A mechanism for simultaneous suppression of tone burst-evoked otoacoustic emissions.

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Abstract

Tone burst-evoked otoacoustic emission (TBOAE) components in response to a 1-kHz tone burst are suppressed by the simultaneous presence of tone bursts at higher frequencies. To date, the underlying cause of this "simultaneous suppression" of TBOAEs is unclear. This paper describes a potential mechanism based on local nonlinear interactions between basilar membrane (BM) travelling waves, and tests the extent to which it is able to account for this specific suppression phenomenon. A simple mathematical model based on local nonlinear interactions was developed, and its predictions for a range of tone burst pairs were compared to corresponding TBOAE suppression data recorded from fourteen normally-hearing human ears at a level of 60 dB p.e. SPL. Model predictions and mean TBOAE suppression data showed close agreement for all pairs of tone bursts. These results suggest that simultaneous suppression of TBOAEs can be explained solely in terms of the local nonlinear interaction-based mechanism. However, the involvement of other mechanisms, involving components generated at places basal to their characteristic place along the BM, cannot be excluded.

Keywords: Tone burst-evoked otoacoustic emissions; suppression; tone bursts; model

Abbreviations: Basilar membrane, BM; Click-evoked otoacoustic emission, CEOAE; Input-output, I-O; Local nonlinear interaction, LNI; Root mean square, rms; Spontaneous otoacoustic emission, SOAE; Stimulus frequency otoacoustic emission, SFOAE; Synchronised spontaneous otoacoustic emission, SSOAE; Tone burst-evoked otoacoustic emission, TBOAE; Transient-evoked otoacoustic emission, TEOAE.

1. Introduction

Otoacoustic emissions (OAEs) are evident as sound emanating from the healthy cochlea, which may be recorded using a sensitive microphone placed in the ear canal. A commonly studied OAE is elicited in response to a brief transient sound, such as a click or a tone burst. This is referred to as the transient-evoked otoacoustic emission (TEOAE), or more specifically click evoked-otoacoustic emission (CEOAE) and tone burst-evoked otoacoustic emission (TBOAE). Because their presence is reliant on the normal functioning of the physiological processes that enhance hearing sensitivity and selectivity at low sound levels (i.e. the cochlear amplifier), CEOAEs (and to a lesser extent TBOAEs) are used widely in the clinical setting as a non-invasive assessment of cochlear function (e.g. Hall, 2000; Robinette and Glattke, 2002).

TEOAEs (i.e. CEOAEs and TBOAEs) exhibit a number of distinctive nonlinear behaviours. In the amplitude domain, TEOAEs exhibit compressive growth with increasing stimulus level, as evidenced by their input-output (I-O) function. Estimates of TBOAE I-O nonlinearity, based on the gradient of a regression line fitted to the I-O function plotted on log-log scales, indicate typical values in the approximate range of 0.5 to 0.7 dB/dB (Rutten, 1980; Norton and Neely, 1987; Epstein and Florentine, 2005). This static compressive I-O nonlinearity is a direct consequence of the limited range of stimulus levels for which the cochlear amplifier effectively operates (e.g. Nuttall and Dolan, 1996; Rhode and Recio, 2000).

In the frequency domain, previous investigators have demonstrated that the amplitude of TBOAE components at a particular frequency is reduced, or suppressed, by the simultaneous

presence of additional tone bursts at higher frequencies. Specifically, Xu et al (1994) reported that, at moderate and high stimulus levels, the simultaneous presence of a pair of tone bursts at 2 and 3 kHz suppressed TBOAE components in response to a 1 kHz tone burst (relative to the off-line sum of the responses to the three tone bursts presented individually). In a similar study, Yoshikawa et al (2000) described suppression of 1 kHz components caused by the simultaneous presence of an additional tone burst at 1.5, 2 or 3 kHz. Suppression was greatest for pairs of tone bursts that were closely spaced in frequency, i.e. 1 and 1.5 kHz, though at the highest level of stimulation significant suppression was observed for pairs of tone bursts that were relatively well separated, i.e. 1 and 2 kHz. Killan and Kapadia (2006) characterised the effect of tone burst level on suppression magnitude and demonstrated a near- monotonic increase with increases in stimulus level.

The mechanism responsible for this "simultaneous suppression" of TBOAEs remains unclear. It has been argued that in order for the higher frequency tone burst to cause suppression of response components at lower frequencies, those components must have originated from the (remote) basal region of the basilar membrane (BM) excited by the higher frequency tone burst (Xu et al, 1994; Yates and Withnell, 1999). Consistent with this argument, a small number of reports have provided evidence for a basal origin of OAE components in response to broadband clicks (Avan et al, 1997; Carvalho et al, 2003, Murnane and Kelly, 2003), via intermodulation distortion (e.g. Withnell and Yates, 1998), as well as in response to single pure tones (Siegel et al, 2005). However, given the short latency of these basal-source components, i.e. less than approximately 2 ms post-stimulus onset (e.g. Siegel et al, 2005; Notaro et al, 2007; Withnell et al, 2008), they will be removed from the response waveform as a consequence of discarding early waveform segments dominated by unwanted stimulus

ringing. It is therefore unlikely that basal-source TEOAE components play a significant role in simultaneous suppression of TBOAEs.

It is more likely that suppression is governed by a mechanism that is compatible with the dominant view of TEOAE generation. This states that, even at high stimulus levels, the main source of TEOAEs is linear coherent reflection of stimulus-evoked travelling waves off place-fixed perturbations in cochlear mechanics (e.g. Shera, 2004; Kalluri and Shera, 2007; Sisto and Moleti, 2008; Withnell et al, 2008). This view is compatible with the concept of "generator channels" tonotopically distributed along the BM, within which TEOAE components at a particular frequency are generated locally (i.e. at their characteristic place) in response to stimulus components at the same frequency (e.g. Kemp and Chum, 1980; Tavartkiladze et al, 1994; Prieve et al, 1996; Zettner and Folsom, 2003; Kalluri and Shera, 2007). These generator channels are thought to be compressively nonlinear, reflecting the I-O nonlinearity exhibited by TEOAEs[1]. A basis for understanding simultaneous suppression of TBOAEs which is consistent with the dominant view of TEOAE generation has been suggested by Killan and Kapadia (2006). They argued that simultaneous suppression can be understood simply in terms of *local* nonlinear interactions between the BM travelling waves caused by the tone bursts. These local interactions are governed by the compressive I-O nonlinearity of TEOAE generator channels and the tuning of the cochlear filter, as described below.

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[1] The compressive nonlinearity of generator channels is not inconsistent with the view that TEOAE arise via linear coherent reflection. It simply reflects the influence of the cochlear amplifier on travelling waves. The physical mechanism responsible for reflecting the travelling waves is essentially linear (Zweig and Shera, 1995; Shera, 2004).

Stimulation by a tone burst with frequency *f* and amplitude *A* would excite a specific BM location, or generator channel, tuned to *f*. If a pair of identical tone bursts (each with frequency *f* and amplitude *A*) was presented simultaneously, the generator channel would experience twice the excitation relative to that experienced for the individual tone burst. However, because the generator channel is compressively nonlinear, i.e. its I-O function has a slope less than 1 dB/dB, TBOAE components output from the channel in response to simultaneous presentation will have smaller amplitude than corresponding components in the off-line sum of the individual responses. This principle can be extended to pairs of tone bursts that are closely spaced in frequency (e.g. a tone burst with frequency *f* and another with frequency slightly higher than *f*). These tone bursts will cause travelling waves that overlap[2] when presented simultaneously, so that both tone bursts will cause excitation of the generator channel tuned to *f*. The generator channel will therefore experience increased excitation when tone bursts are presented simultaneously compared to individual presentation of the tone bursts As with the example of two identical tone bursts, the compressive nonlinearity of the generator channel will result in the simultaneous response being reduced (i.e. suppressed) relative to the sum of the individual responses. Because the amount of overlap between travelling waves will be less than that for identical tone bursts, suppression caused by the slightly higher frequency tone burst will also be less than that caused by a pair of identical tone bursts. In contrast, tone bursts that are well separated in frequency (e.g. a tone burst with frequency *f* and another with frequency considerably higher than *f*) will produce travelling waves that overlap only a small amount, if at all. Consequently, there will

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[2] It has been questioned whether such tone bursts cause travelling waves that overlap sufficiently for the nonlinear interactions to occur (Yates and Withnell, 1999). However, direct observations of BM motion at the apical turn of the cochlea suggest tuning of the BM response is sufficiently broad for travelling waves in response to tone bursts to overlap (Cooper and Rhode, 1996).

be little difference in the excitation of the generator channel tuned to f, regardless of whether the tone bursts were presented individually or simultaneously. Thus, minimal or no suppression would be expected.

Whilst in theory this local nonlinear interaction (LNI) -based mechanism is able to account for simultaneous suppression of TBOAEs, no formal test has been reported, and the extent to which it can account for suppression remains unclear. An understanding of the mechanism underpinning the phenomenon has important implications for the interpretation of TBOAEs in the clinical setting. If simultaneous suppression is caused by nonlinear interactions occurring locally at the characteristic place of the suppressed components, TBOAEs can be considered to be a frequency-specific assessment of cochlear function. In contrast, frequency-specificity cannot be assumed if suppression reflects the significant involvement of components generated at remote basal BM locations. The aim of the present study, therefore, was to test the extent to which the LNI-based mechanism can account for simultaneous suppression of TBOAEs. A simple mathematical model of the mechanism was developed and predictions of suppression were obtained for pairs of tone bursts presented either simultaneously or individually following the experimental paradigm employed by Yoshikawa et al (2000). These were then compared to corresponding TBOAE suppression measurements obtained from normal human ears. The degree of correspondence between model predictions and TBOAE suppression was used to demonstrate the extent to which the LNI-based mechanism can account for suppression. It was reasoned that any substantial differences would argue against such a mechanism.

2. Materials and methods

2.1 Description of model

A simple mathematical model of the LNI-based mechanism was developed. The modelling approach taken was similar to that used to test mechanisms responsible for suppression of CEOAE by additional clicks closely spaced in time (Kapadia and Lutman, 2000a) and rate suppression (Kapadia and Lutman, 2001).

A single TEOAE generator channel was represented by a linear filter in series with a compressive I-O function. The filter element of the model was a static gammachirp (bandpass) filter with impulse response:

$$g(t) = at^{n-1} \exp[-2\pi b \text{ERB}(f)t] \cdot \cos[2\pi ft + c\ln(t) + \phi] \qquad \text{for } t > 0, \qquad (\text{Eq.1})$$

where a, n, b and c are parameters and $\text{ERB}(f)$ is the equivalent rectangular bandwidth of the filter at frequency f (see Irino and Patterson, 1997) and ϕ is the initial phase[3]. The output of the filter in response to a tone burst was then taken as an estimate of excitation at a BM location corresponding to the peak frequency of the filter. Tone bursts with centre frequencies close to the peak frequency would cause greater excitation than tone bursts at more remote frequencies, and two such tone bursts presented simultaneously would cause greater excitation than either presented individually. For Eq. 1, when $a = 1$, $n = 4$, $b = 1.81$, c

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[3] f in this case is the asymptotic frequency. The peak frequency of the filter, $f_p = f + cb\text{ERB}(f)/n$.

= –2.96 and ERB(f) = 24.7 + 0.108f (in Hz) the resultant filter has been shown to be a good approximation of psychophysical and physiological estimates of the cochlear filter (e.g. Irino and Patterson, 1997, 2001). To model suppression of response components in the region of 1 kHz, a gammachirp filter with a peak frequency of 1.2 kHz was used. This frequency was chosen as the cochlear amplifier element responsible for response components at 1 kHz is shifted a small distance basalward, corresponding to approximately 0.2 kHz (e.g. Neely and Kim, 1986; Kolston, 2000; Irino and Patterson, 2006). The impulse (panel A) and magnitude (panel B) responses of the gammachirp filter used are shown in Figure 1.
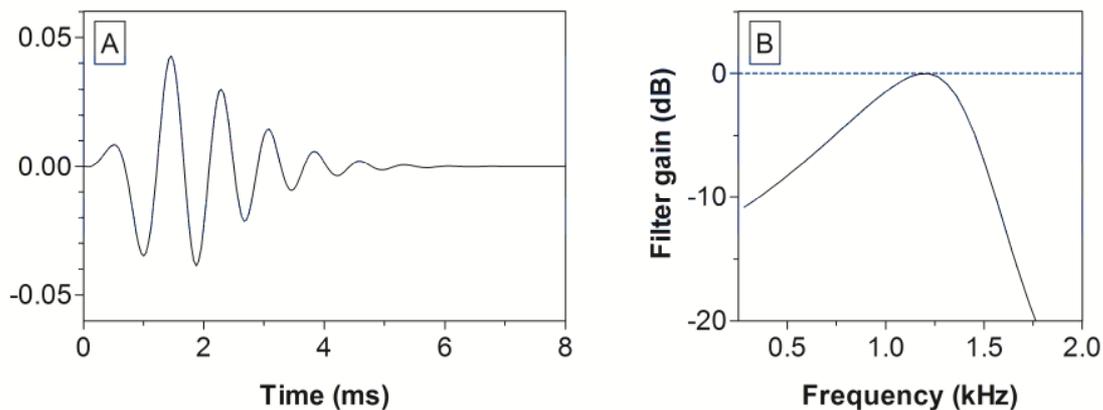
Figure 1 A. Impulse and B. Magnitude response for the gammachirp filter described by Eq. 1, with variables as described in the text and peak frequency at 1.2 kHz. The ordinate scale represents amplitude in arbitrary units.

The static compressive I-O component of the generator channel was modelled using a simple power function of the form

$$y = Ax^m \qquad (0 < m \le 1). \tag{Eq. 2}$$

When m = 1, Eq. 2 describes a linear relationship between input and output. As m approaches zero, Eq. 2 becomes progressively more nonlinear. Panel A of Figure 2 shows the output of Eq. 2 when A = 1 and m = 0.5 for a single 1 kHz tone burst of amplitude 10 arbitrary units[4]. The output is clearly compressed relative to the input tone burst. Further, because $\log(y) = \log(A) + m\log(x)$, the function obtained by applying scaled versions of the 1 kHz tone burst to Eq. 2 describes a straight line with a gradient of m dB/dB (when plotted on axes scaled in dB). Values of m based on estimates of typical TBOAE I-O nonlinearity reported in the literature were used (Rutten, 1980; Norton and Neely, 1987; Epstein and Florentine, 2005). Panel B of Figure 2 shows the I-O functions obtained from Eq. 2 for values of m (i.e. 0.5, 0.6 and 0.7).
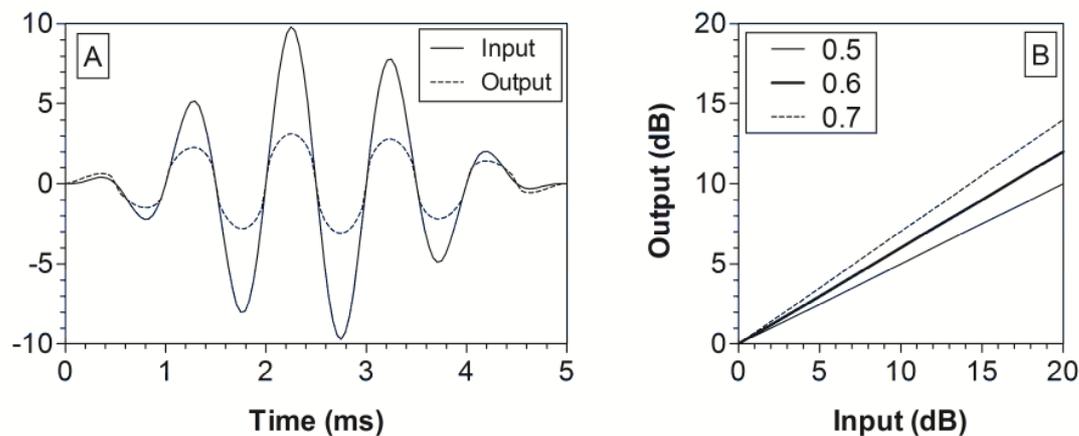
Figure 2 A. The resultant output (dashed line) given by Eq. 2, when A = 1 and m = 0.5, in response to a 1 kHz tone burst of amplitude 10 arbitrary units (solid line). B. The I-O functions obtained from Eq. 2 when A = 1 and m = 0.5, 0.6 and 0.7.

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[4] For negative values of x, the absolute value was raised to the exponent m, before multiplying by -1.

The tone bursts applied to the model were cosine-windowed sinusoids (rise-fall = 2.5 ms; plateau = 0 ms) with an amplitude of 10 arbitrary units. Following the paradigm used by Yoshikawa et al (2000), tone bursts were presented in pairs (TB_1 and TB_2) either simultaneously or sequentially; TB_1 had a fixed centre frequency of 1 kHz, whereas TB_2 had a centre frequency of 0.5, 1.0, 1.5, 2.0 or 3.0 kHz. Simultaneous presentation was achieved by digital summation of TB_1 and TB_2 to form five "complex" stimuli. Responses (model outputs) to successive TB_1 and TB_2 were summed to form five "composite" responses. Suppression was then calculated as the ratio (expressed in dB) of the root mean square (rms) amplitude of the filtered composite output to that of the corresponding filtered complex output. In order to compare suppression across tone burst pairs, suppression curves were generated by plotting the suppression value obtained for each pair of TB_1 and TB_2 against the difference between the centre frequencies of TB_1 and TB_2 (TB_2–TB_1), referred to as Δf.

It is important to note that the model does not intend to represent faithfully the individual physiological processes that occur in the cochlea following stimulation by simultaneous tone bursts. Rather, the model aims to provide a simple indication of the LNI-based mechanism for a single generator channel. The restriction of the model to a single channel is based on the assumption that suppression results from local nonlinear interactions within TEOAE generator channels, and that TEOAEs recorded in the ear canal are the simple sum of the outputs from a number of generator channels. Limiting the model to a single generator channel located at 1.2 kHz excludes the involvement of basal-source response components.

2.2 Simultaneous suppression of TBOAEs measurements

Subjects were normally hearing adults with hearing threshold levels of 15 dB HL or better at octave frequencies between 0.25 and 8 kHz and normal middle ear status as confirmed by tympanometry. It has been suggested that spontaneous otoacoustic emissions (SOAEs) can influence the nonlinear behaviour of TEOAEs, such that ears with SOAE behave differently from ears without SOAE (Probst et al, 1986; Kulawiec and Orlando, 1995). Ears that exhibited synchronised SOAEs (SSOAEs) were therefore excluded from the study. SSOAEs were measured using the Otodynamics ILO 292 system, with an SSOAE being deemed to be present if it had amplitude greater than –25 dB SPL and 3 dB above the noise floor. TBOAE measurements were obtained from one ear (9 right, 5 left) of each of 14 subjects (8 female, 6 male) aged between 20 and 31 years (median = 22 years).

All recordings were made in a sound-attenuated room, with the subject comfortably seated. The measurement probe was sealed into the ear canal with a soft plastic tip and was taped into position. Subjects were instructed to remain quiet and still for the duration of the testing. The study was completed with the approval of the School of Healthcare Research Ethics Committee. All TBOAE measurements were made using a custom-built system. Purpose-written software controlled the synchronised input and output of a RME HDSP 9632 personal computer soundcard (Haimhausen, Germany). Stimuli were presented via the earphone of an Otodynamics (London, UK) probe via a purpose-built amplifier. The signal measured by the probe microphone was input to the soundcard via a further amplifier and high-pass filter (cut-off at 500 Hz with roll-off slope > 12 dB/octave). This input signal was sampled at a rate of 24 kHz and time-averaged within two separate buffers, creating two replicate recordings, each resulting from 500 averages.

Stimuli were the same as those applied to the model. TB_1 had a fixed centre frequency of 1 kHz and TB_2 had centre frequencies of 0.5, 1.0, 1.5, 2.0 or 3.0 kHz. TB_1 and TB_2 were presented in pairs sequentially and simultaneously (as a complex stimulus resulting from the digital addition of TB_1 and TB_2). In order to minimise potential order effects, for each pair of TB_1 and TB_2, sequential and simultaneous presentations were presented in a random order. All stimuli were presented at 60 dB p.e. SPL (as calibrated within a passive 2 cm^3 cavity) and at a presentation rate of 50/s. At this presentation rate, mean subjective threshold for the 1 kHz tone burst was approximately 15 dB p.e. SPL. As the cochlear amplifier begins to overload at levels above 25 dB sensation level (e.g. Nuttall and Dolan, 1996; Rhode and Recio, 2000), presentation of tone bursts at 60 dB p.e. SPL would be expected to cause the response characteristic of the cochlea to be nonlinear.

Time-averaged recordings were stored on disk and analysed off-line. A mean response waveform was calculated for all TB_1, TB_2 and complex stimuli. Pairs of mean TB_1 and TB_2 response waveforms were then added together to form "composite" waveforms. The first 8 ms segment of each composite and complex waveform was removed in order to eliminate stimulus ringing. Removal of such a substantial portion of the response waveform is typical when using tone bursts (e.g. Wilson, 1980; Prieve et al, 1996; Killan and Kapadia, 2006). Finally, to determine suppression in the region of 1 kHz and to enable comparison with model predictions, all waveforms were filtered using a bandpass filter with low and high cut-off frequencies of 0.7 and 1.3 kHz. Suppression was then calculated as the ratio of the rms amplitude of corresponding filtered composite and complex waveforms and was plotted against Δf, to form suppression curves.

3. Results

3.1 Model predictions

Panel A of Figure 3 shows the composite and complex waveforms predicted by the model when $A = 1$ and $m = 0.5$, for a pair of identical tone bursts with amplitude of 10 arbitrary units and frequency of 1 kHz, i.e. $\Delta f = 0$.   The waveforms are delayed in time as a consequence of being filtered by the bandpass filter. The complex waveform (dashed line) is clearly reduced (suppressed) relative to the composite waveform (solid line).   Panel B of Figure 3 shows the resultant suppression curves obtained by plotting suppression magnitude (calculated as the ratio of the rms amplitude of composite and complex waveforms) against Δf, for $m = 0.5, 0.6$ and 0.7. The main behaviours of suppression predicted by the model can be summarised as i) suppression was greatest at $\Delta f = 0$; ii) as Δf increases from zero suppression magnitude reduces, with only minimal suppression predicted at $\Delta f = 1.0$ and 2.0; iii) a small amount of suppression is evident at $\Delta f = -0.5$, though less than at $\Delta f = 0.5$ and iv) at all Δf suppression magnitude can be seen to increase progressively as m decreases, i.e. as the model becomes more nonlinear.
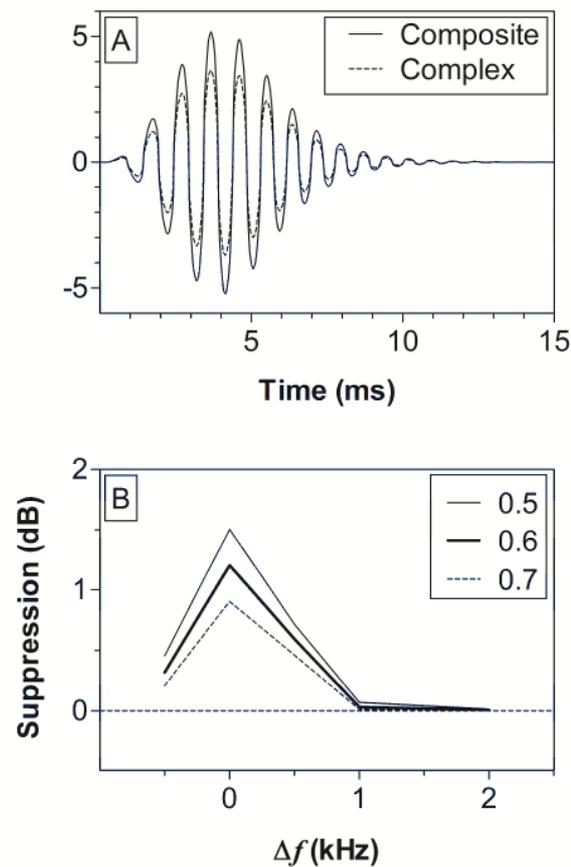
Figure 3 A. Composite (solid line) and complex (dashed line) model outputs when $A = 1$ and $m = 0.5$ for $\Delta f = 0$. The ordinate scale represents amplitude in arbitrary units. A reduction (suppression) of the complex waveform relative to the composite is evident. Suppression curves predicted by the model when $A = 1$ and $m = 0.4, 0.5$ and 0.6 are shown in panel B.

3.2 Simultaneous suppression of TBOAE

Panels A-E in Figure 4 show the composite and complex TBOAE waveforms obtained from a representative subject. Again, as a consequence of filtering, all waveforms show a constant delay. A close correspondence clearly exists between composite and complex waveforms, however suppression is apparent, especially at $\Delta f = 0$ and 0.5. This is confirmed by the suppression curve shown in panel F of Figure 4.
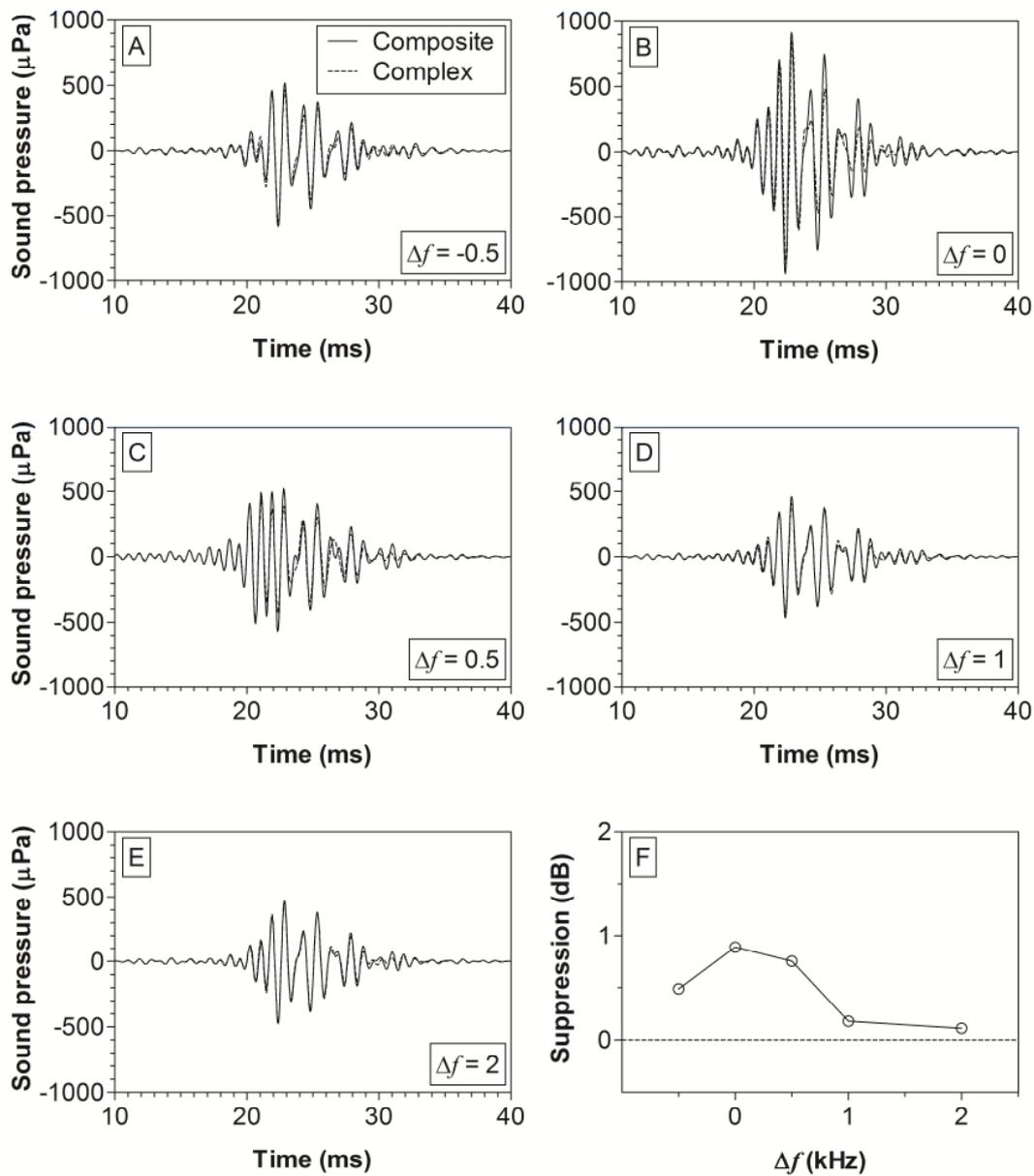
Figure 4 A-E. Filtered composite (solid line) and complex (dashed line) TBOAE waveforms from a representative subject. A close correspondence between composite and complex waveforms is evident, however suppression of the complex waveform is evident when $\Delta f = 0$ and 0.5. The corresponding suppression curve is shown in panel F.

Figure 5 shows the mean suppression curve measured in real ears ($n = 14$). Consistent behaviours with those predicted by the model are evident. That is, i) mean suppression was

greatest at $\Delta f = 0$; ii) suppression magnitude decreased with increasing Δf and iii) at $\Delta f = -0.5$ suppression magnitude was less than that obtained at $\Delta f = 0.5$. Statistical testing[5] revealed suppression was significant at $\Delta f = 0$ and 0.5 ($t = 7.63$, $p < 0.001$ and $t = 3.65$, $p < 0.001$ respectively). At $\Delta f = -0.5$, 1.0 and 2.0 suppression was not significant ($t = 0.90$, $p = 0.185$; $t = 1.83$, $p = 0.034$ and $t = 0.18$, $p = 0.429$ respectively).
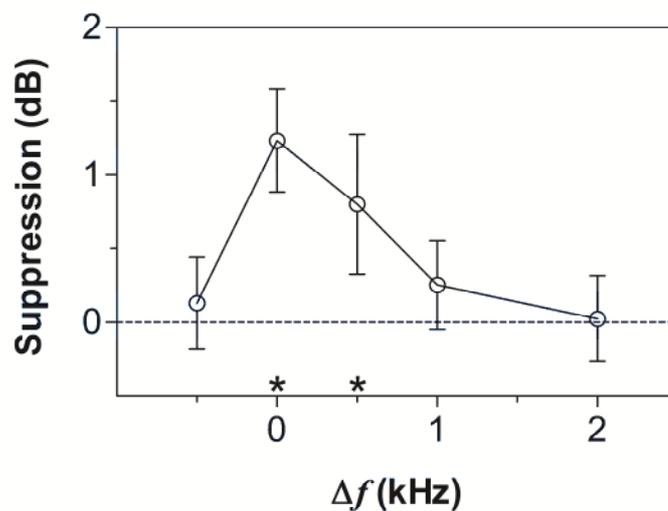
Figure 5 Mean TBOAE suppression curve. Error bars represent 95% confidence intervals. Asterisks indicate those Δf where mean suppression was significantly different from zero.

The similarity between model predictions and mean TBOAE suppression is clearly evident in Figure 6. The mean TBOAE data (open circles) from Figure 5 are plotted with model predictions when $A = 1$ and $m = 0.5$ and 0.7 (upper and lower dashed lines respectively), i.e. the full range of suppression predicted by the model for the values of m used. Visual inspection shows a close correspondence, both in terms of the dependence of suppression on Δf and suppression magnitude. Where differences between the model predictions and mean

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[5] A Bonferroni corrected significance level of $p < 0.01$ was used to allow for multiple testing.

TBOAE data are apparent they are less than the variation evident within the TBOAE data. A rms difference of 0.16 dB was obtained between mean TBOAE suppression and model predictions when $m = 0.6$.
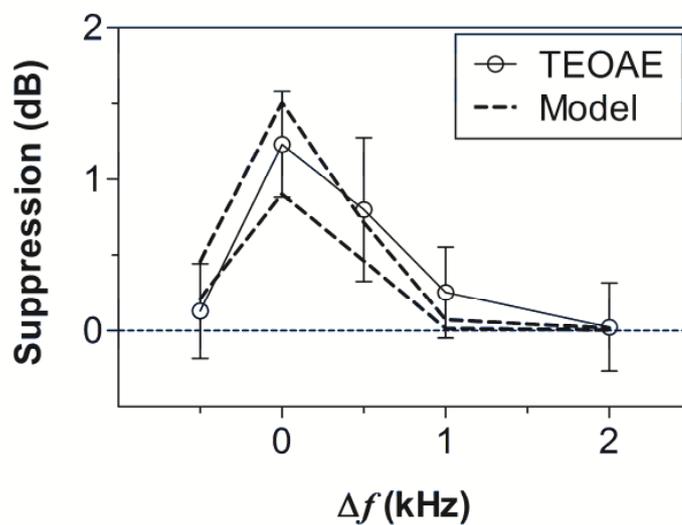
Figure 6 Mean TBOAE suppression curve (open circles) from Figure 5 and the suppression curves predicted by the model when $A = 1$ and $m = 0.5$ and 0.7 (dashed lines).

4. Discussion

Previous investigators have demonstrated that the amplitude of TBOAE components at a particular frequency are suppressed by the simultaneous presence of additional tone bursts at higher frequencies (Xu et al, 1994; Yoshikawa et al, 2000; Killan and Kapadia, 2006). The aim of the present study was to test the extent to which a mechanism implied by Killan and Kapadia (2006), termed here the LNI-based mechanism, could account for this simultaneous suppression of TBOAEs. In order to do this, a simple mathematical model of the LNI-based mechanism was developed and its predictions were compared to simultaneous suppression data obtained from normal human ears.

A close correspondence between model predictions and mean TBOAE data was observed, both in terms of the magnitude of suppression and its dependence on the frequency separation between tone bursts. Further, because the model predicts that suppression increases with increasing I-O nonlinearity (as shown in Figure 3), and TEOAE I-O nonlinearity is compressive (so that nonlinearity increases with stimulus level), the model indirectly predicts that TBOAE suppression magnitude will increase with increasing tone burst level. Such level-dependence is in agreement with the data reported by Killan and Kapadia (2006). These findings therefore provide no reasons to reject the LNI-based mechanism as the mechanism responsible for simultaneous suppression of TBOAEs. Thus, it is tentatively suggested here that this suppression phenomenon is chiefly governed by the LNI-based mechanism (at least for the range of stimulus frequencies used in this study).

As well as being compatible with the prevailing view of TEOAE generation (e.g. Shera, 2004; Kalluri and Shera, 2007; Sisto and Moleti, 2008), the findings of this study are in keeping with those from a number of other investigations. First, our results suggest that simultaneous suppression can be explained by TEOAE I-O nonlinearity (as manifest in the compressive nonlinearity of TEOAE generator channels). This is consistent with reports of other TEOAE suppression phenomena that have shown a significant underpinning role for TEOAE I-O nonlinearity, e.g. suppression caused by a pure tone (e.g. Kemp and Chum, 1980), additional clicks closely spaced in time (e.g. Kemp and Chum, 1980; Kapadia and Lutman, 2000a, b; Harte et al, 2005) and high stimulus presentation rates (Kapadia and Lutman, 2001; Lineton et al, 2006).

Second, in addition to previous reports, the present study obtained results for a 1 kHz tone burst paired with a lower frequency tone burst, i.e. 0.5 kHz. Suppression in this case was shown to be minimal, with smaller magnitude than that obtained for the pairing of 1 and 1.5 kHz tone bursts. This asymmetrical suppressive effect is in keeping with suppression behaviour exhibited by stimulus frequency otoacoustic emissions (SFOAEs) (Brass and Kemp, 1993; Keefe et al, 2008). A similarity between SFOAE and TBOAE suppression behaviour implies support for the LNI-based mechanism. Being responses to single pure tones, SFOAE generation (and therefore suppression) is widely assumed to be governed by linear coherent reflection, consistent with the existence of nonlinear generator channels tonotopically distributed along the BM (e.g. Talmadge et al, 2000; Goodman et al, 2003; Lineton and Lutman, 2003; Shera, 2004; Kalluri and Shera, 2007). If it is assumed that equivalence of suppression behaviour implies a shared suppression mechanism, then simultaneous suppression of TBOAEs must also be underpinned by such mechanics.

Third, the present findings are consistent with those reported by Jedrzejczak et al (2008). They re-analysed the data of Yoshikawa et al (2000) using an analysis technique that identified resonant frequencies, or modes, within TBOAE waveforms. It was shown that suppression occurred only when tone bursts overlapped in frequency and resonant modes were excited by both tone bursts.

Despite the above arguments in favour of the LNI-based mechanism, the results of the present study do not exclude other mechanisms wherein TBOAE components are generated at BM places basal to their characteristic place. However, because of their short latency (Siegel et al, 2005; Notaro et al, 2007; Withnell et al, 2008), basal-source response components would have been removed prior to analysis (along with unwanted stimulus components) making it unlikely that they could influence suppression. This will also have been the case for previous reports of simultaneous suppression of TBOAEs (Xu et al, 1994; Yoshikawa et al, 2000; Killan and Kapadia et al, 2006). Further, the results of the present study are difficult to reconcile with an involvement of such basal-source components. For example, Withnell and Yates (1998) demonstrated that when TEOAEs are dominated by basal-source components, the presence of a pure tone caused suppression of components at lower frequencies, whilst those at the pure tone frequency were not affected. Similarly, Yates and Withnell (1999) reported that TEOAE components at a particular frequency were unaffected by the simultaneous presence of stimulus components at the same frequency. Clearly, these behaviours are at odds with those observed in the present study, i.e. maximum suppression when tone bursts had the same frequency whereas well separated (or remote) tone bursts caused no suppression. However, in order to make more confident conclusions

about the role of basal-source components in the production of simultaneous suppression of TBOAEs, further investigation would be required. To this end, a detailed parametric study, utilising time-frequency analysis techniques that allow decomposition of TBOAEs based on their likely origin (Notaro et al, 2007; Goodman et al, 2009) is currently being undertaken.

Finally, the conclusion that simultaneous suppression of TBOAEs can be accounted for by local nonlinear interactions at the characteristic BM place of the suppressed components supports the use of TBOAEs as a frequency-specific assessment of cochlear function in the clinical setting.

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