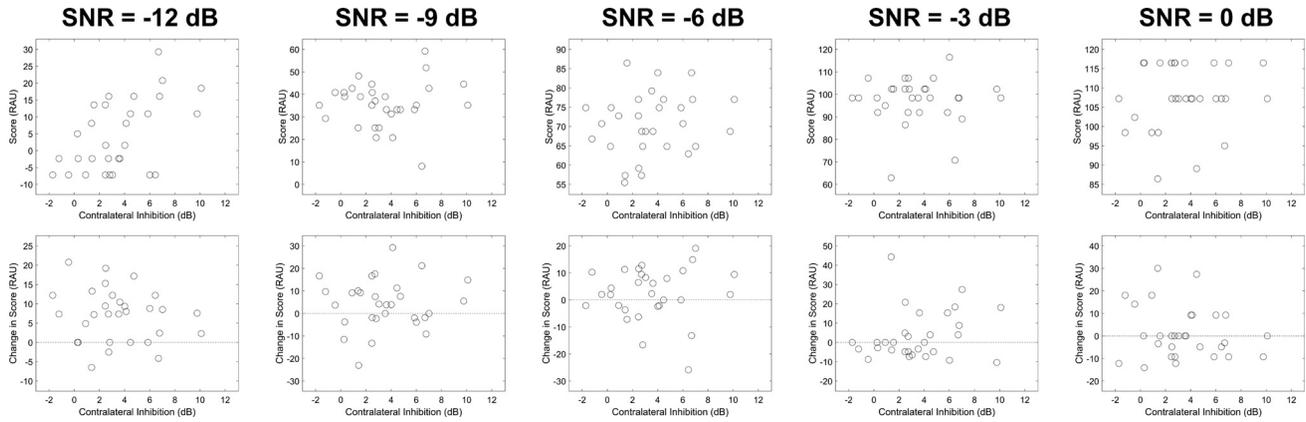


FIG. 3. Scatter plots of CRM score as a function of contralateral inhibition. Each column displays results for a different SNR. The top row displays score in the CAS- condition on the y axis. The bottom row displays the difference in score (CAS+ minus CAS-) on the y axis. Open circles represent data points from individual participants. The dashed horizontal lines on the bottom row demarcate improvements (points falling above the line) versus decrements (points falling below the line) in score. Note that the scale of the y axis limits is different across panels.

the threshold for CAS-,  $r_s(28) = -0.3837$ ,  $p$ -adjusted = 0.0364, and with the threshold for CAS+,  $r_s(28) = -0.4435$ ,  $p$ -adjusted = 0.0282. These results indicated that increases in contralateral inhibition were associated with lower thresholds and were consistent with our predictions. Because slopes were normally distributed and did not contain outliers, we computed Pearson correlation coefficients ( $r$ ) to assess the association between slope and contralateral inhibition. Contralateral inhibition magnitude was not significantly correlated with the slope of CAS-,  $r(28) = -0.2326$ ,  $p$ -adjusted = 0.4323, or with the slope of CAS+,  $r_s(28) = -0.0837$ ,  $p$ -adjusted = 0.6600.

### C. Response time

For each participant, response times across the 50 trials were reduced to the median value due to the presence of outliers. Box and whisker plots of the distribution of median response times across participants are shown in Fig. 6. Median responses times, as well as the variability in response time across participants, decreased with increasing SNR, consistent with previous work (Sarampalis *et al.*, 2009; Houben *et al.*, 2013). One participant had an unusually short median response time (95.7 ms) at 0 dB SNR in CAS+, shown as the outlier falling below the lower whisker in Fig. 6. Examination of the raw data revealed that there were no errors in data coding, and so this data point was included. There were no significant differences in median response

time between the CAS+ and CAS- conditions at any SNR, as assessed with Wilcoxon signed-rank tests (adjusted  $p > 0.05$  in all cases). Additionally, response time was not significantly correlated with the magnitude of contralateral inhibition in either the CAS+ or CAS- condition, as assessed with Spearman rank correlations (adjusted  $p > 0.05$  in all cases). These results were contrary to prediction.

## IV. DISCUSSION

### A. Mechanisms that may underlie MOCR involvement in hearing in noise

It has long been speculated that the MOCR is implicated in aiding listening in background noise. One putative mechanism is antimasking of transient signals in the presence of noise. Experimental MOCR activation in animals reduces the impact of background noise on neural encoding of transient stimuli (Winslow and Sachs, 1987, 1988; Kawase

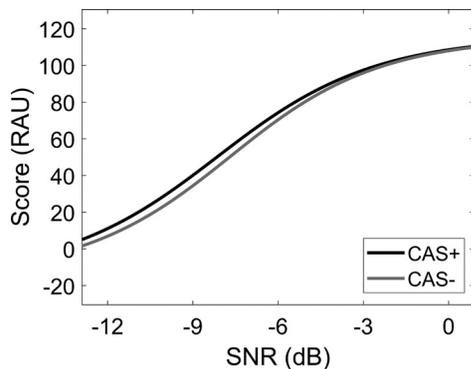


FIG. 4. Mean psychometric functions for the CAS+ and CAS- conditions.

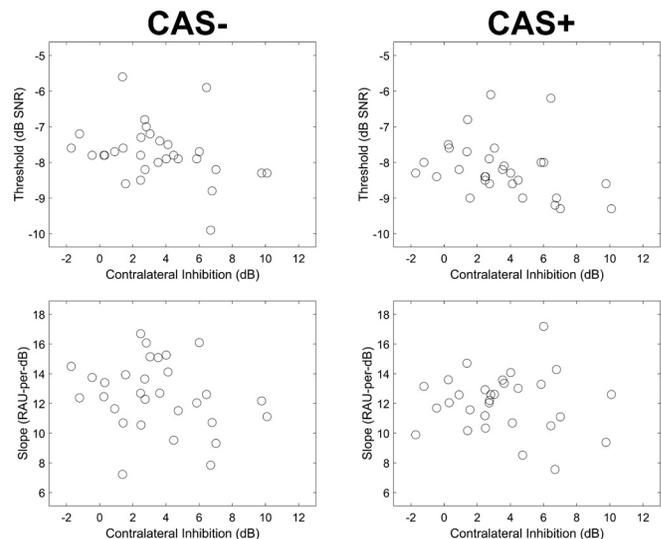


FIG. 5. Scatter plots of threshold as a function of contralateral inhibition (top row) and slope as a function of contralateral inhibition (bottom row). Left and right columns display results in the CAS- and CAS+ conditions, respectively. Open circles represent data points from individual participants.

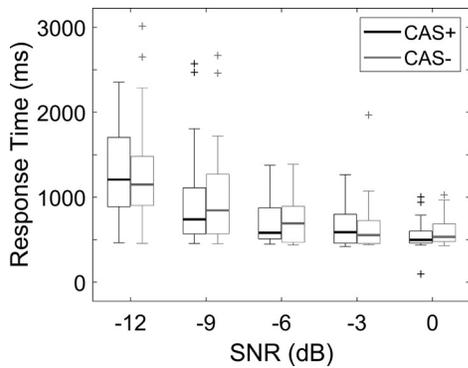


FIG. 6. Box and whisker plots of median response time across SNR for CAS+ and CAS-. Figure format is identical to that of Fig. 2.

*et al.*, 1993). These studies demonstrated that auditory nerve fiber firing rates in response to signals in noise were less saturated with MOCR activation compared to without MOCR activation. Speech signals in the presence of noise may benefit from such an effect because of the differential effect of the MOCR on steady-state versus transient sounds, where the MOCR reduces responses to steady-state sounds (e.g., the noise) more than the rapidly fluctuating speech signals (Lieberman and Guinan 1998). Surgical lesioning of the MOC bundle in animals impaired discrimination of vowel sounds (Dewson, 1968; Hienz *et al.*, 1998), possibly due to removal of the antimasking effect. Antimasking can be conceptualized as an increase in the SNR. For a speech perception task, a small increase in SNR (e.g., 3 dB) improves performance on the CRM task by at least 20%, at least at the low end of the psychometric function (Brungart, 2001; Eddins and Liu, 2012). This suggests that the MOCR may improve speech perception in noise through an increase in the SNR.

MOCR activation may also contribute to improved speech recognition in noise via intensity discrimination. As discussed in Sinnot *et al.* (1985), intensity discrimination is important for recognizing stop consonants and formant transitions. In humans, stronger MOCR activity was associated with smaller difference limens for pure-tone intensity discrimination (Micheyl *et al.*, 1997). Surgical lesioning of the MOC bundle impairs intensity discrimination of sinusoids presented in noise (May and McQuone, 1995). An additional mechanism may be through changes to basilar membrane input-output functions, which become more linear with MOCR activation. Marrufo-Pérez *et al.* (2018a) discussed that such an effect could allow the basilar membrane to follow the dips and peaks in amplitude-modulated waveforms and improve encoding of such signals (see Fig. 1A in Marrufo-Pérez *et al.*, 2018a). Modulations in the waveform of speech carry important information on place, manner, and voicing (Rosen, 1992). The MOCR therefore may aid listening in noise through one or more mechanisms that benefit perception of complex signals such as speech.

We found that introducing CAS significantly improved score at an SNR of  $-12$  dB and significantly lowered the threshold of the psychometric function. Additionally, we found that threshold of the psychometric function was significantly negatively correlated with contralateral inhibition

(i.e., lower thresholds were associated with stronger inhibition). These findings appear to be consistent with an improvement in SNR that may be mediated by antimasking. However, our use of sentence materials do not allow for an examination of how the MOCR impacts intensity discrimination, frequency discrimination, and temporal cues in isolation. We intend to examine how the MOCR contributes to perception of these variables in isolation in our future work.

## B. Comparison with studies supporting MOCR involvement in hearing in noise

Our results, coupled with those of other human experiments, provide support for the hypothesis that the MOCR contributes to speech-in-noise perception. The current results suggest that participants with stronger MOCR activity performed better on the task than participants with weaker MOCR, at least at more challenging SNRs, and is broadly consistent with the conclusions of other human studies (Giraud *et al.*, 1997; Kumar and Vanaja, 2004; Abdala *et al.*, 2014; Mishra and Lutman, 2014; Bidelman and Bhagat, 2015; Maruthy *et al.*, 2017; Mertes *et al.*, 2018). Additionally, our contralateral inhibition results were consistent with a number of studies (e.g., Collet *et al.*, 1990; Backus and Guinan, 2007). A minority of participants showed increases rather than decreases in TEOAE amplitude in the presence of contralateral noise. Such decreases are consistent with previous work and may be due to the MOCR increasing the impedance discontinuities in focused areas of the cochlea, which would cause an increase in reflection-source OAE amplitudes in some participants (Berezina-Greene and Guinan, 2017).

However, some of the specific findings of our study were in contrast to results of previous studies. We found that CAS did not significantly change scores at SNRs besides  $-12$  dB. Giraud *et al.* (1997) constructed psychometric functions for monosyllabic words in noise for SNRs from  $-30$  to  $+20$  dB, which resulted in mean scores ranging from 0% to 100% correct, respectively. They found that introducing CAS significantly improved score in the middle range of their SNRs, but not at the most or least challenging SNRs (see Fig. 1 in Giraud *et al.*, 1997). It may be possible that at very poor SNRs, elicitation of the MOCR is insufficient to improve perception. However, our psychometric functions also ranged from approximately 0% to 100%, so it was unexpected that the only significant change was at the most challenging SNR. Kumar and Vanaja (2004) found that CAS significantly improved monosyllabic word recognition in noise for the lowest SNRs they examined. However, they also found that the change in word recognition (CAS+ minus CAS-) was significantly correlated with contralateral inhibition. Similar findings were reported by Mishra and Lutman (2014). We did not find that the change in score was significantly correlated with contralateral inhibition, but rather the score in CAS- was correlated with contralateral inhibition. It is unclear why these specific results were in contrast to the findings of other studies, but it is possible that the differences in speech tasks, ipsilateral and contralateral noises, contralateral inhibition measurement and analysis,

and/or participants yielded the discrepant findings. Additionally, it is possible that simple subtraction of scores obtained without versus with contralateral MOCR activation is an oversimplification of a nonlinear system. Lilaonitkul and Guinan (2009) found that contralateral and ipsilateral inhibition of 1 kHz stimulus frequency OAEs were similar when elicited with a broadband noise (the same noise in each ear). However, it should be noted that we presented different stimuli to each ear. Further work is needed to optimize comparisons of speech-in-noise performance without versus with contralateral MOCR activation.

Mertes *et al.* (2018) recently reported that the magnitude of contralateral inhibition was not significantly correlated with score on two speech-in-noise tasks (including the CRM), but it was correlated with the slope computed across two SNRs. We expanded upon this study by including a broader range of SNRs and measuring performance on the CRM with ipsilateral masking noise and bilateral noise. We predicted that contralateral inhibition would be significantly correlated with the slope, but we did not find a significant correlation. There were several methodologic as well as participant differences between the current study and Mertes *et al.*, including participant age and hearing status. Our participants were young adults with normal hearing, whereas the participants of Mertes *et al.* were primarily older adults with mild high-frequency sensorineural hearing loss. Age and hearing loss both impact contralateral inhibition (Lisowska *et al.*, 2014) and speech perception in noise (Helfer and Wilber, 1990), so direct comparisons between studies should be interpreted with caution.

Because we constructed psychometric functions across a range of SNRs, this allowed us to estimate threshold of the psychometric function. Mertes *et al.* (2018) only measured performance across two SNRs and some of their participants did not reach 50% correct performance at the highest SNR (see Fig. 4 in Mertes *et al.*, 2018), so threshold could not be estimated. Visual inspection of the results of Giraud *et al.* (1997) suggest that the threshold, but not the slope, of the psychometric function is altered by introduction of CAS (see Fig. 1 in Giraud *et al.*, 1997), but the authors did not correlate these psychometric measures with their contralateral inhibition results. Additional study is needed to establish the best metrics of how the MOCR is involved in speech-in-noise performance.

### C. Evidence against MOCR involvement in hearing in noise

Taken together, the studies discussed above provide support that MOCR activity is associated with speech understanding in noise. Despite these converging lines of evidence, there have been a smaller number of human studies that have shown no link and therefore warrant discussion. Scharf *et al.* (1997) examined multiple psychoacoustic tasks in patients who underwent unilateral vestibular neurectomy, which consequently also sections the olivocochlear fibers located by the vestibular nerve. Damage to the MOCR was substantiated by a lack of contralateral inhibition in ears that

underwent surgery. Despite this, patients showed no significant difference on these tasks compared to a control group (except for a selective attention task that points to the corticofugal nature of the auditory efferent system). These data are consistent with the animal work of Trahiotis and Elliott (1970) and Igarashi *et al.* (1972), who found no significant effect of lesioning the MOC bundle on masked thresholds in cats.

The important limitation of these studies was that the psychoacoustic signals of interest were tones rather than complex stimuli. Igarashi *et al.* (1972) discussed that pure-tone stimuli may not have been sufficient activators of the MOCR. Studies that utilized more complex sounds such as vowels showed that experimental lesions of the MOC bundle negatively impacted discrimination (Dewson, 1968; Hienz *et al.*, 1998). Additionally, human vestibular neurectomy patients did not show an improvement in monosyllabic word recognition in the presence of CAS, whereas a control group did show improvement that was attributed to the MOCR (Giraud *et al.*, 1997).

It appears that the use of complex versus tonal stimuli alone does not account for the discrepant findings. Several human studies have found no significant correlation between the magnitude of contralateral inhibition and speech-in-noise performance. Wagner *et al.* (2008) found no significant correlation between speech reception thresholds for sentences in noise and contralateral inhibition measured using distortion product otoacoustic emissions (DPOAEs). Contralateral inhibition was measured at frequencies in which the distortion and reflection components of the DPOAEs (Shera and Guinan, 1999) were in cancellation (a “dip” frequency). However, this method has been criticized as assessing MOCR-induced changes in DPOAE phase due to a differential effect on the distortion versus reflection components (Abdala *et al.*, 2009), so these measures may not have captured the MOCR effect at frequencies where the distortion and reflection components were in phase.

Mukari and Mamat (2008) found that adults aged 50–60 yr had significantly lower contralateral inhibition of DPOAEs, as well as poorer reception thresholds, for sentences in noise compared to adults ages 20–30 yr. However, there was no significant correlation between the contralateral inhibition values and speech reception thresholds. Individual data were not shown, but the results may suggest that contralateral inhibition and speech perception are related but not in a linear fashion. Stuart and Butler (2012) reported that contralateral inhibition was not significantly correlated with reception thresholds for sentences in noise measured in the presence of ipsilateral and bilateral noise. The authors discussed that the relationship between contralateral inhibition and speech performance appears to be highly dependent upon stimulus parameters. de Boer and Thornton (2008) found a significant association between contralateral inhibition and discrimination of synthetic syllables /bi/ versus /di/ in noise, where larger inhibition was associated with better discrimination. However, this group later found that contralateral inhibition had the inverse relationship with discrimination of /ba/ versus /da/ in noise, where larger inhibition was associated with poorer discrimination (de Boer *et al.*,

2012). The authors speculated that differences in the stimulus spectra across the two studies may have contributed to the differing results. However, it is important to note that most other studies employing monosyllabic words or sentences, which would have a broad range of spectral characteristics, have still shown a positive correlation.

It is important to note that mechanisms other than the MOCR may explain improvements in speech perception in the presence of noise. [Marrufo-Pérez et al. \(2018b\)](#) compared the effect of ipsilateral noise on speech reception thresholds in normal-hearing individuals and cochlear implant users (other noise conditions were tested in the normal-hearing group only). Because cochlear implant users do not benefit from the MOCR, this comparison allowed the authors to infer the contribution of the MOCR to the results. Thresholds were measured in the presence of ipsilateral noise that was either turned on at the same time as the speech or turned on 300 ms prior to the speech. In both groups, thresholds were significantly improved when the noise preceded the speech. The magnitude of the improvement was not significantly different between groups. The authors discussed that auditory nerve adaptation to the presence of noise may explain their results since this process would be present in both normal-hearing listeners and cochlear implant users. Future work should consider the MOCR in addition to other central auditory mechanisms underlying the perception of speech in noise.

#### D. The MOCR and listening effort

We explored the effect of contralateral noise on response time on the CRM task as well as the association between response time and contralateral inhibition, which is to the authors' knowledge the first examination in the context of the olivocochlear efferent system. Although there was a trend of decreased median response time in the presence of CAS+ versus CAS- at some SNRs, the differences were not statistically significant. Furthermore, response time and contralateral inhibition were not significantly correlated. These results should be interpreted with caution because we did not provide any instruction regarding how quickly participants should respond. Previous studies measuring response time have included explicit instructions that participants should respond as quickly as possible ([Sarampalis et al., 2009](#); [Houben et al., 2013](#)).

#### E. Recommendations for future research

The conflicting results across studies suggest that the role of the MOCR for listening in noise may be highly dependent upon factors such as the SNR, which may influence the extent to which the MOCR does or does not provide a benefit for hearing in noise. We unexpectedly found that the MOCR contributed to perception at the lowest SNR we tested. Additional examination using low SNRs and step sizes smaller than 3 dB could provide further insight into the MOCR role for hearing in challenging listening conditions. We also recommend that test-retest reliability be established for the speech-in-noise task utilized in order to determine if

changes in score with versus without CAS fall outside this variability.

Regarding response time, we did not provide any specific instructions regarding how quickly participants should respond because this examination was added as an exploratory measure. Previous studies using response time as a metric of listening effort have instructed participants to respond as quickly as possible. Therefore, we recommend that future work into the association between MOCR inhibition and response time ensure that subjects are instructed to respond as quickly as possible.

Our TEOAE time analysis window of 8–18 ms excluded short-latency TEOAE components that occur soon after the stimulus onset (e.g., [Mertes and Goodman, 2013](#); [Sisto et al., 2015](#)). Additionally, we used a linear TEOAE extraction paradigm to capture the full MOCR effect on TEOAEs ([Guinan, 2006](#)). Visual inspection of our recordings in an IEC 711 ear simulator (Larson Davis, Depew, NY) and several human ears showed significant stimulus artifact in the first 5 ms that could obscure any short-latency TEOAEs. The MOCR inhibits short-latency components of stimulus-frequency OAEs ([Berezina-Greene and Guinan, 2017](#)), so the MOCR also likely inhibits short-latency TEOAEs. However, it is not known whether including these short-latency components in the analysis would have impacted the associations with speech-in-noise recognition examined in the current study. Future studies of the MOCR could include short-latency components through nonlinear TEOAE extraction methods (e.g., [Keefe, 1998](#)), although these methods will subtract out some portion of the MOCR effect on TEOAEs ([Guinan, 2006](#)).

Finally, we must note that the current study only focused on the auditory efferent system at the level of the brainstem and cochlea. There is an extensive corticofugal system that modifies output of subcortical and brainstem structures, which can modulate MOCR activity (for a review, see [Terreros and Delano, 2015](#)). Our speech-in-noise task likely invoked a combination of the MOCR and the corticofugal effect on the MOCR, although we only quantified the MOCR effect. Characterizing the corticofugal contributions may allow for better predictions of speech-in-noise abilities. For example, one important function of the corticofugal network appears to be aiding in selectively attending to sound stimuli ([Terreros and Delano, 2015](#)), and selective attention abilities are correlated with speech-in-noise performance ([Strait and Kraus, 2011](#)). Quantifying the effects of selective attention on OAEs (e.g., [Wittekindt et al., 2014](#)) may allow for a more complete characterization of the auditory efferent system that could be combined with contralateral inhibition assessments of the MOCR.

#### V. CONCLUSIONS

The relationship between MOCR function and speech-in-noise perception was studied in a group of normal-hearing young adults. Presentation of contralateral noise significantly improved score at the lowest SNR. The correlation between score at the lowest SNR and magnitude of contralateral inhibition approached significance. Contralateral inhibition was

significantly correlated with the threshold of the psychometric function. Contralateral noise did not significantly change response time on the CRM task and response time was not correlated with contralateral inhibition. Our overall findings provide support for a role of the MOCR for listening in noise at poor SNRs. These results are consistent with previous work showing that the relationship between the MOCR and hearing in background noise appears to be highly dependent upon the listening situation.

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<sup>1</sup>Due to a programming error, three participants were inadvertently presented with the same randomized order of CRM test conditions: CAS- at 0 dB SNR; CAS- at -6 dB SNR; CAS+ at -9 dB SNR; CAS- at -9 dB SNR; CAS+ at -12 dB SNR; CAS+ at -3 dB SNR; CAS+ at 0 dB SNR; CAS+ at -6 dB SNR; CAS- at -3 dB SNR; CAS- at -12 dB SNR. Five additional participants were inadvertently presented with the same randomized order of CRM test conditions: CAS+ at -3 dB SNR; CAS+ at -9 dB SNR; CAS+ at 0 dB SNR; CAS- at -12 dB SNR; CAS+ at -12 dB SNR; CAS- at -6 dB SNR; CAS+ at -6 dB SNR; CAS- at -3 dB SNR; CAS- at -9 dB SNR; CAS- at 0 dB SNR. The error was corrected and did not affect the remaining participants. Visual inspection of the speech-in-noise scores revealed no apparent differences between these eight participants and the remaining participants, so the data were included. However, order effects cannot be ruled out entirely.

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