Suppression tuning of distortion-product otoacoustic emissions: Results from cochlear mechanics simulation

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(Received 4 July 2012; revised 28 November 2012; accepted 20 December 2012)

This paper presents the results of simulating the acoustic suppression of distortion-product otoacoustic emissions (DPOAEs) from a computer model of cochlear mechanics. A tone suppressor was introduced, causing the DPOAE level to decrease, and the decrement was plotted against an increasing suppressor level. Suppression threshold was estimated from the resulting suppression growth functions (SGFs), and suppression tuning curves (STCs) were obtained by plotting the suppression threshold as a function of suppressor frequency. Results show that the slope of SGFs is generally higher for low-frequency suppressors than high-frequency suppressors, resembling those obtained from normal hearing human ears. By comparing responses of normal (100%) vs reduced (50%) outer-hair-cell sensitivities, the model predicts that the tip-to-tail difference of the STCs correlates well with that of intra-cochlear iso-displacement tuning curves. The correlation is poorer, however, between the sharpness of the STCs and that of the intra-cochlear tuning curves. These results agree qualitatively with what was recently reported from normal-hearing and hearing-impaired human subjects, and examination of intra-cochlear model responses can provide the needed insight regarding the interpretation of DPOAE STCs obtained in individual ears.

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PACS number(s): 43.64.Kc, 43.64.Jb [CA]

Pages: 951–961

I. INTRODUCTION

How do we measure a person's cochlear filtering bandwidth? This question is difficult to answer due to lack of non-invasive techniques to measure human intra-cochlear acoustic responses directly. Recently, Gorga *et al.* (2011b) obtained indirect estimates of cochlear tuning sharpness of normal-hearing (NH) humans from distortion-product (DP) otoacoustic emissions (OAEs) suppression data (Gorga *et al.*, 2011a). The measurements of Gorga *et al.* have also been repeated on subjects that have mild to moderate hearing loss (25 to 45 dB) of cochlear origin (Grulke *et al.*, 2012). Surprisingly, the sharpness of the distortion-product otoacoustic emission (DPOAE) suppression tuning curves (STCs) of the hearing-impaired (HI) group is similar to that of the NH group when compared at the same stimulus conditions. In the present work, we present results of simulated DPOAE suppression based on a computer model of cochlear mechanics. The model has previously been adjusted to produce realistic DPOAEs for a wide range of stimulus conditions (Liu and Neely, 2010). To follow up, this paper presents (1) a comprehensive set of simulated DPOAE suppression data at various stimulus conditions, (2) simulation of impaired cochlear responses by lowering the mechano-electrical transduction (MET) sensitivities of the outer hair cell (OHC), (3) comparison between the DPOAE STCs and the intra-cochlear frequency responses of the “normal” and the “impaired” models, and (4) comparison of the results of simulation against human

data. We are not aware of any previous computer simulations of DPOAE STCs, so these comparisons may provide the needed insight regarding the interpretation of suppression tuning data obtained in individual ears.

Sharpness of tuning in human hearing can be either estimated from psychoacoustic experiments or inferred from OAE measurements. In a psychoacoustic experiment, a subject is typically asked to detect a narrowband probe signal that is presented with a masker at a nearby frequency (e.g., Vogten, 1978; Moore *et al.*, 1984). The masker can either be presented simultaneously with the probe or it can precede the probe by a short duration (Fastl and Zwicker, 2007); the latter procedure is referred to as a *forward-masking* experiment. In either case, the level of the masker is adjusted so as to determine the threshold at which masking just happens. Then, the frequency of the masker is varied, and the masking threshold is plotted against masker frequency to obtain a psychoacoustic tuning curve (PTC). The *Q*-values of PTCs obtained from a simultaneous-masking procedure typically range between 7 and 12 and increase from low to high frequency (e.g., Glasberg and Moore, 1990; Oxenham and Snera, 2003). The *Q*-values of forward-masking PTCs are higher, increasing from 10 at 1 kHz to nearly 20 at 8 kHz (Oxenham and Snera, 2003). Although measurement of PTCs can be made rigorous and sufficiently unbiased by loss of attention, it often requires the subjects to participate actively for long hours in the lab. This may limit its feasibility for clinical applications when time is precious or when a patient is not capable of doing so.

One possible alternative to measure the frequency selectivity of the cochlea is to infer it from measurement of

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stimulus-frequency otoacoustic emissions (SFOAEs), which refer to cochlear emissions in response to pure-tone stimuli (Shera *et al.*, 2002). Theoretic analysis showed that SFOAEs can result from partial reflection of cochlear traveling waves due to random perturbations in cochlear mechanical properties (Zweig and Shera, 1995; Shera and Guinan, 1999). The reflection is prominent when cochlear tuning is *tall and broad*, in this case the phase-gradient latency of SFOAEs equals the round-trip traveling time between the stapes and the characteristic place in the cochlea.¹ It has been observed that in many other mammalian species the sharpness of cochlear tuning is proportional to the phase-gradient delay of SFOAEs (Shera and Guinan, 2003; Shera *et al.*, 2010). Thus, if the same relation holds for humans, SFOAEs would provide a way to estimate human cochlear tuning objectively and non-invasively.

In the theory of linear filtering, correlation between sharpness of tuning and group delay is a feature of *minimum-phase* systems. When a linear and time-invariant system is minimum-phase, the system's magnitude response uniquely determines the phase response and its phase response determines the magnitude response up to a scaling factor (Oppenheim and Schaffer, 2010). This lays the foundation of inferring the sharpness of tuning (inversely proportional to the bandwidth of magnitude spectrum) from the group delay (inversely proportional to the transition bandwidth of phase spectrum). The minimum-phase property was first observed in basilar-membrane transfer functions by Zweig (1976) but has been questioned in recent years based on results of laser vibrometry measurements (Ruggero and Temchin, 2007; Recio-Spinoso *et al.*, 2011).

To estimate cochlear frequency selectivity from OAE measurements without assuming a correlation between latency and sharpness, one can resort to *suppression tuning* experiments. Both SFOAE and DPOAE suppression experiments have been conducted on humans (Keefe *et al.*, 2008; Bentsen *et al.*, 2011; Brown and Kemp, 1984; Gorga *et al.*, 2011a) as well as other animals (Guinan, 1990; Brown and Kemp, 1984; Martin *et al.*, 1987, 1998). In these experiments, a fixed probe stimulus [which consists of two primary tones for DP experiments or a single tone for stimulus-frequency (SF) experiments] was delivered and the level of OAEs was monitored with or without introducing a suppressor tone. The presence of the suppressor decreased the level of OAEs, and the amount of decrement depended upon the frequency and level of the suppressor. Thus, tuning curves were derived either by plotting the frequency dependence of the required suppressor level for a fixed amount of OAE decrement (Brown and Kemp, 1984; Gorga *et al.*, 2011a), or by plotting the dependence of OAE decrement against the frequency of suppressors at a fixed level (Bentsen *et al.*, 2011).

The advantage of OAE suppression measurement is obvious—an entire tuning curve is derived, in contrast to OAE latency measurement from which only the sharpness of tuning is inferred. However, suppression tuning experiments seemed rather to provide information about the bandwidth of cochlear amplification processes than the bandwidth of cochlear transfer function *per se* (Keefe *et al.*, 2008). Bentsen *et al.* (2011) measured SFOAE phase-gradient latency and conducted

SFOAE suppression tuning experiments using a two-tone suppression (2TS) paradigm on the same listeners. They found that the Q -value of SFOAE STCs was significantly lower than that was inferred from SFOAE phase-gradient latency. This finding led to a conclusion that the 2TS estimate of cochlear tuning probably represents the suppression mechanisms rather than the cochlear filtering bandwidth. Compared to SFOAE STCs, DPOAE STCs might be even harder to interpret because DPs are known to be generated across a broad region in the cochlea (Gorga *et al.*, 2011a). In the literature, the Q -values of the DPOAE STCs in NH humans (Gorga *et al.*, 2011a), ranging from 4 to 10 at the lowest stimulus level tested, were similar to those of SFOAE suppression tuning (Keefe *et al.*, 2008; Bentsen *et al.*, 2011), similar to those of PTCs as derived from simultaneous-masking experiments (Glasberg and Moore, 1990) but lower than those measured from forward-masking experiments (Oxenham and Shera, 2003) or inferred from phase-gradient latencies of SFOAEs (Shera *et al.*, 2002; Bentsen *et al.*, 2011).

Results from these experiments raise a general question: How do OAE suppression-tuning responses relate to the filtering mechanisms inside the cochlea? The advantages of a computer modeling approach are two-fold: First, it is easy to look at the responses inside the model cochlea, and secondly, one can change the physical parameters and observe systematically how intra-cochlear and suppression-tuning responses vary. In the present work, we aimed to investigate the relation between DPOAE suppression tuning and intra-cochlear tuning using a computer model of cochlear mechanics developed in recent years (Liu and Neely, 2009, 2010). Section II reviews the cochlear mechanics model; in particular, a simulation was conducted under an impaired setting by reducing the OHC sensitivity and results are compared to those of the original, normal setting. Section III describes the procedures to repeat the experiments of Gorga *et al.* (2011b) in computer simulations, and Sec. IV presents the suppression growth function (SGF) and STCs derived thereafter. The STCs are compared against the tuning curves obtained inside the model cochlea and discussions of the simulation results, including comparisons against human data, are given in Sec. V.

II. PROPERTIES OF THE COCHLEAR MECHANICS MODEL

A previously developed model of cochlear mechanics (Liu and Neely, 2010) was adopted for computer simulation. For any given acoustic stimulus in the ear canal, the model calculates the motion of the middle ear and traveling waves along the cochlea in both the forward and reverse directions, using a time-domain finite difference method. The traveling waves were amplified through a feedback mechanism provided by OHC electromotility; the feedback was nonlinear due to properties of the MET channels located in the hair bundles of OHCs.

Parameters of the model were chosen to be physically realistic—such as elasticity, mass, and resistance of the cochlear partition, and the membrane capacitance, the hair-bundle transconductance, and somatic electromotility of the OHCs [numerical values were listed in Liu and Neely (2010)]. The

model has been shown to produce dynamic range compression on the basilar membrane that is typical of lab animals (Ruggero *et al.*, 1997) and DPOAEs that are typical of humans (Johnson *et al.*, 2006). The model also showed quasi-linear filtering effects for broadband noise stimuli (de Boer, 1997; Liu and Neely, 2012), which agree with recently reported experimental findings (Recio-Spinoso *et al.*, 2009). Important details of the model are described below.

A. Linear analysis: The traveling waves and the NDR

The computer model was constructed to simulate the propagation of cochlear traveling waves along a single direction x . The time and spatial derivatives of two wave variables, namely the acoustic pressure $p(x, t)$ and the volume velocity of cochlear fluid $u(x, t)$, are related by Newton's second law

$$\partial_x p(x, t) = -\frac{\rho}{A(x)} \partial_t u(x, t), \quad (1)$$

where $A(x)$ is the cochlear cross sectional area and ρ is the fluid density. To analyze the model's responses in the linear regime, it is convenient to apply Laplace's transform to all the time-varying functions so Eq. (1) becomes

$$\partial_x P(x, s) = -z(x, s)U(x, s), \quad (2)$$

where P and U are the Laplace transform of p and u , respectively, and $z(x, s) = s\rho/A$ is the series impedance per unit length. Similarly, the spatial derivative of $U(x, s)$ is related to $P(x, s)$ as follows:

$$\partial_x U(x, s) = -y(x, s)P(x, s), \quad (3)$$

where $y(x, s)$ is a shunt admittance that characterizes the transverse velocity of the cochlear partition in response to the pressure difference between the scala vestibuli and the scala tympani. It is assumed in the model that $y(x, s)$ relates to an OHC feedback transfer function $H(x, s)$ in the following manner (Liu and Neely, 2009):

$$y(x, s) = G(x) \cdot \frac{1}{Z_b(x, s)[1 + H(x, s)]}, \quad (4)$$

where $Z_b(x, s)$ describes the mechanical impedance of the basilar membrane, and $G(x)$ is a frequency-independent scaling factor that depends on the geometric structure of the organ of Corti. Given the series impedance and the shunt admittance, a propagation function $k(x, s)$ can be calculated

$$k(x, s) = \sqrt{z(x, s) \cdot y(x, s)} \quad (5)$$

$$= -\gamma(x, s) + j\kappa(x, s), \quad (6)$$

where $\gamma(x, s)$ and $\kappa(x, s)$ denote the attenuation rate along the cochlea and the wavenumber function, respectively, the unit of both being 1/cm. Note that $k(x, s)$ depends on the OHC feedback loop gain $H(x, s)$; when $H(x, s) = 0$, the cochlea reduces to a purely passive system and the wave can propagate only in places where $y(x, s)$ is stiffness-dominated [because $z(x, s)$ is

always mass-dominated in the model]. Next, we summarize how the OHC feedback gain $H(x, s)$ is calculated.

B. OHC feedback and its role in traveling waves amplification

The present modeling of the OHC began with consideration of the MET in the stereocilia. The MET channels were assumed to act like a combined velocity and displacement sensor, such that the receptor current i_r relates to the reticular-lamina (RL) displacement ξ_r by the following non-linear equation:

$$i_r(t) = I_{\max} \tanh\left(\frac{\alpha_v \dot{\xi}_r + \alpha_d \xi_r}{I_{\max}}\right), \quad (7)$$

where α_v and α_d denote the velocity- and displacement-sensing constant, respectively,² and $I_{\max}$ denotes the maximal level of alternating current flowing into the OHC. When the stimulus level is sufficiently low, the model can be considered to operate in the linear regime, and an OHC forward transduction ratio H_f , can be defined as follows:

$$H_f(x, s) = \alpha_v + s^{-1}\alpha_d. \quad (8)$$

The unit of $H_f(x, s)$ is [A/(cm/s)]. As shown in Eq. (7), when the stimulus level is high, the receptor current saturates so nonlinear responses such as distortion products can be observed (to be discussed in Sec. II C).

Next, somatic motility of the OHC was characterized by an electro-mechanical transduction ratio T , its unit being displacement per charge accumulation [cm/C], or velocity per unit amount of current [(cm/s)/A], equivalently. The product of the transduction ratios H_f and T sets an open-loop gain for the OHC feedback; however, due to factors such as electrical leakage of the membrane and mechanical load of the OHC, the gain is modified so a close-loop gain $H(x, s)$ is calculated as follows:

$$H(x, s) = \frac{H_f(x, s) \cdot T}{1 + (G + sC)(T^2 Z_{\text{OHC}}(s) + 1/sC_g)}. \quad (9)$$

In Eq. (9), the OHC membrane leakage conductance G , the membrane capacitance C , the gating capacitance C_g that is the signature of somatic motility, and the mechanical impedance $Z_{\text{OHC}}(s)$ which the OHC contraction force pulls upon, were all set to vary smoothly from the base to the apex in the present model of the cochlea.

Substituting Eq. (9) into Eq. (4) and using Eq. (6), we can identify where in the model and at what frequencies do the traveling waves experience *negative damping* {i.e., where $\text{Re}[k(x, s)] > 0$ }. A previous analysis showed that when α_v and C_g are properly chosen, every place x in the current model can provide negative damping within a certain frequency range up to its characteristic frequency (Liu and Neely, 2009). Thus, traveling waves at any fixed frequency experience amplification in the model by means of negative damping when passing through a negative damping region (NDR) (Neely and Kim, 1983), and for every frequency, the associated NDR is located basal to its characteristic place.

FIG. 1. Magnitude spectrum along the model cochlea: Normal (solid line) vs impaired (dotted-dashed line). RL displacement gain with respect to stapes is plotted against frequency at six places along the cochlea (5 to 30 mm from the stapes in 5-mm steps). Hearing loss was simulated by introducing 50% of reduction in the OHC MET sensitivity parameters.

One advantage of computer “experiments” is the relative ease of changing parameters to see how responses vary. To demonstrate, we simulated OHC-originated hearing loss by introducing a 50% reduction in the MET sensitivity parameters α_v and α_d [see Eq. (7)] throughout the entire model cochlea. Magnitude responses inside the “NH” model and the “HI” model are shown in Fig. 1 for comparison purposes. Note that the loss of OHC sensitivity causes the magnitude spectrum to become shorter in height, and the sharpness near the tip of the spectrum is also reduced. The height and the sharpness of transfer functions (or tuning curves) will be defined and analyzed quantitatively in Sec. IV when more data from simulation are presented.

C. Cochlear nonlinear responses: DPs and 2TS

The present model of cochlear mechanics assumed that the only site of nonlinearity is at the MET channel of the OHCs, as shown in Eq. (7). Because of this nonlinearity, the present model has been shown to produce dynamic range

FIG. 2. (Color online) Model responses to two-tone stimuli. (A) and (B) show, respectively, the magnitude and phase of RL displacement along the original (NH) model cochlea. (C) and (D) show the responses from a HI model in which the OHC MET transduction ratio is reduced by 50%. In each panel, the solid lines show responses to moderate primary levels at $L_1 = 30$ dB and $L_2 = 30$ dB SPL, and the dashed lines show responses to a much higher $L_1 = 70$ dB while keeping L_2 unchanged. The primary levels (L_2, L_1) in dB SPL are shown in the legends. The primary frequencies are fixed at $f_2 = 4$ kHz and $f_1 = f_2/1.22$. In all cases, the cubic DP at frequency $2f_1 - f_2$ reaches its maximum magnitude near its characteristic place [(A) and (C), near 1.4 cm]. The phase of the DP is most advanced near where the f_2 -magnitude is peaked [e.g., (B), near 1.0 cm], indicating that the DP is generated in that vicinity and subsequently propagates in both the forward and backward direction.

compression inside the model cochlea as well as DPOAEs out of it (Liu and Neely, 2010). What was previously not reported was the DP generation mechanism inside the model. Figure 2 shows the model's responses to a two-tone complex that consists of frequencies $f_1 = 3283$ and $f_2 = 4000$ Hz ($f_2/f_1 = 1.22$). The level of the 4-kHz tone is fixed at $L_2 = 30$ dB while L_1 is varied so changes in the responses along the model cochlea can be observed and analyzed. When both tones are at low-to-moderate levels ($L_2 = 30$ and $L_1 = 30$ dB), Fig. 2(A) shows that the distortion product at $f_{DP} = 2f_1 - f_2$ can be observed along the cochlea (see the light green solid line); its level grows to reach a maximum at a location near 1.4 cm from the stapes, which is the characteristic place for the DP frequency. Figure 2(B) shows that the phase of DP is most advanced at approximately the 1.2-cm place, where the excitation pattern of f_1 and f_2 tones overlap. This indicates that DP is generated in that vicinity and propagates in both the forward and backward direction.

When L_1 is increased from 30 to 70 dB, the DP level generally increases inside the model cochlea (see the light green dashed line) but the response to the f_2 tone is *suppressed* (comparing the black solid line and the black dashed line). The model predicts that, due to the strong presence of the f_1 tone inside the model cochlea, the best place (BP) of f_2 is shifted basally [Fig. 2(A)] and so is the generation site of DP [Fig. 2(B)].

Two-tone responses of the HI model are shown in Figs. 2(C) and 2(D). Compared to NH results in Fig. 2(A), the magnitudes of both the f_1 and f_2 components are reduced by about 16 dB at their respective characteristic places, and consequently the magnitude of DP is also reduced. For input levels $(L_2, L_1) = (30, 70)$ dB sound pressure level (SPL), the DP displacement magnitude dropped by about 20 dB as compared against NH [and for $(L_2, L_1) = (30, 30)$, the DP magnitude is too low to be viewed in the plot]. In Fig. 2(C), we also note that the suppression of the f_2 tone by the f_1 tone is not as prominent as in Fig. 2(A).

The purpose of plotting the intracochlear nonlinear responses is to illustrate an example of (1) how the DPs are generated in the model, (2) how it is linked to 2TS, and (3) how both of them change when the OHC sensitivity in the model is lowered. A more comprehensive study of these mechanisms is beyond the scope of the present study and left as a potential future research direction. Presented next are our attempts to introduce yet another tone and suppress the existing two-tone complex and their distortion products thereof. Stimulus conditions described immediately below were set similar to those reported by Gorga *et al.* (2011a) and Gruhlke *et al.* (2012).

III. SIMULATION OF DPOAE SUPPRESSION: STIMULUS CONDITIONS

To simulate suppression of DPOAEs, the stimulus conditions were set as follows:

- (1) The primary tone frequencies were $f_2 = 8, 4, 2, 1$, or 0.5 kHz.
- (2) The primary-tone frequency ratio was fixed at $f_2/f_1 = 1.22$.

- (3) The primary-tone level L_2 was 20, 30, 40, or 50 dB SPL.³
- (4) For every L_2 , the primary-tone level L_1 was determined by $L_1 = 0.4L_2 + 49$ (dB SPL).⁴
- (5) For fixed (f_1, f_2) and (L_1, L_2) , the suppressor frequency f_{sup} was varied between $0.5f_2$ and $1.5f_2$ so an STC could be obtained. Twenty-three equally spaced suppressor frequencies were tested: $f_{sup} = \{129, 139, 149, \dots, 339, 349\}$ times $f_2/240$, respectively.

Before running DPOAE suppression simulations, the cubic-distortion levels L_{DP}^0 at $f_{DP} = 2f_1 - f_2$ were recorded for all combinations of f_2 and L_2 . Then, for each (f_2, L_2) -combination, a pure-tone suppressor was introduced. For each fixed suppressor frequency, the suppressor level L_{sup} was increased from 20 to 90 dB SPL in 5-dB steps and the level L_{DP} of DPOAE was recorded by monitoring the short-time Fourier transform (Allen and Rabiner, 1977) of the acoustic pressure in the ear canal.⁵

Conceptually, L_{DP} should decrease as L_{sup} increases; this can be expected by referring to Fig. 2—one can imagine that the presence of the suppressor tends to reduce both the magnitude of f_1 and f_2 inside the cochlea, and subsequently the DPs due to the nonlinear interaction between the two primary tones. The suppressor level $L_{sup} = \theta$ that is required to decrease L_{DP} by 3 dB can be regarded as a suppression threshold. Any further increase in L_{sup} should cause L_{DP} to decrease further. For primary tones at fixed (L_1, L_2) and (f_1, f_2) , the threshold θ should vary as suppressor frequency f_{sup} changes. Therefore, a tuning curve can be obtained by plotting θ against f_{sup} .

A linear-regression procedure (Gorga *et al.*, 2011b) was adopted to estimate θ at each given f_{sup} and fixed (L_1, L_2) and (f_1, f_2) . The procedure fits a straight line that best describes the SGF (that is, DPOAE decrement $\Delta = L_{DP}^0 - L_{DP}$ as a

FIG. 3. Suppression threshold estimation. This curve-fitting method assumes that the DPOAE SGF is approximately given by $\Delta = 10 \log_{10}(1 + 10^{a(L-\theta)/10})$, where Δ is the DPOAE decrement, θ is the 3-dB decrement threshold, and a is an asymptotic slope of the growth function (dB/dB). The two curves with filled and empty markers, respectively, show the growth function with different values of θ and a . Note that at $L \gg \theta$, we have $\Delta \approx a(L - \theta)$, which are shown as solid lines. To find the asymptotic line from suppression growth data, a transformed variable is defined as $D = \log_{10}(10^{\Delta/10} - 1)$; then, we conduct a least-square linear fit of D against L . The intersection of the regression line with $\Delta = 0$, marked with a square, gives an estimate of the threshold θ .

TABLE I. Definition of mathematical symbols.

Symbol	Definition
L_1, L_2	Level of primary tones
f_1, f_2	Frequency of primary tones ($f_2 > f_1$)
L_{sup}	Suppressor level
f_{sup}	Suppressor frequency
f_{DP}	Cubic distortion frequency $2f_1 - f_2$
L_{DP}	Level of DPOAE at f_{DP}
L_{DP}^0	Level of DPOAE when no suppressor is present
Δ	DPOAE decrement; $\Delta = L_{\text{DP}}^0 - L_{\text{DP}}$.
θ	Estimated suppression threshold for 3-dB decrement in DPOAE
$\Delta(L_{\text{sup}})$	SGF
$\theta(f_{\text{sup}})$	STC

function of L_{sup} at a high suppressor level L_{sup} . An intersection of the line and the L_{sup} -axis produces an estimate of θ . Details of the fitting procedure are explained in Fig. 3. A definition of mathematical symbols are summarized in Table I.

IV. DPOAE SUPPRESSION GROWTH FUNCTIONS AND TUNING CURVES

A. Suppression growth functions

Figure 4 shows the DPOAE decrement $\Delta = \Delta(L_{\text{sup}})$ as a function of suppressor level for the primary frequencies $f_2 = 4$ kHz and $f_1 = f_2/1.22$. Suppression-growth functions $\Delta(L_{\text{sup}})$ were shown across 12 suppressor frequencies (out of 23 suppressor frequencies tested) and at 4 primary-tone levels ($L_2 = 20$ to 50 dB SPL in 10-dB steps).

By inspection, a few general trends are noted. First, for fixed f_2 and f_{sup} , the SGF $\Delta(L_{\text{sup}})$ usually shifts to the right as

L_2 increases from 20 to 50 dB. In other words, it usually takes a higher level of L_{sup} to suppress DPOAE by a certain amount (in dB) for high-level primary tones than for low-level primary tones. This agrees with the argument that a suppressor needs to dominate the motion at DP generation sites, primarily near the f_2 -BP, for suppression to be effective (Brown and Kemp, 1984). As primary-tone levels increase, the required dominance level for the suppressor also increases. This rightward shift of ΔL_{sup} is, however, not as orderly for low-frequency suppressors (e.g., $f_{\text{sup}} = 2318$ or 2650 Hz) as for high-frequency suppressors (e.g., $f_{\text{sup}} \geq 4318$ Hz).

Secondly, the rate at which $\Delta(L_{\text{sup}})$ increases against L_{sup} is generally more rapid for low-frequency suppressors (e.g., $f_{\text{sup}} = 2985$ Hz) than for high-frequency suppressors (e.g., $f_{\text{sup}} = 4984$ or 5316 Hz). This result agrees qualitatively with experimental data (Gorga et al., 2011b) and can be attributed to the basalward shift of f_{sup} -BP as L_{sup} increases (McFadden, 1986)—for a low-frequency suppressor at $f_{\text{sup}} < f_2$, the basalward shift of its BP along the cochlea makes it easy to “mask” activities near the f_2 -BP so the DPOAE level drops rapidly as L_{sup} increases beyond the threshold θ . If fact, the basalward shift is shown in Fig. 2 as well [in Fig. 2(A), comparing the excitation pattern of $L_1 = 70$ dB against $L_1 = 30$ dB]. For a high-frequency suppressor at $f_{\text{sup}} > f_2$, however, a basalward shift means the excitation pattern associated with the suppressor moves away from the f_2 -BP, making it difficult to produce a large DPOAE decrement. Therefore, its SGF is shallower compared to low-frequency suppressors.

Listed below are a few other noteworthy details in the SGFs shown in Fig. 4:

FIG. 4. (Color online) DPOAE SGFs at $f_2 = 4$ kHz and $f_1 = f_2/1.22$. In each panel, suppressor frequency f_{sup} (Hz) is printed at the top-left corner and the DPOAE decrement is plotted against the suppressor level. Markers indicate primary-tone levels (L_1, L_2): $\circ$ is (57, 20); ∇ is (61, 30); $\triangle$ is (65, 40), and $+$ is (69, 50); in dB SPL.

- (1) Some of the SGFs for low frequency suppressors (e.g., $f_{\text{sup}} = 3651$ Hz) do not monotonically increase against L_{sup} .
- (2) Continuing from above, in a few cases the amount of suppression can be negative at certain L_{sup} (e.g., $f_{\text{sup}} = 2650$ Hz, $L_{\text{sup}} = 55$ dB SPL).
- (3) Even when an SGF is monotonic, the shape of the function may not fit well with the formula

$$\Delta(L) = 10 \log_{10}(1 + 10^{a(L-\theta)/10}), \quad (10)$$

which is depicted in Fig. 3. For instance, for $f_{\text{sup}} = 4649$ Hz and for all $L_2 = 40$ dB, the SGF resembles piece-wise linear functions with break-points at L_{sup} near 45, 55, and perhaps 75 dB, respectively.

The details mentioned above indicate that Eq. (10) does not adequately describe the SGFs obtained in simulation. Consequently, the threshold estimation method illustrated by Fig. 3 might produce results that are sensitive to data exclusion rules (see Sec. IV B) and difficult to interpret. Nevertheless, our current best attempts in plotting DPOAE STCs—that is, the estimated suppression threshold against f_{sup} for all combinations of f_2 and L_2 —are reported in Sec. IV B.

B. STCs

For each combination of (f_2, L_2) , a tuning curve $\theta = \theta(f_{\text{sup}})$ was derived from SGFs $\Delta(L_{\text{sup}}; f_{\text{sup}})$ using the method depicted in Fig. 3 with a few data-exclusion rules: First, all points with an insignificant decrement $\Delta < 0.02$ dB were excluded mainly to avoid problems caused by negative

Δ ; secondly, all points with $\Delta > 20$ dB were excluded mainly to avoid problems caused by piecewise linearity of the growth function beyond its nominal threshold. Tuning curves obtained after such processing are shown in Fig. 5.

Most of these tuning curves show that suppression is most effective when the suppressor frequency is close to f_2 . However, some curves show double local minima, one near f_2 and the other at approximately 0.25 octave higher (see arrows in Fig. 5, e.g., the tuning curve for $f_2 = 2$ kHz and $L_2 = 20$ dB SPL; again for $f_2 = 4$ kHz and $L_2 = 30$ dB SPL). In Sec. V, we shall discuss why these double-tips exist in the STCs and their impact on succeeding data analyses.

C. General trends in the shape of tuning curves

By inspecting the tuning curves in Fig. 5, one might attempt to recognize how the shape of tuning curves varies with primary-tone levels and frequencies. To describe the shape of tuning curves in a quantitative manner, we adopted the definition of *tip-to-tail difference* (TTD) and Q_{ERB} given by Gorga *et al.* (2011a): For a tuning curve $\theta = \theta(f_{\text{sup}})$, its TTD is defined as

$$\text{TTD} = \theta(f_2/2) - \theta_m, \quad (11)$$

where $\theta_m = \min \theta(f_{\text{sup}})$ is the minimum of the tuning curve. The *equivalent-rectangular-bandwidth* (ERB) Q -factor of a tuning curve is defined as

$$Q_{\text{ERB}} = f_2 / \Delta f, \quad (12)$$

where Δf is the ERB estimated by the following formula:

FIG. 5. (Color online) DPOAE STCs derived from suppressor growth functions. Markers, identical to those in Fig. 4, indicate primary-tone levels (L_1, L_2) in dB SPL— $\circ$ is (57, 20); ∇ is (61, 30); $\triangle$ is (65, 40), and $+$ is (69, 50), respectively. The arrows point to additional local minima other than the ones near f_2 in some STCs.

FIG. 6. Tip-to-tail differences and Q_{ERB} of DPOAE STCs shown in Fig. 5. Markers indicate the primary tone frequency f_2 .

$$\Delta f = \int_{f_i}^{f_h} 10^{-\{\theta(f) - \theta_m\}/10} df. \quad (13)$$

Here, $[f_i, f_h] \simeq [0.5f_2, 1.5f_2]$ is the range of suppressor frequencies tested in simulation.

In Fig. 6, TTD and Q_{ERB} are plotted against L_2 at different f_2 's. Note that the TTD decreases monotonically as a function of L_2 for every f_2 . In contrast, Q_{ERB} does not exhibit a clear decreasing trend against L_2 . At fixed L_2 , the Q -factor seems to have an increasing trend against f_2 , with the 0.5-kHz tuning curve being the least sharply tuned and the 8-kHz curve the most sharply tuned. However, our results show that Q_{ERB} does not increase monotonically against f_2 at any fixed L_2 .

D. Simulation of DPOAE STCs: Normal vs impaired

We attempted to simulate the hearing loss of an OHC-origin by reducing the MET sensitivity parameters by 50%. DPOAE suppression-tuning simulations were repeated in this impaired model for all conditions with $f_2 = 4000$ Hz and $f_1 = f_2/1.22$. The resulting STCs are shown in the right panel of Fig. 7 in comparison with those of the normal model. Generally speaking, a higher L_{sup} is required to suppress DPOAE in the impaired than in the normal model for it takes a stimulus at higher levels to saturate the OHC recep-

FIG. 7. Comparison of simulated DPOAE STCs in a normal (left panel) vs impaired (right panel) cochlear model. Stimulus conditions were set exactly the same for both simulations, but the impaired results were obtained by introducing a 50% reduction in the OHC MET sensitivity parameters α_p and α_d throughout the model cochlea.

FIG. 8. TTD (A) and Q_{ERB} (B) of DPOAE STCs at $f_2 = 4$ kHz; being compared are simulated data (NH-sim, HI-sim) and human data (dashed lines). NH and HI human data were published by Gorga *et al.* (2011a) and Gruhlke *et al.* (2012), respectively. Numerical values in the legend (e.g., 20 to 25) indicates the range of hearing loss in dB for each group of the HI subjects.

tor currents. Also note that the double-tips that occur in some normal STCs do not show up in any of the impaired STCs. Moreover, minima of STCs tend to be located at a frequency above f_2 for the normal curves but below for the impaired. These discrepancies will be discussed in Sec. V.

The TTD and Q_{ERB} of STCs in Fig. 7 were calculated and results are shown in Fig. 8. Human measurements (Gorga *et al.*, 2011a; Gruhlke *et al.*, 2012) are also shown for comparison. In general, the TTD and Q_{ERB} obtained from the present computer model are both lower than those obtained in human ears. Nevertheless, both TTD and Q_{ERB} show a decreasing trend against L_2 , in agreement with human results.

V. DISCUSSION

A. Comparing STCs and intra-cochlear tuning curves (ICTCs)

As pointed out by Gorga *et al.* (2011a), DPOAE STCs might represent cochlear excitation along a broad region. On one hand, because the probe signal consists of two tones instead of one, it is possible to suppress the level of DPOAEs by suppressing either the primary tone f_1 or f_2 . In animal models, it has been shown that DPOAE can be most effectively suppressed by introducing a suppressor near f_1 or f_2 (Fig. 5, Brown and Kemp, 1984). On the other hand,

FIG. 9. RL iso-displacement tuning curves measured at the characteristic place of 0.5, 1, 2, 4, and 8 kHz, respectively. For each place, the required displacements were varied in 10-dB steps from -10 to 50 dB (re: 1 nm).

TABLE II. TTD and Q_{ERB} of DP STCs vs ICTCs (*sup.* = suppression; *int.* = intra-cochlear).

Frequency	TTD (dB)		Q_{ERB}	
	<i>sup.</i>	<i>int.</i>	<i>sup.</i>	<i>int.</i>
0.5 kHz	19.7	16.7	3.64	4.67
1.0 kHz	29.4	23.9	3.95	5.53
2.0 kHz	31.4	29.9	5.01	6.40
4.0 kHz	29.8	29.4	4.85	6.97
8.0 kHz	24.4	24.6	5.06	7.45

suppression of DP may also be effective if the suppressor shuts down the traveling-wave amplification process in the NDR associated with the f_2 tone. This point will be discussed more thoroughly in Secs. VB and VC.

To check if the current computer model is more sharply tuned inside the cochlea than derived from DPOAE suppression tuning, we calculated the RL iso-displacement tuning curves, referred to as ICTCs hereafter, by varying the SF from 0.25 to 10 kHz and computing the responses at the characteristic place of 0.5, 1, 2, 4, and 8 kHz, respectively. The results are shown in Fig. 9. By inspection, the ICTCs vary more smoothly across frequency and intensity than the DPOAE STCs shown in Fig. 5; we do not find any double-tips, and the tails of tuning curves across different intensities do not cross over as in STCs. Also, the shift of the tip of tuning curves toward low frequency is prominent at high intensity for ICTCs but not for STCs.

Overall, these differences show that DPOAE STCs do not accurately represent ICTCs inasmuch as the present computer model predicts it. In Table II, we summarize TTD and Q_{ERB} of STCs and ICTCs at the lowest probe level; being compared here are the DPOAE STCs for primary tone level $L_2 = 20$ dB SPL and the ICTCs for iso-displacement at -10 dB relative to 1 nm.⁶ Results show that the TTDs of STCs tends to be higher than those of ICTCs (the only exception is at 8.0 kHz) while the Q_{ERB} of STCs is consistently lower than those of ICTCs.

B. Comparing simulated STCs to human data

The presence of double-tips in simulated DPOAE STCs limits the way we can interpret the Q_{ERB} data shown in Fig. 6. For instance, among 2-kHz STCs, it is not entirely reasonable to compare the Q_{ERB} of the curves at $L_2 = 20$ dB and $L_2 = 50$ dB because one of the curves has two sharp tips but the other has a broad valley instead.

In the analysis of Gorga *et al.* of human DP suppression-tuning data, double-tips were also observed in several individual tuning curves [Fig. 9 of Gorga *et al.* (2011a)]. When tuning curves were averaged across NH ears, however, none of the double-tips remained [Figs. 2 and 3 of Gorga *et al.* (2011a)].

The presence of the double-tips in DPOAE STCs might be due to the basalward spread of the NDR. The present model agrees with animal-experiment data in that, for every frequency, a cochlear traveling wave receives amplification through a NDR basal to its BP (Shera, 2007). In the DP-suppression context, if the NDR for f_2 is sufficiently spread toward the base of the cochlea, it is possible that a suppressor

can most effectively reduce the overall f_2 traveling-wave amplitude if it dominates the motion in the f_2 -NDR and shuts down the amplification process there. This requires the suppressor frequency to be slightly higher than f_2 and possibly gives rise to the second tip in some of the tuning curves in Fig. 5.

The tail region (i.e., near $f_{\text{sup}} = f_2/2$) of STCs is not simple to analyze, either. At any given suppressor frequency, the required suppressor level for a 3-dB DPOAE decrement can vary quite randomly against the primary-tone level; in many cases it is actually *easier* to suppress DPOAE of high-level primaries by 3 dB than to suppress DPOAE of low-level primaries by the same amount.

It is also quite peculiar that many of the STCs seem to reach a local maximum near $f_{\text{sup}} \approx 0.75 f_2$. Note that $0.75 f_2$ is close to the DP frequency $2f_1 - f_2 = 0.78 f_2$. This may imply that the suppressor can interact with the DP component near its BP, causing the phase of the reflected DP component to change and thereby altering the level of DPOAEs. Though not shown in this paper, this suppressor-DP interaction possibly explains the “negative suppression” observed in some SGFs for low-frequency suppressors (e.g., $f_2 = 8000$, $f_{\text{sup}} = 5299$ and 5969 Hz). When the DP component reflected from the BP interferes destructively with the “direct” DP component generated near the f_2 place, it is possible to increase the level of DPOAE by suppressing the reflected DP component.

Local maxima in the tail region also occurred in a few individual human DPOAE STCs [Fig. 9 of Gorga *et al.* (2011a)] but the occurrence is not common among subjects. Consequently, all the averaged human DPOAE STCs vary smoothly against frequency in the tail region [Fig. 2 of Gorga *et al.* (2011a)]. The smoothness of human DPOAE STCs in the tail regions probably owes to the fact that, during the DPOAE measurement, an optimal L_1 was chosen individually at every frequency. This may have eliminated the chance for destructive interference to happen between the reflected and direct components of DP.

In the present simulation, although tails of individual DPOAE STCs look complicated, it is rather surprising that monotonicity is preserved when TTD is plotted against L_2 for every f_2 (Fig. 6). This means that in the current model the TTD of STCs correlates well with the TTD of ICTCs because the TTD of ICTCs also decreases monotonically (Fig. 9, by inspection) at a sufficiently large displacement level. Therefore, between TTD and Q_{ERB} of DPOAE STCs, the current model asserts that TTD is the better indicator for healthy cochlear tuning.

This assertion coincides with the most recent comparison of DPOAE STCs between a NH group (Gorga *et al.*, 2011a) and a HI group (Gruhlke *et al.*, 2012). Subjects from the HI group, having hearing loss of 20 to 45 dB, showed a reduced TTD in their DPOAE STCs, and the amount of TTD reduction correlates positively with the severity of their hearing loss [Fig. 8(A)]. However, the Q_{ERB} ’s of their DPOAE STCs were similar to those of the NH group across different primary levels and frequencies [Fig. 8(B)]. No clear trends can be observed there against the severity of hearing loss. In several cases, HI subjects had an even higher Q_{ERB} than those of NH subjects. This result is reproduced by the present computer modeling approach—the Q_{ERB} of the impaired

FIG. 10. Q_{ERB} of the present cochlear model's displacement magnitude spectrum: Normal vs impaired. The magnitude spectrum is defined as the displacement gain at the RL with respect to the stapes (see Fig. 1). The impaired simulation was achieved by reducing the OHC MET sensitivity by 50% along the cochlea. Q_{ERB} of the HI model is not shown beyond 25 mm because due to loss of MET sensitivity, peaks in the magnitude spectrum cannot be found and the model does not preserve tonotopic mapping there in the most apical region.

model is higher than that of the normal model in three out of four cases [Fig. 8(B)], even though the tuning inside the normal model is consistently sharper than the impaired model along the entire length of the cochlea (Fig. 10).

C. Dependence of Q -value of DPOAE STCs upon width of NDR

The width of the NDR can possibly account for the observation that the Q_{ERB} of DPOAE STCs value could be higher for the impaired model than the normal model. The existence of the NDR causes a forward-traveling wave to receive amplification when it propagates along the cochlea, and the amplification process can be visualized in Fig. 2(A)—the NDR for the f_2 -tone is distributed from a “shoulder” point (the transition point near the 0.8-cm place at which the magnitude is about to increase rapidly) and into the cochlea until the traveling-wave magnitude reaches the peak (near the 1.1-cm place). The present model predicts that, with a loss in the OHC MET sensitivity [Fig. 2(C)], the NDR for the f_2 -tone becomes narrower; hence the wave receives insufficient gain and the TTD drops down accordingly. Now, by inspecting Fig. 7 again, it becomes noteworthy that the tip region in the DPOAE STCs of the normal model extends much beyond 4 kHz and into the high frequency region, a result of high frequency suppressors shutting down activities that happen in the f_2 -NDR. This high-frequency extension is not as prominent in the STCs of the impaired model because the f_2 -NDR becomes narrower. Therefore, even though the intra-cochlear displacement is more narrowly tuned for the normal model (Fig. 10), the existence of a spatially-distributed NDR forces the DPOAE STCs to widen, thus the overall width of the STCs ends up being comparable to those of the impaired model which has a narrower NDR for the f_2 tone.

VI. CONCLUSIONS

In the present computer simulation study, we calculated DPOAE SGFs and derived DPOAE STCs thereupon. The

SGFs resemble those obtained in human ears in terms of the frequency-dependence of the slope; the SGFs increase more rapidly against the suppressor level for low-frequency suppressors than for high-frequency suppressors. The STCs derived from SGFs, as expected, do not vary as smoothly across frequencies and levels as ICTCs do.

The present study did not establish simple ways to relate the Q_{ERB} of STCs to the Q_{ERB} of ICTCs. In fact, it reproduced results from recent experiments (Gruhlke *et al.*, 2012) which showed that the Q_{ERB} of DPOAE STCs may not correlate well with the amount of hearing loss. Some features in measured DPOAE STCs, such as the detailed shape of the curves and their absolute levels, are not reproduced in the present model precisely. For instance, the Q -values and TTDs tend to be lower for the computer model than measured in NH human subjects. Nevertheless, the present study has produced probably the most comprehensive DP suppression data ever obtained in simulation, and it is remarkable that the data can be analyzed in the same way as human data are analyzed, and certain qualities of human STCs, from normal or impaired subjects, are reproduced in the model. Future work is warranted to revise the model by reconsidering the cochlear hydromechanics (e.g., one- vs two- or three-dimensional) or the OHC electrophysiology and produce DP suppression that matches more closely with human data.

ACKNOWLEDGMENTS

This work is supported by the National Science Council of Taiwan (Grant Nos. NSC-100-2220-E-007-010 and 101-2221-E-007-029) and by NIH-NIDCD Grant No. R01-DC8318. The authors thank the three anonymous reviewers for very valuable critiques on a previous version of this manuscript.

¹Here we ignore middle-ear latency.

²All the physical parameters here are assumed to vary along the x direction; hereupon the spatial dependence will occasionally not be shown in favor of brevity.

³Gorga *et al.* (2011b) used fixed sensation level (SL) instead of SPL. Here SPL is used because SL is not defined for the computer model.

⁴This fixed relation between L_1 and L_2 is similar to what was used by Kummer *et al.* (1998) for measuring DPOAE in human ears. For every combination of (L_2, f_2) , Gorga *et al.* (2011b) determined L_1 empirically and *individually* to ensure that maximal DPOAE is achieved before the suppressor was introduced. Here the formula for choosing L_1 is not optimized to produce maximal DPOAEs.

⁵The selection of f_{sup} as an integer multiple $f_0 = f_2/240$ simplifies signal processing for it becomes possible to set the window length at $1/f_0$, an exact common multiple of the periods of the primary tones, the suppressor, and *all the distortion products*. Thus, spectral splattering of the primary tones and the suppressor due to truncation in time does not affect the estimates of DP levels.

⁶Even though we cannot equate a RL iso-displacement level with any given iso-suppression level under the setup of DPOAE suppression experiments, the comparison at the lowest level tested is still meaningful. It shows how sharp (Q_{ERB}) and tall (TTD) the respective type of tuning curves can ever get.

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