Frequency Specificity of the Ipsilateral, Contralateral and Binaural Medial Efferent Reflexes in Humans

by

Watjana Lilaonitkul

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ABSTRACT

A variety of evidence indicates that the brain controls the gain of the cochlea in a frequency specific manner through the medial olivocochlear efferent pathway but the degree of frequency specificity in humans is poorly understood. To study the frequency specificity of the ipsilateral, contralateral and binaural medial efferent responses in humans, changes due to the presence of bands of noise in ear canal sound pressure of stimulus frequency otoacoustic emissions (SFOAEs) were recorded. Changes in the SFOAEs produced by 40 dB SPL test tones with frequencies near 1 kHz or at 2.6 kHz were monitored with a sensitive microphone in the ear canal. In the first paradigm, efferent activity was elicited by a octave band of noise at 60 dB SPL with the noise center frequency varied relative to the test tone frequency. The results show that the maximum efferent effect was for noise bands centered near, but not always at, the test frequency, and that in some cases noise bands centered as far as 2.5 octaves from the test frequency elicited significant response. Equal-response tuning curves at a test frequency of 0.77 kHz gave Q20 values which suggest that the width of efferent tuning is greater than that of afferents in humans. In a second paradigm, the bandwidth of the elicitor noise was varied from 1/8 to 4 octaves, keeping the energy level constant at 60 dB SPL and its center frequency (on a log scale) fixed at the test-tone frequency. The efferent response generally increases with increasing bandwidth, up to bandwidths of 2-4 octaves. Overall, the results show that the medial efferent acoustic reflexes in humans exhibit some frequency specificity, but also that they integrate acoustic energy over a wide range of frequencies that are much broader than has previously been thought.

Thesis Supervisor: John J. Guinan, Jr.
Title: Associate Professor in Health Science and Technology
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Background

A) Medial Olivocochlear Anatomy

The Medial Olivocochlear (MOC) efferents originate in the medial part of the superior olivary complex and terminate in the cochlea (Rasmussen, 1946, 1960). There, the MOC fibers synapse directly on outer hair cells (OH) and on the radial auditory nerve fibers beneath inner hair cells (Smith, 1961). The MOC pathway is binaural - meaning that MOC fibers can be activated by elicitor sounds presented to the same ear or the opposite ear (see figure 1). With regards to the strength of activation by ipsilateral versus contralateral sound simulation, studies done on anesthetized cats and guinea pigs show that roughly 2/3 of the MOC fibers are driven best by ipsilateral sounds while the other 1/3 are driven best contralaterally (Robertson and Gummer, 1985, Liberman and Brown, 1986, Brown 1989). Curiously enough, anatomically, it is known that in most species, MOC fibers project predominantly to the contralateral cochlea (Guinan, 1996). These fibers receive input from the contralateral cochlea and therefore produce the ipsilateral reflex.

Figure B1: Schematic illustration of the basic plan of the MOC pathways to the right ear. MOC neurons are effectors in acoustic reflexes driven from either the ipsilateral (black lines) or contralateral (gray lines) ears. Higher centers may act to modulate the strength of this reflex, or may activate MOC neurons directly, without changing the acoustic reflex.

MOC fibers are myelinated (Guinan, Warr, and Norris 1983) and almost all of the information on efferent physiology, obtained from studies done on anesthetized animals, is attributed to the MOC efferents rather than the lateral olivocochlear (LOC) efferents.
This is because the LOC efferents are unmyelinated. This makes their stimulation with extracellular currents significantly harder and almost impossible for recording to be done with high impedance pipet electrodes (Guinan, Warr, and Norris 1983). From anesthetized animal studies, which combined electrophysiology and tracer labeling, MOC efferents have been shown in to have tonotopic maps corresponding at least roughly to those of afferents (Robertson and Gummer, 1985; Liberman and Brown, 1986; Brown, 1989). Hence, it seems probable that efferents tuned to some particular frequency can receive signals from afferents tuned to similar frequencies – making the feedback efferent response frequency specific.

B) MOC reflex and the cochlear amplifier

Efferent stimulation has been shown to depress basilar membrane motion at low sound levels in guinea pigs with maximum depression around the center frequency of up to 20-22 dB in the region of 18-20 Hz (Dolan and Nuttall, 1994). In response to sound the basilar membrane motion causes the reticular lamina to shear radially relative to the tectotial membrane and bends the OHC stereocilia in the process. Ion channels open from this bending which results in an ionic flux and a change in membrane potential. OHCs are motile and can shorten and elongate as a result of a change in its membrane potential (Santos-Sacchi and Dilger, 1988). The OHC motility is somehow coupled back into the basilar membrane motion such that an in-phase coupling would amplify the forward traveling wave and vice versa. This process of OHC-induced amplification and suppression of the basilar membrane motion is also known as the ‘cochlear amplifier’ and is illustrated in figure B2 (adapted from Guinan, 1996). MOCs richly innervate and are thought to change the properties of OHCs. The exact mechanism of this efferent control is unknown.

C) Effects on Otoacoustic Emissions

Otoacoustic emissions (OAEs) are sounds that are produced within the cochlea, spontaneously or as a result of some stimulation, that can be recorded in the ear canal (Kemp, 1978). The phenomenon of OAEs is attractive for the studies of efferent responses because they can be measured non-invasively. An efferent-induced depression of the forward traveling wave amplitude along the basilar membrane results in a reduction in the energy reflected back in the backward traveling wave (Guinan, 1996). Hence, most of the existing literature on humans quantifies the amount of efferent activation as the suppression of some kind of evoked otoacoustic emission in the presence of an efferent elicitor.
Figure B2. (Adapted from Guinan, 1996) Schematic illustrating the presumed action of the cochlear amplifier at one place along the basilar membrane, and medial efferent inhibition of the cochlear amplifier. Trace A is a snapshot of normal basilar membrane motion in response to a tone. The cochlear amplifier at the circled place moves the basilar membrane at that place and creates waves that travel away in both directions (Trace B). The forward traveling wave (Trace B) adds to the sound-driven wave and amplifies it. Note that the amplitude scale is exaggerated in traces B and D relative to the traces A and C. For traces C and D, medial efferents are activated and reduce the gain of the cochlear amplifier so that the motion created by it is smaller (the amplitude is smaller in trace D than in trace B). Since there is less amplification at many places along the basilar membrane, the resulting traveling wave is less (the amplitude is smaller in trace C than in trace A).

D) Experimental Issues:

Stimulus Frequency Otoacoustic Emissions (SFEs) are used to assay the frequency resolution of the MOC reflexes applied to the contralateral ear, ipsilateral ear or both. This type of OAE corresponds to the generation of additional acoustic energy from the cochlea at the frequency of a low-level, constant tonal stimulus (Kemp and Chum, 1980a; Probst, et al. 1990). Quantitatively, at low to moderate sound levels, SFEs can be explained as arising from coherent scattering of the cochlear traveling waves off small irregularities or random perturbations in the cochlear mechanics (Zweig et al. 1995). Its magnitude and phase can be recorded non-invasively from the ear canal (Kemp and Chum, 1980a). One advantage of using SFE to monitor efferent activation is that the test tone itself elicits little or no efferent activity (Maison et al. 1999). Some existing literature uses the suppression of the 2f1-f2 distortion product otoacoustic emission (DPOAEs) as a measure of efferent activity. An advantage of using SFEs over DPOAEs is that less energy in the SFE tonal stimulus is needed as compared to the primary levels needed to produce a distortion product of the same magnitude. Interpretation of efferent
effects on SFEs is also than with DPOAEs because the effect of efferent-induced change at the primaries and the distortion product is not well understood.

D-1) Group Delay: Middle Ear Muscle effect vs. MOC effect

In studying MOC efferents using acoustic reflex assays, it is important to be sure that the response recorded was not a result of stapedius muscle contraction due to loud sounds. The effects of MOC efferent versus the middle ear muscles (MEMs) can be distinguished by their group delays. The MOC-induced effect is observed as a change in the SFE, which is generated within the cochlea. Hence, it has a long delay - on the order of many ms (~10 ms has been recorded for a 1kHz test tone) due to the time needed for energy to propagate along the cochlea partition to the CF place as well as the delay associated with the action of the cochlear amplifier (Yates, 1995, Patuzzi, 1996). MEM contractions change the impedance of the middle ear and the travel time for sound to reach the middle ear and return to the ear canal is significantly shorter than the travel time into the cochlea and back (Backus et al. 1999).

So how can we measure this time delay associated with responses that originated deep within the cochlea? In a linear dispersive medium, where the wave velocity is a function of frequency, group delays \((-d\phi/df)\) is the appropriate measure of energy transport (Elmore and Heald, 1969). \(\phi\) is the phase delay in cycles and \(f\) is frequency in cycles per second. The group delay is the time delay that would give the same rate of change of phase with increase in stimulus frequency.

In order for any amplitude-modulated wave to propagate through a medium unchanged, the group velocity must be independent of frequency (Elmore and Herald, 1969), which we know is not true in the highly nonlinear cochlea. To deal with this, we need to make sure that \(df\) is small enough so that the group velocity within that region varies slowly enough to be assumed constant. Then the measure of the group delay is useful to us since a fixed time delay between the stapes vibration and the basilar membrane vibration at a particular cochlear location would produce a linear relationship between phase and frequency (Patuzzi, 1996).

D-2) Ipsilateral and Binaural measurements

In the case of a contralateral elicitor, the resultant trace of the change in SFE vs. time directly reveals the time course of the MOC effects. The situation is not as simple in the case of the ipsilateral or binaural reflex. This is because the elicitor could have as one of its frequency components, the test tone frequency which could directly interfere with the measurements. However, this problem could be solved by reversing the polarity of the noise on alternate bursts within the run - which upon pair-wise averaging, most of the interference effect would cancel out (Backus et al, 1999).

Another problem is that the elicitor can suppress the test tone via the two-tone suppression mechanism, which arises as a consequence of the cochlea's nonlinearity (Probst et al., 1990). We need to make sure that we do not mistake this measure as MOC
The way to circumvent this problem is by exploiting the fact that suppression effects disappear almost instantaneously after the offset of suppression but the MOC-induced change in SFE decays on a much slower time scale. Hence, an elicitor consisting of long noise bursts with a 50 ms wait period after the offset and an analysis window restricted to the period 50 – 150 ms after the elicitor is turned off can be used for the measured MOC-induced effects.

D-3) State of alertness and the effects on MOC response

Another important issue to keep in mind is that selective attention is known to modify the active micromechanical properties of the cochlea, presumably by modulating efferent activity (Puel et al., 1988). There is also evidence for a reduction in OAEs when the subject is asleep (Morlet et al., 1994) as well as when the subject is asked to perform a competing visual task during the recordings (Puel et al., 1988). To help control alertness, or at least prevent sleep, we allow subjects to take frequent breaks during intervals between experimental runs. Another possibility, which we have yet to implement, is to design some task for the subjects to perform in between the recordings so as to keep the subject alert and thereby maximize the efferent effect on SFE measurements.
Introduction

The Medial Olivocochlear (MOC) efferent fibers provide feedback control of the cochlear operation. They can be excited by both ipsilateral and contralateral sound stimuli, thus forming a descending binaural-reflex pathway (Fex 1962). This efferent bundle originates in the medial part of the superior olivary complex and synapses directly on the numerous outer hair cells (OHCs) which are embedded in the organ of Corti (OC). In the early 1980’s, Brownell et al. reported motile behavior of OHCs when they were stimulated in vitro with extracellular current (Brownell et al. 1985). In vivo, it is believed that OHCs form the active and motile elements that provide the mechanical gain for the nonlinear and highly tuned basilar membrane (BM) motion (Davis 1983, Dallos and Evans 1995). This phenomenon is also known as the cochlear amplifier and can increase the vibration of the BM and OC 1000-fold or more, allowing the cochlear to detect fluctuations in acoustic sound pressure as low as 1/2,000,000,000 of normal atmospheric pressure (20 μPa rms), (Fay 1988). So by altering the state of the OHCs, the MOC fibers provide direct control of the sensitivity and frequency specificity of the cochlear amplifier.

From studies done on anesthetized animals, anatomical and physiological findings of the MOC system appear to support the idea that it can be activated in a highly frequency-selective manner. Single fiber studies in cats showed that each MOC fiber branches within the cochlea to innervate OHCs up to a span of one octave in cochlear frequency and that the MOC efferents respond to ipsilateral and contralateral sound with sharp tuning (Liberman and Brown, 1986). Intracellular labeling done on cats and guinea pigs also revealed that there is at least a rough match between the afferent and efferent tonotopic maps (Liberman and Brown, 1986, Brown, 1989).

In contrast to the substantial amount of published animal studies, significantly less has been established in humans. Basic anatomical information like the spread of single MOC neurons’ peripheral projections in humans cannot be obtained directly with existing techniques used in animals. However, a more indirect way, which is non-invasive, is to employ acoustic reflex assays to learn more of the extent of the efferent control along the frequency axis.

Over the years, various non-invasive techniques have been developed that allow for a way to indirectly infer aspects of the frequency specificity of the medial efferent reflexes in humans. In 1978, Kemp reported a phenomenon where sounds are emitted by the cochlea, either spontaneously or in response to an acoustic stimulation. These are now known as Otoacoustic emissions and they can be recorded in the ear canal. Later on, it was reported that the electrical stimulation of the MOC efferents altered the cochlear mechanics in some way, producing a change in the distortion tones measured acoustically from the ear canal (Mountain, 1980). In accordance with current thoughts that the OHCs have an active role in generating OAEs (Mountain and Hubbard 1989), control of cochlear responsiveness via the MOC efferents can be inferred non-invasively through the measurements of OAEs.

In past human studies, the magnitude of OAE suppression was usually expressed as the dB change in emission magnitude produced by the presence of an elicitor of efferent activity, usually with the elicitor delivered to the contralateral ear. The effects
reported in existing literature are usually on the order of 1-3 dB (e.g. Collet et al., 1990 a, b; Veuillet et al., 1991, Maison et al. 2000).

Among the very few human data on the frequency specificity of the efferent-induced effect, only two show plots of the frequency specificity and it is only for the contralateral reflex extending from 1-2 kHz (Ryan et al. 1991, Chery-Croze et al., 1993, Norman and Thorton 1993, Maison et al. 2000). There are no published data on ipsilateral nor binaural reflex frequency selectivity in humans or animals.

In this present study our aim is to determine the frequency resolution of the MOC reflex in humans. We record the changes in SFEs at a fixed frequency due to a contralateral, ipsilateral or binaural narrow-band noise elicitor at a range of frequencies. The scope of this study shall cover a range of frequency regions from 0.77 to 2.6 kHz. To gain some insight on the span of efferent control along the frequency axis, two paradigms were used:

1) For a fixed test tone, the center frequency of a Narrowband Noise (NBN) MOC-elicitor was systematically varied. The total energy within the noise band was kept constant. The effects from ipsilateral, contralateral or binaural elicitors were compared. At each test tone frequency, MOC tuning functions with evoking bursts at several sound levels (that do not evoke MEM effects) were studied.

2) We accessed the limit of bandwidth for the MOC eliciting activity at the cochlear region of interest by varying the bandwidth of the NBN elicitor stimuli, whose center frequency is placed at the test tone frequency. The range of bandwidths will be from 1/16 octave to 4 octaves.

With so little known, a better understanding of human MOC functional capacity in terms of its frequency specificity is necessary and would help bring us one small step closer to understanding the MOC functional significance.
Measurement Methods

Subjects

In each of the four subjects, measurements were done from both ears. All subjects had no history of auditory pathology and had hearing levels no higher than 20 dB SPL between 500 Hz and 4000 Hz at octave intervals on a 1/3 octave narrowband noise audiogram. On a test using broadband noise, hearing levels were also less than 20 dB SPL. The subjects were seated in a comfortable reclining chair in a soundproofed room isolated from vibrations (Ver et al. 1975).

Sound Stimuli

Stimulus waveforms were generated and the responses were recorded and averaged digitally using a custom-built data-acquisition system implemented in LabVIEW6 and Matlab. The sampling frequency was typically set to 20kHz. Acoustic signals were transduced using 2 Etymotic Research ER10c acoustic assemblies, one in each ear and each assembly consisting of 1 ER10b low-noise microphone with ER-10c foam tips to keep the probe in place within the ear canal. The sound stimuli were mixed acoustically using 2 separate sound sources to prevent intermodulation distortion in the electrical to acoustic transduction of the two signals. In-ear calibrations were done at regular intervals during all measurement sessions to ensure that the stimulus tones and the components of the noise band elicitors delivered had constant level and constant starting phase in the ear canal at all frequencies.

To induce SFEs, a pure tone stimulus was presented continuously to both ears at 40 dB SPL. We used either narrow or broadband noise to elicit efferent response in the ipsilateral ear, contralateral ear or in both ears. The spectral components of the noise band had equal level (i.e. were ‘flat’ across frequencies) and random starting phases which were uniformly distributed between -π and π. The noise level was set to 55 or 60 dB SPL during the experiment and the phase polarity of its spectral components was flipped in alternate runs so that noise elicitor will cancel in the averaging of measurements.

In the time domain, stimulus presentation is done as follow:

1) The `Baseline window` begins at 0 ms and ends at 500ms. During this time, only the continuous tone that produces the SFE, without the elicitor was presented.
2) The elicitor noise burst began at 500 ms and lasted for 2500ms. The waveform of interest was recorded in a 100 ms window placed 50 ms after from the falling edge of the noise-burst. This will be referred to as the `Response window`.
3) Finally, a rest period of 1500 ms intervened between the elicitor and the next stimulus baseline.
Artificial rejection

Real-time artifact rejection was implemented in the time domain by taking the difference between successive data buffers to ensure that the each of the following values falls below a set criterion.

1) the maximum absolute difference between the 2 buffers
2) the DC component or the average value in the ‘Baseline window’
3) the average rms. value from the mean in both the ‘Baseline window’ and the ‘Response window’.

Spontaneous Otoacoustic Emissions

The presence of spontaneous otoacoustic emissions (SOAEs) in each subject was determined by recording the ear-canal pressure with the absence of any stimulus tone or elicitor. If any SOAEs were detected, the stimulus tone for that particular subject was chosen to be at least 50 Hz away from adjacent SOAEs to prevent entrainment – a phenomenon where the emissions are synchronized by external tones (Long et al. 1988, Long and Tubis, 1988a, Long et al. 1990, Tubis et al., 1989, van Dijk and Wit, 1988).
Analysis Methods

Spontaneous Otoacoustic Emission

The recorded ear-canal signal, without the presence of any sound stimuli, was processed by taking its Fourier transform (FFT). To reduce the amplitude of the random peaks in the spectra due to random noise, the FFTs were averaged in frequency (Probst, 1990).

SFE Envelop Extraction

We performed a fast Fourier transform (FFT) on the raw pressure waveform. A band of positive spectral components surrounding the stimulus test tone frequency was selected. These frequency components were then shifted down so as to center the stimulus frequency at zero Hz. A 10th order recursive exponential filtering window was then used to remove higher frequency components of noise arising from the equipment or the subject. This filter was defined by Shera and Zweig (1993a) as,

$$\hat{S}_n(f; f_{cut}) = \frac{1}{\Gamma_n(\lambda_n f / f_{cut})},$$

where \(\tau_{\text{cut}}\) is the cutoff frequency and the function \(\Gamma_n(\tau)\) is defined recursively as:

$$\Gamma_n(\tau) = e^{\Gamma_{n-1}(\tau-1)}, \text{ with } \Gamma_1(\tau) = e^{\tau^2}.$$

The window \(\hat{S}_n(f; f_{cut})\) has a maximum value of 1 at \(f=0\).

The scale factor \(\lambda_n\) is chosen such that the window falls to the value of 1/e at \(f = f_{cut}\):

$$\lambda_n = \sqrt{\gamma_n}, \text{ where } \gamma_{n+1} = \ln(\gamma_n + 1) \text{ with } \gamma_1 = 1.$$

\(\hat{S}_n(f; f_{cut})\) has a much sharper cutoff than standard window functions (e.g., the Hamming, Blackman, etc.) and does not have any side-lobes. On the other hand, since this function has no poles, it produces considerably less ‘ringing’ than the boxcar window, which provides an infinitely sharp cutoff.

The larger the number of iterations, \(n\), the sharper the roll-off. In this present study, we chose \(n = 10\) and \(f_{cut} = 90\) Hz. The values were chosen during preliminary studies done on a small cavity and these parameters appeared to give the best compromise between reduction of the background noise spectrum and edge-smearing effects in the time domain, suitable to the time scale of the effect we are studying.

The result from this spectral component extraction is a complex-valued signal, which represents the magnitude and phase of the SFE envelope with time. No phase distortion is added. The process also automatically downsamples the waveform buffer.
Baseline Subtraction

To measure the change in SFE produced by MOC activity, we can calculate the vector difference between the ear canal sound pressure produced by the continuous test tone with or without the presence of the MOC activity evoked by another sound stimulus (Guinan, 1986). Once the processed wave is transformed back into the time-domain, the complex mean of the signal in the ‘Baseline window’ is subtracted from the whole waveform. The complex average in the response window now contains the magnitude and phase of the sound pressure due to the MOC-induced change in SFE, $P_{\Delta SFE}$.

![Sound Pressure Diagram](image)

Figure M2. Vector diagram illustrating method of baseline subtraction
Labels:
1: $P_{SFE}$ with elicitor
2: $P_{BASELINE}$
3: $P_{TEST TONE}$
4: $P_{SFE}$ without elicitor

It can be seen from the diagram that,
$$P_{\Delta SFE} = P_{SFE \ with \ elicitor} - P_{SFE \ w/o \ elicitor} = P_{RESPONSE} - P_{BASELINE}$$

$\phi_{\Delta SFE}$, is the leading phase referenced to the baseline. This makes it independent from phase gitters in the equipment.

Group Delay Calculations

The group delay test can be applied, using BBN as the elicitor, at one frequency to determine the highest sound level that evokes efferent effects but without evoking significant MEM effects. Several studies have shown that the stapedius muscles are excited significantly more with BBN than NBN, with the energy level kept constant.
Margolis et al., 1980). Based on this, the maximum BBN level will also be used for the NBN.

Data Analysis

Two analysis time windows are of interest to us. The first window is from 3050-3150 ms from the start. This window basically starts 50 ms after the elicitor is turned off and is used to analyze the ipsilateral, contralateral and binaural response. The second window is from 2450-2950 ms - during which the elicitor is on. This 500 ms window is used to analyze the contralateral response – for analysis that don’t compare to ipsilateral or binaural responses.

After the raw wave is heterodyned and the complex baseline average value is subtracted from it, the processed waveforms are synchronously averaged point by point with corresponding processed waveforms from different sessions. Points in the analysis window of the final averaged heterodyned waveform are then averaged to give a complex number which contains the magnitude and phase information of the response.

Noise Analysis

Due to lack of data to sufficiently characterize the statistics of the signal, standard statistical detection and estimation techniques could not be employed. So for the following set of results and subsequent discussion, signals that are at least 3 standard deviations of the equipment noise above the mean of the noise floor are considered significant.

The statistics of the noise were calculated from an estimate probability mass function where the sample space consisted of |ΔP<sub>SFE</sub>| obtained from non-overlapping time windows from all runs without an elicitor within the series. In order to pool the noise data across experiment sessions, we made the assumption that equipment noise was the main noise contributor at frequency ranges above 400 Hz and that its statistical characteristics are independent of the subject’s physiology. Below 400 Hz, physiological noise, which varies considerably from subject to subject, becomes important. The statistics of the noise was remarkably consistent across subjects which seems to be in agreement with this assumption. Preliminary noise data also seems to suggest that the phase is approximately distributed uniformly between -180 and 180 degrees. Hence, phase information was ignored when estimating the statistics of the noise.

The number of points used to estimate the probability mass function was 242 points when the analysis window for the signal was placed before the elicitor was turned off and 220 points when placed after the elicitor was turned off. To calculate |ΔP<sub>SFE</sub>| noise values within a run, the start of the baseline window was shifted in time steps equal to the analysis window length while keeping the duration between the baseline window and the analysis window constant. The duration between the baseline and analysis window was kept constant to avoid time dependent ‘drifts’ as seen in a random walk model for noise.
From a right-hand probability distribution function of the noise, the probability of the noise taking on a value 3 standard deviations above the mean is approximately 0.5% in all subjects.
Results

Heterodyned waveform from ear-canal pressure measurement

Figure 1 shows a typical time course of the magnitude and phase of the change in ear canal pressure due to a contralateral BBN elicitor. After the onset of the BBN elicitor at 500 ms, the magnitude of the pressure change, $|\Delta P_{SFE}|$, rose gradually over hundreds of milliseconds before reaching saturation. After the BBN elicitor was turned off at 3000 ms, $|\Delta P_{SFE}|$ decays over several hundreds of milliseconds before falling into the noise floor. When $|\Delta P_{SFE}|$ emerged substantially above the noise floor, the phase lag $\psi_{SFE}$ was relatively constant.

![Figure 1. Magnitude and phase of efferent-induced change in sound canal pressure, $\Delta P_{SFE}$, as a function of time. Test tone used was 1.03 kHz at 40 dB SPL. The contralateral elicitor was BBN at 60 dB SPL.](image)

A different time course is observed when an ipsilateral or binaural BBN elicitor is used. Figure 2 shows the magnitude and phase of the change in ear canal pressure due to a BBN elicitor in both ears. At the onset of the elicitor at 500 ms, we observe a sharp rise in $|\Delta P_{SFE}|$ over a time period on the order of 10 ms. This rapid rise is primarily a consequence of the ipsilateral BBN elicitor having frequency components within the vicinity of the test tone, which results in two-tone suppression. Hence, when consecutive measurements from runs with elicitors of flipped polarity are averaged, we get an good
cancellation of the BBN elicitor but suppression dominates the response. This manifests as the change in SFE seen at the elicitor onset. Efferent-induced SFE change occurs simultaneously but at a much slower rate, and in this case, on a smaller magnitude scale than the change induced by suppression. At 3000 ms, when the elicitor is turned off, we see a rapid decrease in $|\Delta P_{\text{SFE}}|$ over tens of ms followed by a slower decrease over hundreds of ms. This rapid decrease corresponds to the decay of the two-tone suppression effect, after which, the slower decay of the efferent effect dominates. The $-\Delta P_{\text{SFE}}$ again seems relatively constant when the $|\Delta P_{\text{SFE}}|$ is above the noise floor. It is interesting to note, however, that the value of $-\Delta P_{\text{SFE}}$ changes when the efferent-induced $\Delta P_{\text{SFE}}$ dominates in magnitude. This trend of phase change is seen in some subjects and not others, with no obvious dependence on the elicitor laterality or test tone frequency.

Figure 2. Magnitude and phase of efferent-induced change in sound canal pressure, $\Delta P_{\text{SFE}}$, as a function of time. Test tone used was 1.03 kHz at 40 dB SPL. The contralateral elicitor was a BBN at 60 dB SPL. 12 runs were averaged. (Subject 68)

Group delay measurement

It is important to check that the change in SFE observed is due to efferent effects within the cochlea and not MEM contraction in response to loud sounds. This can be done by measuring the group delay of the response. Figure 3 shows the magnitude and phase of the change in ear canal pressure due to a BBN elicitor in the ipsilateral ear, contralateral ear, and both ears as a function of test tone frequency. The noise floor is
derived from measurements with no elicitor within the same run. The error bars mark the level equivalent to 3 standard deviations above the mean of the noise floor.

In the example shown in figure 3, using a least square error linear fit on the phase plot, we find that the group delay is approximately 15 ms for all 3 elicitors which agree with existing literature on human SFE group delay for the test tone frequencies within the range used (Dreisbach et al. 1998).
Response Curves

Figure 4 summarizes the ipsilateral, contralateral and binaural response curves at test tone frequencies 0.77 kHz and 1.03 kHz. The elicitor used was a 0.5 octave NBN with a constant energy level but different center frequencies. Data points that are at least 3 standard deviations above the mean noise floor were considered significant. The signal analysis window used was 100 ms in duration and started 50 ms after the elicitor was turned off.

In the set of data where the test tone was 0.77 kHz, the binaural and contralateral response curves in the left ear appears to be asymmetric with the maximum $|\Delta P_{SFE}|$ occurring at elicitor center frequencies below the test tone. The ipsilateral response was too weak under the chosen criterion to make any meaningful observations. The binaural stimulation appeared to elicit changes in SFE even when its center frequency was 2 octaves below or 3 octaves above the test tone. The range was less with the contralateral stimulation, where changes in SFE were observed with elicitor center frequencies 0.5 octave above and 2 octaves below the test tone. The phase of the binaural and contralateral responses appeared relatively constant and remarkably similar in value for a given elicitor center frequency that induced significant efferent response.

In the right ear, the binaural and ipsilateral responses were also more effective towards the lower frequency end. The most effective elicitor in both cases was when the center frequency was at the test tone. Changes in SFE were observed with elicitor center frequencies 0.5 octave above and 2 octaves below the test tone. In this range of elicitor center frequency, it is interesting to note that the phase change of the binaural and
ipsilateral response appears to have a negative slope instead of being approximately constant.

In the data set where the test tone was 1.03 kHz, more effective responses were achieved with noise bands centered above the test frequency in the right ear. This trend is less pronounced in the left ear. There appears to be subtle peaks and valleys in the shape of the response curves although more data is needed to determine whether or not they are significant. Significant binaural response in the left ear was observed with noise bands centered 2 octaves below and 2.5 octaves above the test frequency. The right ear binaural response was induced with noise bands centered 1 octave below and 2.5 octaves above the test frequency.

At both 0.77 and 1.03 kHz test tone, binaural stimulation was almost always more effective in eliciting efferent activity for the elicitor center frequencies used. Noise bands centered near the test tone also appeared more effective than those further away.

Figure 5, shows only contralateral response curves at the same test frequencies as figure 4 but with the analysis window between 2450 and 2950 ms. Recall that the elicitor is gated on from 500 to 3000 ms, and so no ipsilateral or binaural efferent response curve could be reported using this analysis window since it is impossible to separate out the changes in SFE due to suppression.

With the 0.77 kHz test tone, efferent-induced changes in SFE were seen at all elicitor center frequencies used, which ranged from 2 octaves below to 2.5 octaves above the test frequency. The tuning again appears asymmetric about the test frequency with the maximum response occurring with the elicitor centered below the test frequency. In the right ear, response was seen from 2 octaves below to 2.5 octaves above the test frequency. However, the peak response is when the noise band is centered around the test tone. More pronounced peak and valleys, with $|\Delta PSFE|$ difference of approximately 10 dB SPL, are also seen at different elicitor center frequencies.

With the 1.03 kHz test tone, efferent-induced change in SFE was seen when the elicitor was centered at frequencies above the test frequency in both the left and the right ear - up to 2.5 octaves above. Noise bands centered at 1 to 1.5 octaves below the test tone induced almost no SFE change. The response was greatest when the noise band is centered above the test tone. More pronounced peak and valleys are also seen at different elicitor center frequencies in the left ear.

Figure 6 shows the ipsilateral, contralateral and binaural response curves obtained from both the left and right ear of Subject 61 with the 100 ms analysis window starting at 50 ms post elicitor-off. A 2.6 kHz test tone with 0.5 octave NBN elicitor of various center frequencies was used. The left ear response was significantly weaker than that of the right ear. The efferent response observed in both ears appeared to be stronger when the noise bands used were centered above the test tone frequency. In the right ear, binaural and contralateral responses were observed with noise bands centered at frequencies up to 1.5 octaves above the test tone and roughly 1 octave below. The maximum $|\Delta P_{SFE}|$ occurred with the binaural noise band centered at 1.5 octaves above the test tone. The ipsilateral response was weaker and only showed response values above the 3std criteria at 0.5 octave above and below the test frequency. The maximum $|\Delta P_{SFE}|$ in the ipsilateral and contralateral curve was when the elicitor was centered at the test frequency. The phase change appears relatively constant when a significant magnitude of efferent response was observed.
Figure 4. Response curves at test tone frequencies 0.77 kHz (Subject 61) and 1.03 kHz (Subject 68). The magnitude and phase of the change in SFE is plotted as a function of the center frequency of the 0.5 octave NBN elicitor (kHz). The graphs on the left and right correspond to results from the left and right ear respectively. The test tone level and elicitor level were at 40 and 60 dB SPL respectively. 8 averages were used to obtain the data at 0.77 kHz test tone. 12 averages were used to obtain the data at 1.03 kHz test tone. The analysis window was of length 100 ms taken 50 ms after the elicitor was turned off. The vertical bar in each graph marks the test tone frequency used.

- O: Elicitor in Left Ear
- △: Elicitor in Both Ears (change symbol to inverted triangle)
. ★: Elicitor in Right Ear
--: No Elicitor

The error bars are 3 std above mean.
Figure 5. Response curves at test tone frequencies 0.77 kHz (Subject 61) and 1.03 kHz (Subject 68). The magnitude and phase of the change in SFE is plotted as a function of the center frequency of the 0.5 octave NBN elicitor (kHz). The analysis window was of length 100 ms, taken 50 ms before the elicitor was turned off. The graphs on the left and right correspond to results from the left and right ear respectively. The test tone level and elicitor level were at 40 and 60 dB SPL respectively. 8 averages were used to obtain the data at 0.77 kHz test tone. 12 averages were used to obtain the data at 1.03 kHz test tone.

- O - Elicitor in Left Ear
- * - Elicitor in Right Ear
- -- No Elicitor

The error bars are 3 standard deviations above the mean noise floor.
Figure 7 shows only the contralateral response curves with the same experimental parameters as that described in figure 6. However, the analysis window used is from 2450-2950 ms, during which time the elicitor is gated on. The response from the left ear is still too weak to make any credible observation. On the right ear, the contralateral response from 2 octaves down to 1.5 octaves above the test tone appears significant. However, it does not have a bandpass shape within the elicitor center frequency range used. In fact, it appears to correspond more to a highpass band. The phase change appears relatively constant when the magnitude of efferent response was significant.

Figure 8 summarizes the ipsilateral, contralateral and binaural response for a 1.02 kHz test tone in Subject 67 and a 1.2 kHz test tone in Subject 73. We used a 100 ms analysis window starting at 50 ms after the elicitor was off. The left ear in both subjects gave very weak efferent response for the set criteria. In the case where the test tone was at 1.01 kHz, the binaural and contralateral response from the right ear was only seen as far as 1-1.5 octaves below the test tone. No significant response was seen above the test tone. At 1.2 kHz test tone in another subject, binaural efferent response was observed as far as 1 octave above and below the test frequency.

Figure 9 summarizes the contralateral response curves from the left and right ear of Subject 67 and 73 at 1.01 kHz test tone and 1.2 kHz test tone respectively. The analysis window used is the same as that used to obtain figure 7. In both subjects, the left ear contralateral response is also very weak and will not be discussed. In the data set where the test tone was at 1.01 kHz, SFE change was observed with elicitors centered as far as 1.5 octaves below and 2 octaves above the test frequency. There also appears to be 2 peaks in the response, one between 0.5 and 1.5 octaves below the test frequency and one between 0.5 and 2 octaves above the test tone. The phase change of the response appears relatively constant.

With the test frequency of 1.2 kHz, significant response was observed as far as 2 octaves below and 1 octave above the test frequency. The maximum response occurred within the vicinity of 1.5 octaves below the test tone. Another smaller peak was seen around 1 octave above the test tone. The phase seemed roughly constant where efferent response was observed.

The validity of the peaks and valleys seen in all figures would need more data and statistical analysis given the variability of the efferent within the same subject with the same experimental parameters. This issue of intra-subject variability shall be discussed in subsequent sections.
Figure 6. Ipsilateral, contralateral and binaural response curve at 2.6 kHz (Subject 61) test tone from the elicitor-off analysis window. All other parameters and symbols are the same as Figure 4.

Figure 7. Contralateral response curve at 2.6 kHz (Subject 61) test tone from the elicitor-on analysis window. All other parameters and symbols are the same as Figure 5.
Figure 8. Ipsilateral, contralateral and binaural response curve at 1.01 kHz (Subject 67) and 1.2 kHz (Subject 73) test tone from the elicitor-off analysis window. All other parameters and symbols are the same as Figure 4.
Figure 9. Contralateral response curves at 1.01 kHz (Subject 67) and 1.2 kHz (Subject 73) test tone from the elicitor-off analysis window. All other parameters and symbols are the same as Figure 5.
Normalized Response curves

In order to compare the shapes of the response curves for the different test frequencies, we normalized the response curves’ $|\Delta P|_{SFE}$ values and scale the center frequencies of the elicitor so that the curves can be superimposed. Figure 10 and 11 are normalized response curves using the analysis windows 3050-3150ms and 2450-2950 ms respectively. The former window lies in the elicitor-off time, while the latter lies in the elicitor-on time. Normalized $|\Delta P|_{SFE}$ is defined as the difference between the $|\Delta P|_{SFE}$ in dB SPL and the maximum value of $|\Delta P|_{SFE}$ in dB SPL for the given test tone. The center frequency of the noise band was also normalized by dividing it with the test frequency so that the noise band centered around the test frequency takes on the value of 1. The normalized response that satisfies the 3 standard deviation above the noise criteria are marked by their respective symbols while those that do not are not marked. We notice that the peak values from the normalized response curves do not line up. The maximum efferent response can occur with noise bands centered below, at or above the test frequency. There is no obvious dependence of the position of the peak response relative to the test tone on the subject or frequency of test tone.

Variability of efferent response within a subject

Three response curves with relatively large responses were chosen to demonstrate the variability in efferent response under the same stimulus conditions. Figure 12 shows the contralateral response curves analyzed within the 500 ms window which ends 50 ms before the elicitor is turned off. The first, second and third columns correspond to response curves with test frequencies 0.77, 1.03 and 2.6 kHz respectively. The first two rows are the SFE magnitude and phase change obtained from 12 averages. The response values that are at least 3 standard deviations above the mean noise floor when no elicitor is present are marked with a symbol. The bottom row of graphs show the SFE magnitude and phase change of the three 4-average runs which combine to give the 12-average response curves shown above. The points that are marked here correspond to those in the 12-average response curves. The horizontal line represents the 3 standard deviation above the mean noise floor from all runs with 4 averages.

In the 4-average curves, the phase change appears very close in value to that of the 12-average run. The spread of the response values that lie above the 3 standard deviation above the mean noise floor criterion are as large as 4.5, 7.5 and 8.5 dB SPL when the test frequency was 0.77 kHz, 1.03 kHz and 2.6 kHz respectively.
Figure 10. Normalized delta P as a function of normalized center frequency of 0.5 octave NBN elicitor. Normalized delta P is the dB SPL difference between delta P of the raw data and the maximum delta P within an experiment with a fixed text tone. The normalized center frequency of an elicitor is obtained by dividing that center frequency with the test tone frequency. Plots A and B were obtained with binaural elicitors. Plots C and D were obtained with ipsilateral elicitors. Plots E and F were obtained with contralateral elicitors. Plots on the left panel correspond to results from the left ear while the plots on the right panel correspond to the results from right ear. Analysis window used was 100 ms starting 50 ms from the time the elicitor was turned off.

Subject 61: Test tone frequency = 0.77 kHz
Subject 67: Test tone frequency = 1.01 kHz
Subject 68: Test tone frequency = 1.03 kHz
Subject 73: Test tone frequency = 1.20 kHz
Subject 61: Test tone frequency = 2.60 kHz
Figure 11. Normalized contralateral response curves as a function of normalized center frequency of 0.5 octave NBN elicitor. Analysis window was from 2450-2950 ms, during which the elicitor was on. All other parameters and symbols used are the same as figure 10. Note that on the left panel which represents data from the left ear, no data from Subject 73 was plotted as the response was too weak.

Contralateral and binaural efferent tuning curve

Figure 13 shows response curves and the resulting tuning curves obtained with 0.5 octave NBN elicitors of levels of 40, 50, 55, 60 dB SPL at a test frequency of 0.77 kHz. Only the left and right contralateral responses and the right binaural response are presented because the response from the other elicitors did not have a good enough signal to noise.

In many physiological studies, the sharpness of tuning curves is measured with a quantity called Q20. Q20 is defined as the center frequency (CF) divided by the bandwidth 20 dB above threshold.

\[
Q20 \text{ of the left contralateral tuning curve} = \frac{\text{BW with 60 dB SPL}}{\text{threshold frequency}} = \frac{1.3962 - 0.3287}{0.5445} = 1.9605
\]

\[
Q20 \text{ of the right contralateral tuning curve} = \frac{1.2353 - 0.3226}{0.5445} = 1.6762
\]

\[
Q20 \text{ of right binaural tuning curve} = \frac{1.2366 - 0.2226}{0.5445} = 1.8623
\]
Figure 12. Magnitude and phase of the change in SFE obtained with 12 or 4 averages. The top two rows are the SFE magnitude and phase change averaged from 12 sets of data. The errorbars in the top row represent the standard deviation from the 3 runs, each an average of 4 sets of data, which were then averaged to give the resulting response curve. The response values that are at least 3 standard deviations above the mean noise floor are marked with a circle (o). In the phase plot, (o) and (.) are change in phase with and without an elicitor respectively. The bottom 2 rows of graphs are the three 4-average runs. The marked points correspond to those in the top graphs. The horizontal line shown indicates the 3 standard deviation mark above the mean noise floor. Hence points above that line satisfy the criteria of significance.
Figure 13. Graph A shows response curves obtained from the left ear by eliciting MOC efferents with 0.5 octave contralateral NBN elicitor at different levels. Graphs B and C, are response curves obtained from the right ear with contralateral NBN and binaural NBN respectively. The test tone was at 0.77 kHz in all graphs. The NBN levels were 40 dB SPL (-O-), 50 dB SPL (-Δ-), 55 dB SPL (-*-) and 60 dB SPL (-X-). Graphs D, E, and F are the MOC efferent tuning curves obtained from Graphs A, B, and C respectively. The response curves obtained with contralateral NBN were analyzed in the 2450-2950 ms window. The binaural response curve was analyzed in the 3050-3150 ms window.
Ipsilateral, contralateral and binaural responses as a function of elicitor bandwidth

Figure 14 shows the ipsilateral, contralateral and binaural efferent response with elicitors of different bandwidths. The test frequencies studied are 0.77 kHz, 1.03 kHz and 2.6 kHz. For points significantly above the noise floor, we see that the efferent response generally increases with the bandwidth of the noise band elicitor and in some cases reaching saturation. It appears that in the response that saturated, the point of saturation occurs between bandwidths of 2 and 4 octaves. In the case when the test tone was 2.6 kHz, the right binaural and contralateral response levels appear to be higher with a 4 octave band than a BBN. Only the right ipsilateral response with test frequency of 2.6 kHz decreased with increasing elicitor bandwidths. The strength of the binaural response appears to be largest at almost all elicitor bandwidths used. With the limited amount of data presented, the strength of the ipsilateral and contralateral response appears to be somewhat comparable except for the results with the test frequency of 2.6 kHz.

Figure 15 is a redisplay of the results from figure 14 such that the responses obtained with the same elicitor orientation, ipsilateral, contralateral or binaural, are plotted together.

Figure 16 is the normalized data from figure 14. The normalization is done by subtracting the magnitude change in SFE ear canal pressure (in dB SPL) with the magnitude change obtained with a 2 octave noise band. Figure 17 is the same as figure 16 but the normalized data are regrouped into ipsilateral, contralateral and binaural responses. In both figures we note that plots of significant responses have very similar shape and rate of growth. The ipsilateral response in one subject appears to be different; it decreases as bandwidth increases.
Figure 14. Change in magnitude of SFE in the presence of efferent stimulation as a function of elicitor bandwidth centered at the test frequency. Test tone was at 40 dBSPL. Noise bandwidths were 1/8, 1/2, 1, 2 octaves for test frequency 1.03 kHz and 1/8, 1/2, 1, 2, 4 octaves for test frequencies 0.77 and 2.60 kHz. For the plots with test tones 0.77 kHz and 2.6 kHz, the very last points on the right correspond to efferent response to BBN elicitor which spans from 100 to 10 kHz. Elicitor level was 60 dBSPL. Analysis window used was 100 ms in duration starting 50 ms after the elicitor is turned off.

Symbols:  
Inverted triangle = Elicitor in both ears,  
* = Elicitor in right ear,  
○ = Elicitor in left ear,  
● = No elicitor
Figure 15. Same as figure 14 but with the binaural, ipsilateral and contralateral responses plotted separately.

= 0.77 kHz test frequency
* = 1.03 kHz test frequency
◊ = 2.60 kHz test frequency

Data points marked with a symbol were found to be at least 3 standard deviations above the mean noise floor.
Figure 16. Change in magnitude of SFE in the presence of efferent stimulation normalized by the response level obtained with a 2 octave noise band elicitor as a function of elicitor bandwidth centered at the test frequency. Test tone was at 40 dBSPL. Elicitor level was 60 dBSPL. Analysis window used was 100 ms in duration starting 50 ms after the elicitor is turned off.

Symbols:
- Inverted triangle = Elicitor in both ears,
- * = Elicitor in right ear,
- o = Elicitor in left ear,
- • = No elicitor

Plot (A, B) = test frequency of 0.77 kHz
Plot (C, D) = test frequency of 1.03 kHz
Plot (E, F) = test frequency of 2.60 kHz

For the plots with test tones 0.77 kHz and 2.60 kHz, the very last points on the right correspond to efferent response to BBN elicitor which spans from 100 to 10 kHz.
Figure 17. Change in magnitude of SFE in the presence of efferent stimulation normalized by the response level obtained with a 2 octave noise band elictor as a function of elictor bandwidth centered at the test frequency. Separated into Ipsilateral, contralateral and binaural plots.

- = 0.77 kHz test frequency
* = 1.03 kHz test frequency
◊ = 2.60 kHz test frequency

Data points marked with a symbol were found to be at least 3 standard deviations above the mean noise floor.
Discussion

The response curves presented in this study clearly demonstrate a frequency specificity of the efferent-induced change in SFEs but also a surprising ability of distant frequencies to activate the efferents. Response strength and tuning pattern also appeared to vary between the 2 ears within a subject. The greatest change in $|\Delta P_{SFE}|$ were observed when the elicitor noise bands were centered near the test frequency, but not necessarily at the test frequency. Noise bands were also more effective when centered near the test tone rather than further away. We also saw that in some cases, elicitors centered as far as two and a half octaves above and/or below the test tone elicited significant changes in SFE. This is perhaps the most significant finding in this study. The tuning observed is thus broader than the tuning reported in other existing literature on Humans.

For example, Veuillet and colleagues (1991) pointed out a frequency specificity of contralateral NBN stimulation on evoked otoacoustic emission (EOAE) amplitude as seen from data averaged across subjects. The ipsilateral tone pip was limited to 1 and 2 kHz. Significant decrease in the 0.9-1.17 kHz EOAE spectrum band, induced with a 1 kHz tone pip in the ipsilateral ear, was observed with 50 dB contralateral NBN centered at 0.75, 1.0 and 1.5 kHz. With the 2 kHz tone pip, the 1.9-2.1 kHz EOAE spectrum decreased significantly with 1.0, 1.5 and 2.0 kHz-centered NBN elicitors.

In 1993, Chery-Croze et al. reported evidence of MOC frequency selectivity from studies on the suppressive effects of NBN contralateral acoustic stimulation, with an ascending and descending slope of 24 dB/octave at 55 dB SPL, on the distortion product $2f_1-f_2$ of 1 and 2 kHz. The reported data was an average across subjects and the significance in suppression was analyzed using the Student’s t-test. They observed greatest decreases when the contralateral NBN was centered close to the DPOAE. When the $2f_1-f_2$ was fixed at 2 kHz, NBN centered at 1 kHz below the DPOAE induced significant suppression.

Lastly, Maison et al. (2000) reported an experimentally measured plot of EOAE attenuation as a function of contralateral stimulus bandwidths centered at various frequencies away from the 1 kHz tone pip. Again, the data was averaged across subjects. Significant suppression was observed with NBN elicitors centered less than half an octave above and below the nominal center frequency tested.

In the present study, we have clearly demonstrated efferent inhibition from NBN elicitors at much further distance from the test frequency (2 and a half octaves). In accordance with the current belief that the MOCB control on cochlear response involves the inhibition of OHCs onto which they synapse (Guinan, 1986, Liberman and Brown 1986), the dispersion of effective elicitor center frequencies may be explained by the spanning of each MOC efferent fiber on several OHCs (Brown, 1989) as well as the extent to which OHCs at a specific cochlear place affects the cochlear response over a range of frequencies (Guinan, 1996).

The normalized response curves, referenced to the maximum response magnitude in dB SPL, exhibit no obvious similarity in shape. Even with close test frequencies like 1.01 and 1.03 kHz, the contralateral response curves obtained from the different subjects using the 2450-2950 ms analysis window show different tuning patterns for the range of
elicitor used – the former appearing more bandpass while the latter more highpass. The normalized responses were also generally asymmetric on a semi-logarithmic frequency scale and the maximum response usually did not line up with the test frequency. These differences suggest that we cannot assume the existence of a generic response across subjects or even within a subject at different test frequencies.

At this point, it is worth noting that our observations of the detailed tuning pattern are by no means conclusive. Evidence of significant intra-subject variability in the efferent response induced under the same experimental conditions disables us from making any convincing observations on the shape of the response curves. More repeated measurements are needed before any statistically sound estimates can be made. In existing literature, response variability within a subject is usually ignored in data analysis. Perhaps this variability within a subject as well as the variability across subjects under the same experimental parameters contributed to the narrower frequency selectivity observed in the 3 examples (Vieullet et al., Chery-Croze et al., Maison et al.) where data was averaged across subjects.

The MOC tuning curves obtained at a test frequency 0.77 kHz for the contralateral response in the right and left ear as well as the binaural response in the right ear gave Q20 values that are similar in value. The values indicate that efferent tuning is wider than afferent tuning in humans (Shera et al. 2002).

The set of experiments in which we studied the efferent effect as a function of the bandwidth of the elicitor centered at the test frequency also showed that efferents integrate sounds over a wide range of frequencies. The noise bands in this study include 0.125, 0.25, 0.50, 1.0, 2.0, 4.0 octaves NBN and a BBN. The effect of broad band noise, whose spectrum was constant in energy level from 100 Hz to 10 kHz, was also included. The results showed that in almost all cases there is an increase in efferent response with increasing noise bandwidth. The binaural response appears to saturate somewhere between 2-4 octaves. This trend of increasing response with elicitor bandwidth is in agreement with those of Maison et al (2000) where they report increasing efferent-induced suppression in EOAE with increasing bandwidth of the contralateral NBN elicitor. However, their study only included bandwidths of 0.125, 0.25, 0.5, 1.0 and 2.0 octaves and only contralateral sound. This trend may be explained by modeling the peripheral auditory system as an array of overlapping bandpass filters or channels and that the larger MOC activation with wider band stimuli results from a summation of inputs from more channels to the Cochlear Nucleus which ultimately relays the signal to the medial part of the SOC where the cell bodies of the MOCB neurons are located.

Maison et al. (2000) also normalized their data with the number of equivalent rectangular bandwidths (ERBs) that fall between the upper and lower cutoff frequency of the contralateral NBN elicitor. However, a more recent study of otoacoustic and behavioral measurements demonstrated that tuning in the human cochlea at low sound levels is more than twice as sharp as tuning perceived with standard estimates from traditional behavioral measurements and casts doubt on the analysis (Shera et al. 2002).

With regards to the relative strength of the responses, the binaural stimulation is almost always the largest. In the experiment where the center frequency of a 0.5 octave NBN was varied, the relative strength between the ipsilateral and contralateral response appeared to vary with test frequency, elicitor band used and the subject. In the experiment where the elicitor bandwidth was varied while keeping its center frequency at the test
frequency, there is too little data to show whether the ipsilateral or contralateral response is larger. As a first approximation, however, their relative strength appears to be comparable.

Future Studies

It would be very interesting and useful to quantify in some way the statistics of intra-subject efferent response variability. A better understanding of this variability appears to be an important prerequisite before the functional capacity of Human MOCB can be clearly understood. If some tuning patterns can be well-estimated statistically, we can then begin to probe the functionality of the system.

The response curves here are limited to test tones within mid-frequency ranges. We intend to extend the study to higher and lower frequency regions. The difficulty of studying low frequencies involves the extraction of the efferent response which is small compared to the level of physiological noise floor. At higher frequencies, we are then limited by the transfer function of the middle ear which is effective at around 1 kHz in humans. On a different note, to capture finer details of the tuning pattern, noise bands smaller than 0.5 octave would be needed. However, narrower noise bands may be less able to elicit efferent activity large enough to be captured experimentally.
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