Systems/Circuits

Auditory Deprivation during Development Alters Efferent Neural Feedback and Perception

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Auditory experience plays a critical role in hearing development. Developmental auditory deprivation because of otitis media, a common childhood disease, produces long-standing changes in the central auditory system, even after the middle ear pathology is resolved. The effects of sound deprivation because of otitis media have been mostly studied in the ascending auditory system but remain to be examined in the descending pathway that runs from the auditory cortex to the cochlea via the brainstem. Alterations in the efferent neural system could be important because the descending olivocochlear pathway influences the neural representation of transient sounds in noise in the afferent auditory system and is thought to be involved in auditory learning. Here, we show that the inhibitory strength of the medial olivocochlear efferents is weaker in children with a documented history of otitis media relative to controls; both boys and girls were included in the study. In addition, children with otitis media history required a higher signal-to-noise ratio on a sentence-in-noise recognition task than controls to achieve the same criterion performance level. Poorer speech-in-noise recognition, a hallmark of impaired central auditory processing, was related to efferent inhibition, and could not be attributed to the middle ear or cochlear mechanics.

Key words: **deprivation; development; efferent; hearing; otitis media; perception**

Significance Statement

Otitis media is the second most common reason children go to the doctor. Previously, degraded auditory experience because of otitis media has been associated with reorganized ascending neural pathways, even after middle ear pathology resolved. Here, we show that altered afferent auditory input because of otitis media during childhood is also associated with long-lasting reduced descending neural pathway function and poorer speech-in-noise recognition. These novel, efferent findings may be important for the detection and treatment of childhood otitis media.

Introduction

Both good and bad auditory experiences can alter hearing. Conductive hearing loss (CHL) because of otitis media (OM) is a useful model for understanding how reversible auditory deprivation during development affects sound processing in the central auditory system. OM is also the second most common reason for children to go to the doctor (Center for Disease Control and Prevention, 2008). OM can attenuate the overall amplitude of the acoustic signal and alter the temporal fidelity of the input signal to the cochlea (Hartley and Moore, 2003; Lupo et al., 2011; Thornton et al., 2012). Several animal and human studies show degraded sound-evoked activity produced by OM, affecting neural representations of acoustic stimuli and perceptual ability during and after the resolution of the middle ear pathology (Xu et al., 2007; Takesian et al., 2009; Caras and Sanes, 2015; Zhuang et al., 2017; Thornton et al., 2021).

Most human studies show that temporary CHL, because of OM, is associated with a reduced ability to understand speech in the presence of background noise, even after the OM is resolved (Jerger et al., 1983; Gravel and Wallace, 1992; Schilder et al., 1994; Hall et al., 2003; Zumach et al., 2009). However, the neural substrate of deficits in perceptual masking is not yet clear. Studies using auditory brainstem responses show delayed wave III and/or V latencies and increased interwave intervals, indicative of immaturity in neural conduction in children with an OM history despite the resolution of the middle ear fluid (Chambers et al., 1989; Hall and Grose, 1993; Gravel et al., 2006; Borges et al., 2020). These findings suggest the negative effects of OM

Received Nov. 25, 2022; revised May 9, 2023; accepted May 14, 2023.

Author contributions: S.K.M. and D.R.M. designed research; S.K.M. performed research; S.K.M. and D.R.M. analyzed data; S.K.M. and D.R.M. wrote the first draft of the paper; S.K.M. and D.R.M. edited the paper; S.K.M. and D.R.M. wrote the paper.

This work was supported in part by National Institutes of Health, National Institute on Deafness and other Communication Disorders Grant R01DC018046 to S.K.M. and Grant R21DC019598 to D.R.M. D.R.M. was also supported by the National Institute for Health Research Manchester Biomedical Research Center. We thank Hansapanni Rodrigo for statistical consultation.

The authors declare no competing financial interests.

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https://doi.org/10.1523/JNEUROSCI.2182-22.2023

history on brainstem signal processing. However, only one study linked this abnormal brainstem physiology with binaural hearing deficits as measured by the masking level difference (Hall and Grose, 1993).

There are several auditory (subcortical and cortical) and nonauditory (cognitive) mechanisms that contribute to hearing in noise. An important mechanism that enhances the neural representation of transient sounds in the presence of background noise is the efferent auditory pathway. Specifically, the medial efferents descend from the superior olivary complex and project onto the cochlear outer hair cells, forming the last leg of the descending neural pathway, which originates in the auditory cortex (Fuchs and Lauer, 2019). The medial olivocochlear efferents reduce the gain of the cochlear amplifier and inhibit the auditory nerve response to background noise. This action partially restores the dynamic range of the neural response to rapidly changing sounds or acoustic transients (Winslow and Sachs, 1988; Kawase and Liberman, 1993; Kawase et al., 1993; Guinan, 2006). In humans, otoacoustic emissions (OAEs) can be applied to measure the functioning of the brainstem efferent feedback circuitry, also called efferent inhibition or efferent strength. The efferent unmasking mechanism is robust in the juvenile population, enabling tone discrimination in noise and word-in-noise recognition (Kumar and Vanaja, 2004; Mishra, 2020). Medial olivocochlear efferent fibers can modulate cochlear tuning in children (Mishra and Dinger, 2016). Cochlear tuning, sensitive to outer hair cell functioning, forms the basis of frequency selectivity in the auditory system (Evans et al., 1992; King, 1998).

Evidence from human studies suggests that the strength of the efferent unmasking mechanism can be altered by auditory training (Veuillet et al., 2007; de Boer and Thornton, 2008) or listening difficulties (Hunter et al., 2023). Likewise, Liberman et al. (2015) reported reduced lateral (but not medial) olivocochlear efferent innervation and a corresponding loss of afferent synapses in ears with CHL, compared with control ears, ~1 year from a tympanic membrane resection in a mature mouse model. However, the relationship between efferent dysfunction and degraded auditory experience during development, and its impact on perceptual deficits, remains to be elucidated.

In this study, we hypothesized that OM-induced degraded signal transmission through the developing auditory system alters the strength and functioning of the efferent system. Weakened efferent neural feedback would diminish ability to discriminate sounds in noise. To address this hypothesis, we measured speech-in-noise recognition and sound-evoked, medial efferent inhibition of OAEs in children with documented medical histories of degraded auditory experience because of OM and age-matched controls. Both groups had clinically normal auditory function at the time of testing.

Materials and Methods

Participants. Children from the local school district were recruited through flyers. Children were also enrolled from clinical records and an existing database. A total 151 children were enrolled in this study (mean age = 8.1 years; range = 5.0-12.8 years; girls = 76). All children had 20 dB HL or better hearing thresholds at octave frequencies from 250 through 8000 Hz and normal, Type A tympanograms (Jerger, 1970). In addition, wideband absorbance, measured using a HearID system (Mimosa Acoustics), was within the reported normative range for every child (Mishra et al., 2017). No children had clinical histories of communication or behavioral disorders for which they were receiving an intervention, and none was receiving formal musical training. The study protocol was approved by the New Mexico State University Institutional Review Board. Participants' parents signed a written permission form for their children to participate in the study, and all children signed an assent form.

The Control group (n = 99, girls = 48; mean age = 8.02 years, SD = 2.30 years) had no significant history of middle ear disease, that is, two or fewer documented OM episodes since birth and no documented OM in the previous 1 year as shown from medical records and/or parent reports. Ideally, the Control group should have no OM. However, the chances of having no OM episodes in typically developing children are <3% during the first 5 years (Halliday and Moore, 2010). As a result, previous studies have accepted two or fewer OM episodes for enrollment in control groups (Hunter et al., 1996; Tomlin and Rance, 2014; Graydon et al., 2017). All members of the OM group (n = 52, girls = 28; mean age = 8.40 years, SD = 2.23 years) had a documented medical history of bilateral OM with flat or Type B tympanogram and CHL. The last recorded episode of OM occurred at a (mean) age of 3 years (range = 2-4 years) with a median prior history of five episodes. Poorer ear hearing thresholds ranged from 25 to 45 dB HL (mean = 31.3) at the time of the last OM episode (see Fig. 1*A*). Middle ear and audiometry history was collected from clinical records. Information regarding the last OM episode is somewhat incomplete, assuming that any episode after the last recorded one was not significant enough for a clinic visit. All test procedures were conducted in a sound-booth. The ear with a lower hearing threshold at 1000 Hz or the right ear, if thresholds were the same, was selected for running experimental procedures.

Stimulus frequency OAEs (SFOAEs) and contralateral stimulation. SFOAEs, recorded at low probe levels, optimally represent cochlear amplifier gain (Shera, 2004). In addition, among all OAE types, SFOAEs are most appropriate for measuring efferent inhibition (Guinan et al., 2003). However, SFOAE-based efferent assays can show large variations with probe frequency. This was avoided by consistently using a 1000 Hz center frequency within a one-octave band. Considering the non–frequency-specific nature of medial efferent innervation of the cochlea (Brown, 2014), efferents will be acting along the length of the basilar membrane when inhibition of SFOAEs is observed for the 1000 Hz band. Therefore, contralateral inhibition of SFOAEs at 1000 Hz center frequency can be considered as a marker of the efferent neural response across the entire cochlea (Hernández-Pérez et al., 2021).

Detailed equipment setup and calibration for recording swept-tone SFOAEs were previously described by Mishra and Talmadge (2018). The protocol for contralateral stimulation was similar to a previous study (Mishra et al., 2018). Stimulus generation and recording of signals from the ear canal were performed using the ER-10B+ microphone probe assembly (Etymotic Research) controlled via a MOTU 828× audio interface (MOTU) using custom-built programs on RecordAppX software (Long et al., 2008).

Target frequency range for recording SFOAEs was 707-1414 Hz to estimate efferent inhibition at 1000 Hz center frequency. This center frequency has a high SNR for SFOAEs (Abdala et al., 2018; Mishra and Talmadge, 2018), shows robust efferent effects (Mishra and Abdala, 2015) and is free from short-latency components for the probe level (Sisto et al., 2013). SFOAEs were measured via a suppressor paradigm using 40 dB probe and 60 dB suppressor tones. Suppressor frequency was 1.1 times higher than the probe, and both tones were swept from low to high at 0.188 octave/s. Phase was inverted for every other use of the suppressor. SFOAEs were recorded with and without continuous broadband noise presented at 60 dB SPL to the contralateral ear using an ER-2 insert phone. The contralateral interval had a 2 s lead and was interleaved with the probe sweep, with a 3 s gap between two successive sweeps. Children sat on a recliner and were asked to remain calm and quiet. They watched an age-appropriate, silent, close-captioned video during the OAE testing.

Estimates of magnitude, phase, and noise floor were obtained using least-squared filter modeling (Mishra and Talmadge, 2018). SFOAEs were estimated by minimizing the sum of the squared error between the model and the response. Noise floor was the average pairwise sweep difference between the probe-plus-suppressor and the probe alone. Efferent inhibition (%) was computed using a vector difference method that

considers the SFOAE phase and normalizes magnitude to baseline SFOAE to compute efferent inhibition for OAEs with SNR $\geq$ 6 dB (Mishra and Biswal, 2019). Absence of middle ear muscle reflex effects was ensured by using a group delay test with a 4 ms criterion (Mishra and Biswal, 2019). SNR criterion was not met in 4 children, and data from 2 children failed both the group delay test and SNR criterion.

Hearing in Noise Test for Children (HINT-C). Speech recognition threshold (SRT) was measured monoaurally using the American HINT-C (Nilsson et al., 1996). The HINT-C is based on 13, ten-sentence lists that are phonemically balanced. The test was implemented in a Windows laptop with a USB sound card (Scarlett 2i2, Focusrite) and TDH39P headphones. Speech and noise were presented diotically, producing a colocated percept in the head midline. The noise level was matched to the long-term-average spectrum of the sentences and fixed at 65 dB SPL. Speech level was varied adaptively to achieve target SNR. Starting SNR was 10 dB, with an initial step size of 5 dB for the first four trials, reducing to 3 dB. Each run included two distinct sentence lists. The child was instructed to repeat the entire sentence correctly. Scoring was based on correctly reported complete sentences allowing minor variations in articles and verb tenses. SRT was defined as the mean SNR from trials 5-21. The 21st sentence was not presented, but its SNR was predicted from the previous response.

Each child was presented with a practice run for familiarization, followed by two test runs. Thresholds from those two runs were averaged to get an estimate of SRT. If SRTs for the two runs differed by >10%, a third run was used, and the two lowest scores were averaged. The participants were native speakers of American English. Only 100 (Controls = 65) of 151 children were able to complete the HINT-C. Notably, 19 (Controls = 14) of 46 (Controls = 33) 5- to -6-year-olds reliably completed the test.

Experimental design and statistical analysis. Statistical analyses were conducted using Jamovi software (version 2.3.18). Group differences (Control and OM) were compared using Welch's *t* tests. Analysis of covariance (ANCOVA) with age (log-transformed) as a covariate was conducted to compare group differences for efferent inhibition and SRT. Effect sizes (η^2) for ANCOVA were interpreted according to Miles and Shevlin (2001). Hierarchal linear regression was used for modeling efferent inhibition and SRT. All reported model predictors have variance inflation factors <3. An effect was considered significant if $p \leq 0.05$. Missing data were not imputed.

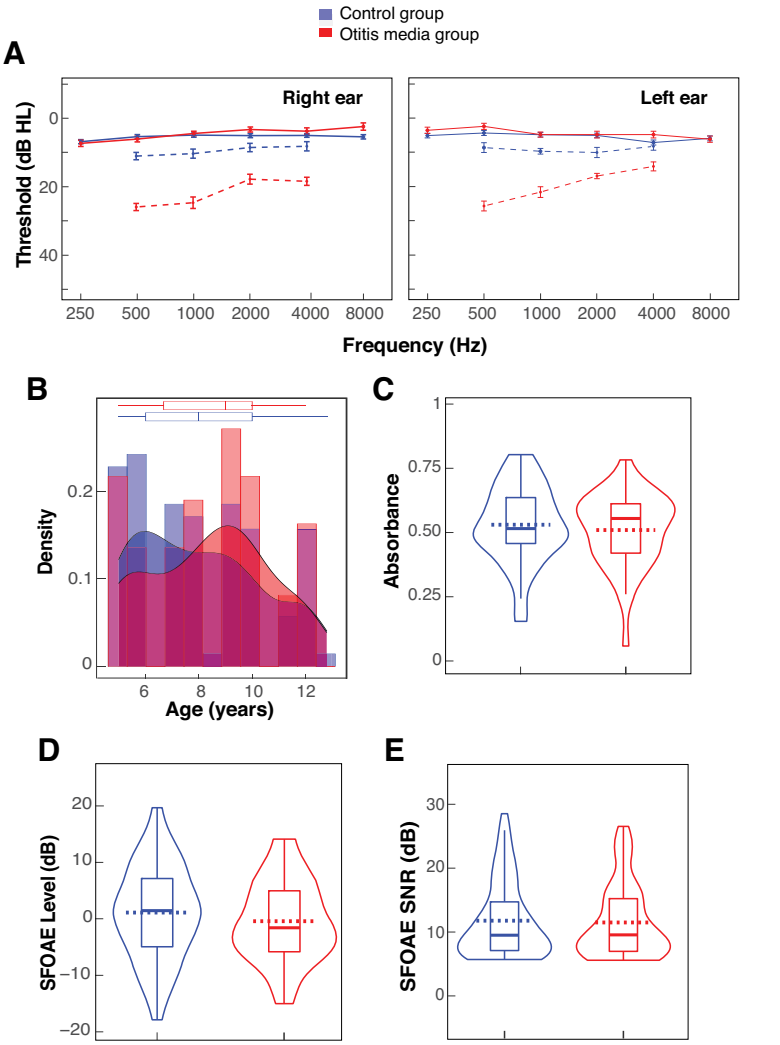

Figure 1. Auditory function in the Control and OM groups. ***A***, Mean pure-tone audiometric thresholds plotted separately for right and left ears. Error bars indicate SD. Solid lines indicate thresholds at the time of testing from all children (Controls: $n = 99$; OM group; $n = 52$). Broken lines indicate thresholds at last OM episode (OM group; $n = 16$; mean age = 3 years) and an equivalent time for Controls ($n = 14$). ***B***, Density plot for age distribution for the two groups with box-whisker plots on the top. ***C***, Violin plots for middle ear absorbance for the control and OM groups. ***D***, Violin plots for SFOAE level. ***E***, SFOAE signal-to-noise ratio for the control and OM groups. For all violin plots, violins represent kernel probability density, boxes represent interquartile ranges (with median and mean as solid and broken lines, respectively), and whiskers represent 1.5 times the interquartile range.

Results

Peripheral auditory characteristics at the time of testing

Hearing sensitivity, middle ear function, and cochlear function were normal in all children at the time of testing (Fig. 1). There was no significant age difference between Control and OM groups (Welch's $t_{(106)} = -0.99$, $p = 0.32$). Hearing thresholds for all audiometric frequencies were $\leq$ 20 dB HL for all children, and there were no significant group differences for any frequencies or ears ($F_{(1149)} = 1.33$, $p = 0.25$). Wideband absorbance (in ratio) did not differ significantly between the groups (control: mean = 0.53, SD = 0.15; OM: mean = 0.51, SD = 0.14; Welch's $t_{(77)} = 0.69$, $p = 0.49$). For brevity and relevance to efferent measurements, absorbance results are presented for 1000 Hz center frequency with one-octave band. However, the two groups did not differ significantly across the measured frequency range (250-6000 Hz). The mean and range of variation in wideband absorbance across children in both groups are consistent with a previous normative study (Mishra et al., 2017).

SFOAE data from six Control children did not meet SNR criterion. For the remaining children, mean SFOAE magnitudes were not significantly different between groups (Fig. 1*D*; control: mean = 1.12, SD = 8.28; OM: mean = −0.41, SD = 7.09; Welch's $t_{(120)} = 1.17$, $p = 0.24$). Likewise, there was no significant difference in SNR for SFOAEs between the two groups (Fig. 1*E*; control: mean = 11.78, SD = 9.50; OM: mean = 11.49, SD = 9.56; Welch's $t_{(108)} = 0.28$, $p = 0.78$).

Effect of OM history on efferent inhibition and SRT

Efferent inhibition (Fig. 2*A*) for the OM group was significantly lower than for the Controls (ANCOVA with a fixed factor of group and age as a covariate; $F_{(1142)} = 7.11$, $p = 0.01$, $\eta^2 = 0.05$). However, the effect size was small. No significant effect of age was observed (age: $F_{(1140)} = 0.61$, $p = 0.44$). Although no

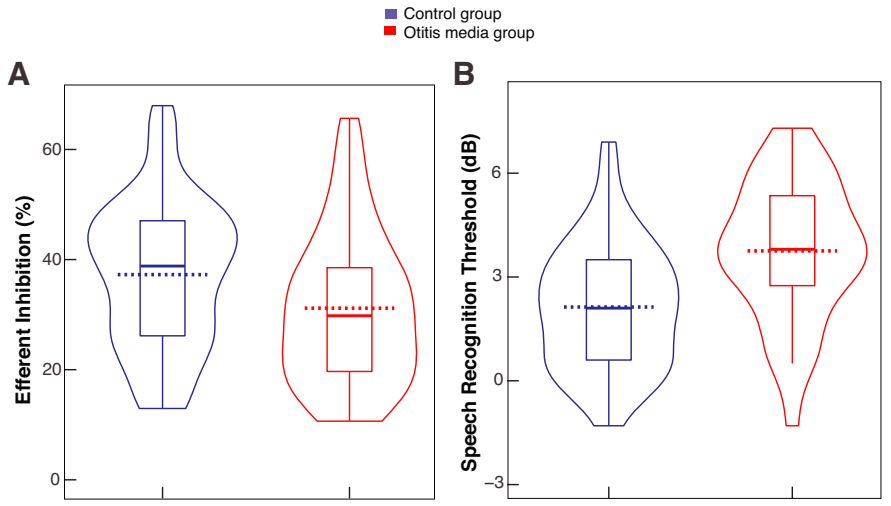

Figure 2. Efferent neural inhibition and speech-in-noise recognition. ***A***, Violin plot of efferent inhibition for control and OM groups. ***B***, Violin plot of SRT for control and OM groups. Violin plots as per Figure 1.

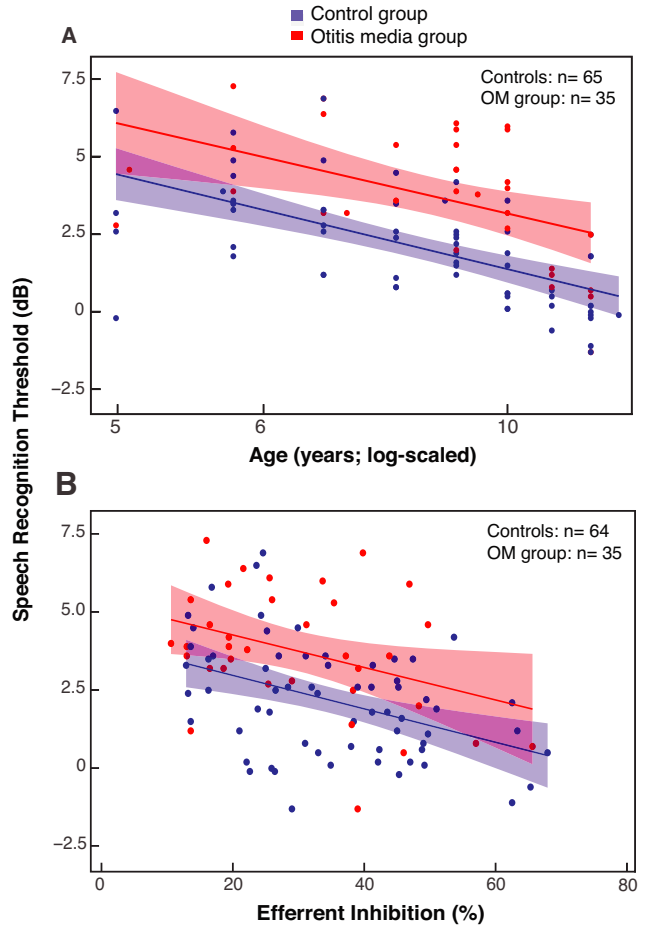

Figure 3. Relationship between efferent inhibition and speech-in-noise recognition. ***A***, SRT as a function of age with 95% CIs as shaded areas. Blue and red represent the control and OM groups, respectively, based on the linear regression model outcomes (Table 1). ***B***, SRT as a function of efferent inhibition. Same display parameters as in ***A***. Data related to Figure 3*B* are included in Extended Data Figure 3-1.

significant difference between the two groups in middle ear transmission (absorbance) or cochlear function (SFOAE) was observed, linear regression including wideband absorbance, SFOAE level, and SFOAE SNR was conducted to ensure these factors did not contribute significantly to efferent inhibition. The model was not significant ($F_{(3,96)} = 0.36$, $p = 0.77$, $R^2_{adjusted} = -0.02$), confirming that neither middle ear nor outer hair cell functioning at time of testing could explain the observed efferent inhibition effects of OM.

Like most speech-in-noise tests, developmental improvements in SRT have been observed for the HINT-C (Porter et al., 2013). To ensure that differences between the two groups can be truly attributed to OM history, age was input as a covariate to the ANCOVA. The OM group had higher (poorer) SRT relative to Controls (Fig. 2*B*), after controlling for age differences (ANCOVA: Group: $F_{(1,97)} = 30.2$, $p < 0.001$; $\eta^2 = 0.17$; age: $F_{(1,97)} = 50.5$, $p < 0.001$; $\eta^2 = 0.28$), with medium effect sizes. The improvement in SRT with age did not differ statistically between groups, as the interaction between age and group was not significant (Fig. 3; ANCOVA: age × group: $F_{(1,96)} = 0.0001$, $p = 0.99$). Figure 3 also plots the relationship between efferent inhibition and SRT (see the SRT modeling section).

To examine whether the duration of normal auditory experience following resolved OM is related to efferent inhibition and SRT in the OM group, separate partial correlations were conducted, controlling for age. Duration following resolved OM (mean = 5 years) was significantly related to SRT (Fig. 4; $r = -0.35$, $p = 0.02$, $n = 35$) but not to efferent inhibition ($r = 0.07$, $p = 0.31$, $n = 51$).

Modeling speech-in-noise recognition

There was a significant age-adjusted correlation between efferent inhibition and SRT (Fig. 3; $r = -0.41$; $p < 0.001$, $n = 99$; Fig. 3; see also Extended Data Fig. 3-1). The two groups were combined to obtain a robust correlation coefficient since a separate model with efferent inhibition × OM group as a predictor was not statistically significant. Having established relationships between efferent inhibition and SRT on one hand, and OM history and SRT and efferent inhibition on the other (Fig. 3), we asked how OM and efferent inhibition combine to account for the variance in SRT. Hierarchical linear regression was performed with SRT as the response variable. In Step 1, the predictors were demographic variables (age and sex); in Step 2, the (nonefferent) peripheral predictors absorbance and OAEs were added; in Step 3, OM history was added; and finally, in Step 4, efferent inhibition was added. Steps 1, 3, and 4 each produced a significant increase in ΔR^2. Intermediate models are presented in the Extended Data (Extended Data Table 1-1). The final model was significant ($F_{(6,66)} = 12.9$, $p < 0.001$, $R^2_{adjusted} = 0.49$), and the model outcomes are presented in Table 1. This model thus explained 49% of the variance observed in the SRT. SRT decreased (improved) with age and level of efferent inhibition and increased (poorer) with OM history. Middle ear, cochlear functioning, and sex effects were not significant.

Discussion

The results of the present study provide new evidence that early OM predicts later speech hearing deficits in noisy environments,

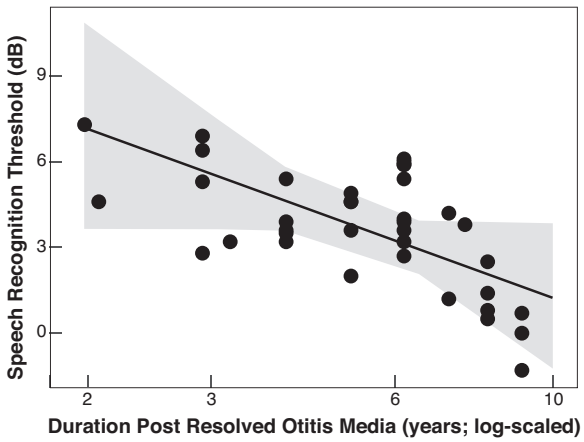

Figure 4. Relationship between time following resolved OM and SRT. Scatterplot represents the age-adjusted relationship between duration and SRT. Line equation: SRT = 7.60 − 8.51 × log(noOM duration) + 2.29 × log(age). Shaded areas represent 95% CIs.

and that medial olivocochlear efferent dysfunction contributes to those deficits. We found significantly lower efferent inhibition and poorer speech-in-noise recognition in children with documented OM history relative to controls. Using standard audiometry, wideband absorbance, and OAEs, we showed that peripheral auditory function was normal and indistinguishable between the two groups, confirming a central origin for the deficits attributable to OM.

Several studies have shown poorer signal detection and discrimination in noise following OM even after the middle ear disease is resolved (Jerger et al., 1983; Gravel and Wallace, 1992; Schilder et al., 1994; Hall et al., 2003; Zumach et al., 2009; Keogh et al., 2010; Gay et al., 2014; Ihlefeld et al., 2016). The neural mechanism by which auditory deprivation associated with OM increases the vulnerability to perceptual masking has not previously been systematically investigated, although it has generally been attributed to neural reorganization in the ascending auditory pathway (Whitton and Polley, 2011).

Methodological considerations

A potential limitation of using clinical records is that the number of OM episodes may not be precise. A prospective, longitudinal, monthly tympanometric assessment of OME from birth to 6 years (see Halliday and Moore, 2010) found that at least half of a quasi-randomly selected sample of children ($n = 112$) had frequent episodes of OM in one or both ears, especially in the first 2 years. However, only a small proportion of these cases would have been detected through infrequent, mostly episodic visits to health care providers. As a result, members of the present Control group may have had undetected asymptomatic OM. Likewise, the OM group may represent children with more frequent OM episodes who received medical intervention. Indeed, the threshold data at the "last OM" (Fig. 1*A*) are consistent with the audiometric profile of a high-prevalence OM subgroup (from Halliday and Moore, 2010) reported by Hogan and Moore (2003, their Table 1). A related issue is that threshold test data in the present study were not available for all episodes of OM, although thresholds from the last OM episode were available. Thus, even in a longitudinal design, it may not be feasible to measure behavioral thresholds reliably in infants and toddlers with OM. When the measurements are feasible, the test results may not be very sensitive to fluctuations that can occur with OM.

Table 1. Summary of the final regression model

Predictor	Estimate	SE	*t*	*p*	Standardized estimate
Intercept[a]	11.84	1.48	7.98	<0.001	
Age	−7.97	1.43	−5.56	<0.001	−0.48
Absorbance	−1.01	1.24	−0.82	0.416	−0.07
SFOAE	$-9.39e^{-4}$	0.02	−0.04	0.964	−0.004
Efferent inhibition	−0.05	0.01	−4.07	<0.001	−0.36
Group					
Controls–OM group	0.96	0.37	2.58	0.012	0.48
Sex					
Female–male	−0.33	0.34	−0.98	0.333	−0.17

[a]Reference level.

Further, only ~60% of children with OM experience mild to moderate CHL (Roberts et al., 2004). Whitton and Polley (2011) claimed that CHL, not the mere presence of OM, determines perceptual and physiological alterations in the auditory system. However, findings from a recent study refuted this claim for a tone-in-noise detection task (McKenna Benoit et al., 2022).

Experience-dependent changes in the efferent neural pathway

Corticofugal fibers originating from neurons in the auditory cortex exert control on the periphery via the olivocochlear efferents (Suga et al., 2000). Specifically, the olivocochlear efferent neurons modulate the afferent neural representation of the acoustic input. Evidence from mature animal models suggests that the brainstem efferent feedback circuitry is critically important for relearning sound localization following injury (Irving et al., 2011). However, very few studies have investigated the plasticity of the efferent neural system. Kraus and Illing (2004) showed that medial (not lateral) olivocochlear neurons are the major source of synaptic reorganization in the ventral cochlear nucleus after cochleotomy in adult rats. In human adults, de Boer and Thornton (2008) have shown that efferent neural strength and associated perceptual masking can be improved through targeted auditory training. Until recently, it was not known whether sound deprivation because of CHL alters olivocochlear efferents. Liberman et al. (2015) showed degeneration of the lateral efferent terminals following chronic CHL in adult mouse models. However, the functional consequences of alterations in lateral olivocochlear neurons are less known.

In this study, we carefully eliminated middle ear and cochlear factors in the efferent inhibition measurements, and demonstrated that children with OM in the first 3 years of life have weaker medial efferent inhibition relative to controls. Similar findings in humans have been reported for the acoustic reflex, another brainstem feedback pathway (Gravel et al., 2006). Although the magnitude of the difference in efferent inhibition between the OM and Control groups was small, a statistically small effect size can have important physiological consequences. For example, a small efferent effect is associated with a relatively larger release from adaptation in the auditory-nerve response to noisy speech, which can enhance speech intelligibility (Brown et al., 2010).

Lack of an age effect on efferent inhibition for children is not surprising, given that the medial efferent pathway is considered to be mature at full-term birth (Chabert et al., 2006; Abdala et al., 2013). In addition, we found no relationship between normal auditory experience following resolved OM and efferent inhibition,

suggesting that efferent dysfunction associated with temporary CHL in early childhood (<3 years) may not recover to normalcy even after 5 years of typical auditory experience.

Efferent control of the cochlear mechanics is reasonably well understood to predict the potential consequences of CHL. Theoretically, CHL because of OM would attenuate the sound level reaching the cochlea. As a result, the medial olivocochlear neurons would not receive optimal stimulation at a sound level for which the efferent action is known to be effective (Collet et al., 1990; Guinan et al., 2003). This may temporarily shift the dynamic range of the efferent neural response. Such altered efferent biomechanics during the early developmental period may take a longer recovery time or may not restore naturally after the OM is resolved. However, the reduced efferent inhibition could potentially be restored via targeted auditory training (de Boer and Thornton, 2008).

Potential mechanisms for perceptual consequences of OM

Despite firm evidence of poorer speech-in-noise recognition because of auditory deprivation associated with OM history or other CHL, the underlying neural mechanisms are less clear. Few previous studies used both neural/physiologic and perceptual measures for studying the neural substrate for poorer perceptual skills in children with OM history. Hall and Grose (1993) reported significant correlations between interaural asymmetries of the interwave intervals of auditory brainstem responses and the masking level difference. Their findings suggested a link between abnormal brainstem processing and binaural hearing deficits in children with OM history. We selected speech stimuli instead of tones as the effects of OM history for speech have been consistently demonstrated. Simpler tones may not reveal the human communication deficits associated with auditory deprivation because of OM, even when OM causes CHL (McKenna Benoit et al., 2022).

Although chronological age, efferent inhibition, and OM history combined explained 50% of the variance observed in the SRT, additional factors, such as central (afferent) auditory and cognitive processes, are likely involved in poorer speech-in-noise recognition in children with OM history.

The time course of the recovery of the neurophysiologic and perceptual effects of OM is unknown. However, the significant relationship between the time since resolved OM and SRT suggests that typical auditory experience (~5 years) following OM may slowly improve SRT. The improvement in SRT, but not efferent inhibition, with the period of normal auditory experience following resolved OM may suggest that children learn coping strategies and/or rely on additional mechanisms to filter out noise from the speech. This interpretation is also supported by substantial literature, suggesting that, in general, children with a history of OM do not sustain long-term auditory, cognitive, or academic deficits (Roberts et al., 2004).

Translational significance

Findings from the present study have implications for understanding the role of the efferent neural system in human hearing and related clinical applications. First, efferent inhibition may be reduced in other forms of developmental deprivation, such as minimal hearing loss and extended high-frequency hearing loss, which are associated with OM history (Hunter et al., 1996; Moore et al., 2020; Hunter et al., 2023). It could contribute to the perceptual deficits observed in such subclinical disorders. Second, medial olivocochlear efferents may play a role in preventing noise-induced hearing damage (Maison and Liberman, 2000). Since children with OM history have a weakened efferent system, they may be particularly vulnerable to future noise-induced cochlear damage or synaptopathy. Third, children with stronger medial efferent feedback may have relatively fewer consequences from auditory deprivation because of OM. This prediction is consistent with the role of efferents in hearing (perceptual and physiologic) development and plasticity (Walsh et al., 1998; Irving et al., 2011; Mishra, 2020). Related to this, enhancing the medial efferent feedback via pharmacological treatment or targeted auditory training could be a successful strategy to rescue and restore afferent neural encoding of sounds and minimize the short-to medium-term perceptual consequences of OM. Finally, medial efferent inhibition shows large interindividual variability in human adults and children (Backus and Guinan, 2007; Marshall et al., 2014; Mishra, 2020). Typically, the OM history is not considered in studies investigating medial efferent reflex in humans. Instead, peripheral auditory function is measured. Considering the deficiencies in efferent function associated with OM history, despite normal peripheral auditory function, we speculate that OM history may partly explain the variability in the efferent inhibition in normal-hearing individuals.

The mean SRT was ~2 dB poorer for children with OM history than controls, which corresponds to a ~15% deficit in speech-in-noise recognition scores according to psychometric functions measured in children, albeit for digits-in-noise (Moore et al., 2019). This perceptual deficit is highly relevant for classroom environments where background noise levels are often high (Crandell and Smaldino, 2000; Knecht et al., 2002) and may impact learning in the classroom.

In conclusion, children with an OM history had significantly poorer SRTs and reduced efferent inhibition, relative to controls. Medial efferent inhibition predicted the ability to understand speech in the presence of noise. These findings suggest that degraded auditory experience because of OM produces relatively long-standing reductions of efferent neural feedback. It also suggests medial efferent inhibition as a possible neural source of the speech-in-noise recognition deficits observed in children with OM history. The findings complement an extensive literature predicting perceptual effects of auditory deprivation because of central auditory degeneration.

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