# In search of basal distortion product generators

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The $2f_1$-$f_2$ distortion product otoacoustic emission (DPOAE) is thought to arise primarily from the complex interaction of components that come from two different cochlear locations. Such distortion has its origin in the nonlinear interaction on the basilar membrane of the excitation patterns resulting from the two stimulus tones, $f_1$ and $f_2$. Here we examine the spatial extent of initial generation of the $2f_1$-$f_2$ OAE by acoustically traumatizing the base of the cochlea and so eliminating the contribution of the basal region of the cochlea to the generation of $2f_1$-$f_2$. Explicitly, amplitude-modulated, or continuously varying in level, stimulus tones with $f_2/f_1=1.2$ and $f_2=8000-8940$ Hz were used to generate the $2f_1$-$f_2$ DPOAE in guinea pig before and after acoustically traumatizing the basal region of the cochlea (the origin of any basal-to-$f_2$ distortion product generators). It was found, based on correlation analysis, that there does not appear to be a basal-to-$f_2$ distortion product generation mechanism contributing significantly to the guinea pig $2f_1$-$f_2$ OAE up to $L_1=80$ dB sound pressure level (SPL).
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## I. INTRODUCTION

Since distortion product otoacoustic emissions (DPOAEs) were first reported in the literature (Kemp, 1979), an ever-increasing view of the complexity of their origin has developed over the years. Viewed as predominantly arising from the nonlinear interaction in the cochlea of a two stimulus tone input with the distortion propagating back to the stapes from the region of nonlinear interaction, i.e., the $f_2$ region (Brown and Kemp, 1984; Martin *et al.*, 1987), it was nevertheless recognized that, for the $2f_1$-$f_2$ DPOAE in particular, the emission measured in the ear canal could represent the sum of distortion generated in the region of $f_2$ and emission arising from the $2f_1$-$f_2$ region (Kim, 1980). This view of the $2f_1$-$f_2$ OAE being the vector sum of contributions from the $f_2$ region and $2f_1$-$f_2$ region was confirmed in the mid to late 1990s (Gaskill and Brown, 1996; Heitmann *et al.*, 1998; Mauermann *et al.*, 1999). Subsequent to the development of Kemp's idea (e.g., Kemp, 1986) of OAEs arising from either a place-fixed or a wave-fixed mechanism (Zweig and Shera, 1995; Talmadge *et al.*, 1998; Shera and Guinan, 1999), it was established that the $2f_1$-$f_2$ OAE not only arises from two discrete sources but that each of these sources has a different predominant mechanism of generation (Talmadge *et al.*, 1999; Kalluri and Shera, 2001). Thus the prevailing view for the origin of the $2f_1$-$f_2$ OAE (and, by extension, all DPOAEs with a frequency less than $f_2$) became one of the emissions being generated in two different cochlear locations, each location involving a different predominant mechanism of generation (see Fig. 1). This view for the origin of the $2f_1$-$f_2$ OAE seems to be applicable to all mammals, but differences in cochlear tuning and cochlear inhomogeneity may provide for the different emphases observed for the two components measured as a vector sum in the ear canal (e.g., Withnell *et al.*, 2003; Schneider *et al.*, 2003).

Additional complexity to the generation of DPOAEs was suggested by Fahey *et al.*, (2000) to provide for observed suppression/enhancement of DPOAEs by a third tone that had a frequency more than an octave above the stimulus tone frequencies. Two mechanisms were suggested to account for this effect: a catalyst mechanism and a harmonic mechanism. The catalyst mechanism involves a concatenation of nonlinear interaction of the three stimulus tone basilar membrane (BM) responses, their harmonics, and intermodulation distortion products. The harmonic mechanism involves the nonlinear interaction of the cochlear harmonic of one of the stimulus tones with the cochlear response to the other stimulus tone. The harmonic mechanism, it is suggested, provides a means for DPOAEs to be produced, in the absence of a third tone, additional to the view outlined in Fig. 1 (Fahey *et al.*, 2000; Martin *et al.*, 2003). This is illustrated schematically in Fig. 2. Because the harmonic mechanism involves nonlinear interaction with a harmonic of either $f_1$ or $f_2$, it is presumably a stimulus level dependent effect.

Evidence for a harmonic and/or catalyst mechanism based on DPOAE suppression tuning curves obtained with a third tone has been presented (e.g., Martin *et al.*, 1999). In rabbit, evidence for suppression/enhancement of the $2f_1$-$f_2$ DPOAE with a suppressor tone frequency associated with a cochlear location remote from that place in the cochlea tuned to the stimulus frequencies is observed concomitant with a cochlea that generates many orders of distortion products (e.g., Fahey *et al.*, 2006). It may be that the rabbit cochlea generates more distortion than other mammals, providing for the conjecture that the suppression/enhancement is an

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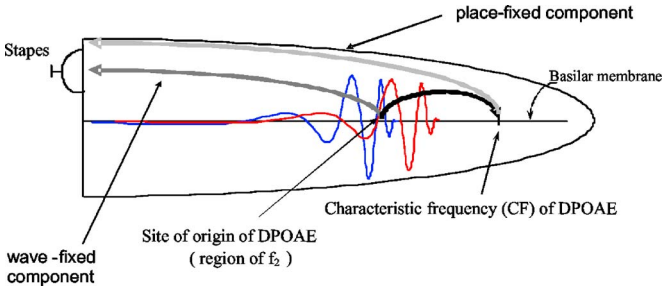


FIG. 1. (Color online) Schematic of origin of $2f_1$-$f_2$ OAE with the cochlea unfurled and scala media represented by the basilar membrane. The OAE measured in the ear canal is a vector sum of contributions arising from the $f_2$ and CF regions.

anomalous effect specific to rabbit but the effect has also been reported in guinea pig (Martin *et al.*, 1999) and humans (Martin *et al.*, 2003).

Fahey *et al.* (2000) suggested that the catalyst mechanism would be dominant in the high-frequency region of the cochlea while the harmonic mechanism would be dominant in the low-frequency region of the cochlea. This conclusion was based on the observation that the harmonic mechanism would not be expected to operate at intermediate and high frequencies where the high-frequency cutoff of the basilar membrane response is sharp. In guinea pig, the slope of the high-frequency cutoff of the basilar membrane "near-threshold" response (inferred from measurements of auditory nerve fiber tuning) increases from about 30 dB/octave below 1 kHz to greater than 100 dB/octave above 3 kHz (Evans, 1972). For fundamental frequencies of 3 kHz and higher, any second (or higher) harmonic produced is spatially confined on the basilar membrane to the region from the stapes to approximately its own characteristic place, i.e., no significant second harmonic is generated on the basilar membrane between the characteristic place of the second harmonic and the characteristic place of the fundamental. The second harmonic in such a case is being generated by nonlinear interaction well basal to the peak of the fundamental traveling wave and so is small, i.e., harmonics do not achieve sufficient amplitude at their own characteristic frequency place to generate sufficient nonlinear interaction to produce quadratic distortion that is a combination of the second harmonic of the fundamental with the BM response to the other stimulus tone. With increasing stimulus level, the basilar membrane response broadens with the peak of the response shifting basally (Johnstone *et al.*, 1986) producing a larger amplitude response to the fundamental tone basal to the characteristic place for the second harmonic. For the generation of $2f_1$-$f_2$, the second harmonic of $f_1$ generated with increasing stimulus level may achieve sufficient amplitude at its own characteristic place to interact nonlinearly with the BM response to the $f_2$ tone to produce $2f_1$-$f_2$ as a quadratic distortion tone.[1]

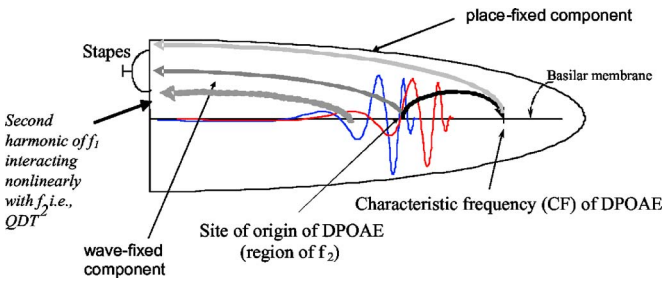


FIG. 2. (Color online) Schematic of origin of $2f_1$-$f_2$ OAE, including the speculated harmonic distortion mechanism, with the cochlea unfurled and scala media represented by the basilar membrane. The OAE measured in the ear canal is a vector sum of contributions arising from the $2f_1$, $f_2$, and CF regions.

The catalyst mechanism requires the simultaneous presentation of three (or more) stimulus tones with distortion products arising out of a concatenation of nonlinear interaction, "this heterodyning down…done in such a way that the value of the $f_3$ frequency is not contained in any final products" (Fahey *et al.*, 2000, p. 1796). For example, the nonlinear interaction in the cochlea of the BM responses to $f_3$ and $f_1$ may generate $f_3$-$f_1$ and the nonlinear interaction of $f_3$ and $f_2$ may generate $f_3$-$f_2$, the two intermodulation distortion products interacting nonlinearly in the region of $f_3$-$f_1$ in the cochlea to produce $f_2$-$f_1$. Fahey *et al.* (2000) give other examples that require the generation of harmonics of the stimulus tones in the cochlea, and so, third order distortion products, for instance (e.g., $2f_1$-$f_2$), are also dependent on a harmonic mechanism for their generation. Pertinent to the operation of a catalyst mechanism is the study of Kim *et al.* (1997) where distortion product otoacoustic emissions were generated by multiple stimulus tone pairs in humans. To test the hypothesis that regions of the cochlea separated by an octave do not significantly interact nonlinearly, Kim *et al.* (1997) examined $2f_1$-$f_2$ OAE generated by each of three stimulus tone pairs presented separately and presented simultaneously with stimulus levels of 65 and 50 dB SPL for each of the stimulus pairs, adjacent $f_2$'s an octave apart. It was found that the effect of using three stimulus tone pairs to generate $2f_1$-$f_2$ OAE produced only a very small difference in $2f_1$-$f_2$ OAE level (less than 1.3 dB) with a tendency for the OAE to be lower in the three pair stimulus condition than for single stimulus pairs. The effect, while small, may be evidence of suppression effects and/or a catalyst mechanism.

More generally than the basal distortion product generation mechanisms proposed by Fahey *et al.* (2000), the spatial extent of generation of an OAE has not been widely examined, particularly at high stimulus levels. Withnell and Yates (1998) found the growth of the $2f_1$-$f_2$ OAE[2] at low to moderate stimulus levels to be analogous to the basilar membrane input-output function and so consistent with a locus of origin of $f_2$ for $f_2/f_1=1.6$. The spatial extent of generation of the $2f_1$-$f_2$ OAE is clearly dependent though on the stimulus frequency ratio ($f_2/f_1$). A number of studies have examined the locus of $2f_1$-$f_2$ OAE generation using iso-suppression tuning curves (e.g., Brown and Kemp, 1984; Kummer *et al.*, 1995; Abdala *et al.*, 1996) and shown it to be near $f_2$. Martin *et al.*, 1998 examined $2f_1$-$f_2$ OAE suppression tuning curves for $f_2/f_1=1.2$, showing the locus of generation in humans to be near, but apical, to $f_2$ for the $2f_1$-$f_2$ OAE. At high stimulus levels, broadened BM excitation patterns with basal-ward shifts in the maximum BM responses provide a larger region of nonlinear interaction between the BM excitation patterns arising from the two pure tone stimuli.

The spatial extent of generation of the $2f_1$-$f_2$ OAE can be examined by acoustically traumatizing the base of the cochlea and so eliminating the contribution of the basal region of the cochlea to the generation of $2f_1$-$f_2$. This experimental paradigm includes examination of a harmonic mechanism, this mechanism not requiring the presence of a third tone to be investigated in terms of its contribution to the generation of DPOAEs, acoustically traumatizing the base of the cochlea eliminating the contribution of a harmonic mechanism to the generation of $2f_1$-$f_2$. Without the addition of a third tone, it does not examine a catalyst mechanism. In this study, we examine evidence for a basal-to-$f_2$ distortion product generator in the guinea pig by comparing $2f_1$-$f_2$ OAE recorded before and after acoustically traumatizing the basal turn using a 12 kHz ~ 100–105 dB SPL tone. To explore the dependence of stimulus level on the spatial extent of generation of the $2f_1$-$f_2$ OAE, amplitude-modulated stimuli were used to generate an amplitude-modulated $2f_1$-$f_2$ OAE, i.e., an OAE that varies in amplitude over time in a stimulus-level dependent manner. Stimulus frequencies in the range $f_2$=8–8.94 kHz were used ($f_2/f_1$=1.2), a frequency range in the same region of the cochlea as that examined by Martin *et al.* (1999), who found $2f_1$-$f_2$ OAE suppression/enhancement effects ~ an octave above $f_1$ in guinea pig for $f_2$=6.2 kHz with $f_2/f_1$=1.2.

## II. METHOD

### A. Animal surgery

Albino guinea pigs (300–550 g) were anesthetized with Nembutal (35 mg/kg i.p.) and Atropine (0.06–0.09 mg i.p.), followed approximately 15 min later by Hypnorm (0.1–0.15 ml i.m.). Neuroleptanaethesia was maintained with supplemental doses of Nembutal and Hypnorm. Guinea pigs were tracheostomized and mechanically ventilated on Carbogen (5% $CO_2$ in $O_2$) with body rectal temperature maintained at approximately 38.5 °C. The head was positioned using a custom-made head holder that could be rotated for access to the ear canal. Heart rate was monitored throughout each experiment. The bulla was opened dorso laterally and a silver wire electrode placed on the round window niche for the recording and monitoring of the compound action potential (CAP). CAP thresholds were recorded between 4 and 20 kHz in 2 kHz steps throughout each experiment. Pancuronium (0.15 ml i.m.) may have been administered to reduce physiological noise associated with muscle contractions.

### B. Signal generation and data acquisition

The method for stimulus delivery has been described previously (Withnell *et al.*, 1998; Withnell and Yates, 1998). Briefly, the acoustic stimuli were delivered by a Beyer DT48 loudspeaker placed approximately 4 cm from the entrance to the ear canal. Ear canal sound pressure recordings were made by a Sennheiser MKE 2-5 electrostatic microphone fitted with a metal probe tube (1.2 mm long, 1.3 mm i.d., 1500 Ω acoustic resistor) positioned approximately 2 mm into the ear canal. The microphone and probe tube combination was calibrated against a Bruel and Kjaer 1/8 in. microphone. The output from the probe tube microphone was amplified 20 dB, high-pass filtered (0.64 kHz, 4 pole Butterworth) and transmitted as a balanced input to one of the analogue input channels of a Card Deluxe sound card (Digital Audio Labs, www.digitalaudio.com) with an additional 4 dB of gain provided by the sound card. The signal was digitized at a rate of 32 000 Hz.

Signal generation and data acquisition was computer controlled using SYSRES (Neely and Stevenson, 2002). Data were recorded for a total of 65.536 s at each $f_2$ stimulus frequency. The stimulus complex consisted of two amplitude-modulated pure tone stimuli, digitally generated and output separately on two different output channels, mixed without amplification and buffered by a Tucker-Davis Technologies HB6 loudspeaker buffer-amplifier. Amplitude-modulated, or continuously varying in level, stimulus tones have been used previously to obtain DPOAE input-output functions (Neely *et al.*, 2005), a technique that produces input-output functions in a shorter time period than that obtained for measurements using discrete stimulus levels (Neely *et al.*, 2005) and also provides for OAE amplitude and phase in an $L_1$-$L_2$-OAE space (a three-dimensional plot of OAE amplitude or phase versus $L_1$ and $L_2$).

### C. Procedure

In this study, amplitude-modulated stimuli were used to generate an amplitude-modulated $2f_1$-$f_2$ OAE, i.e., an OAE that varies in amplitude over time in a stimulus-level dependent manner. This provides for an OAE that can be examined in terms of a basal-to-$f_2$ distortion product generator by comparing $2f_1$-$f_2$ OAE recorded before and after acoustically traumatizing the basal turn where any stimulus-level dependent sensitivity in terms of OAE generation is implicit in the OAE. Stimulus-frequency ratio was held constant at $f_2/f_1$ =1.2 with $f_2$ fixed in value. Ten separate measurements were made for ten different values of $f_2$, $f_2$=8–8.94 kHz with a 94 Hz step size. Amplitude modulation of the two pure tone stimuli provided $L_1$ and $L_2$ varying from approximately 40 to 80 dB SPL. Measurement at each of the ten $f_2$ frequencies was performed twice prior to acoustically traumatizing the base of the cochlea and once after acoustically traumatizing the base of the cochlea.

### D. Traumatizing tone

For all experiments reported in this paper, a 12 kHz, ~100–105 dB SPL tone generated by an Agilent 33120A signal generator connected to a Beyer DT48 loudspeaker via a Tucker-Davis Technologies HB6 loudspeaker buffer-amplifier was delivered to the ear for approximately 10 min to traumatize the basal region of the cochlea. The magnitude of the damage produced was quantified by the measurement of CAP thresholds before and after traumatizing the cochlea. CAP threshold was visually determined using the software equivalent of an averaging oscilloscope (number of responses averaged=40).

### E. Data analysis

Data analysis was performed in MATLAB. The analysis to extract $2f_1$-$f_2$ from the ear canal recording was written in MATLAB (source code written by Dr. Stephen Neely), summarized as follows:

i. a discrete fast fourier transform (FFT) was performed on the data ($2^{21}$ samples recorded at a sampling rate of 32 kHz) recorded at each $f_2$ frequency;
ii. the FFT was subsequently Blackman windowed centered at $2f_1$-$f_2$ and truncated to a size of 8192 bins centered at $2f_1$-$f_2$;
iii. an inverse fast Fourier transform (IFFT) was performed on the truncated, windowed FFT to obtain the signal complex amplitude waveform versus time. Truncation of the data set to 8192 bins produces an IFFT that has been down sampled to a sampling rate of 64 Hz;
iv. the IFFT waveform was divided into two equal lengths and then averaged;
v. the Hilbert envelope (the modulus of the IFFT) was then calculated; and
vi. the amplitude was then obtained from the Hilbert envelope according to the formula amplitude $=20^{*}\log[|\mathrm{IFFT}|/(2^{*}10^{-5})]$.

This analysis to extract $2f_1$-$f_2$ from the ear canal recording is identical to that reported in Neely *et al.* (2005). A microphone probe tube correction was applied after step (vi).

The $2f_1$-$f_2$ OAE recorded at each $f_2$ frequency was measured twice pre-trauma and once post-trauma. To examine quantitatively the effect of damage to the basal region of the cochlea on the amplitude of the $2f_1$-$f_2$ OAE obtained, a correlation coefficient was calculated for

i. the log of the Hilbert transform of the two pre-trauma $2f_1$-$f_2$ OAE measurements and
ii. the log of the Hilbert transform of the initial pre-trauma measurement versus the log of the Hilbert transform of the $2f_1$-$f_2$ OAE measurement recorded post-trauma to the base of the cochlea.

The two pre temporary threshold shift (TTS) recordings provided a lower boundary for the correlation coefficient when there has been no change in cochlear status. Measurement of the $2f_1$-$f_2$ OAE over an extended $f_2$ frequency range (8–8.94 kHz) provides additional data for comparison of pre versus post TTS measurements. Calculating the correlation coefficient for two wave forms in time incorporates any differences in frequency and/or phase of the Hilbert envelope but not simple scaling changes of the waveform. Alteration in the gain of the active process associated with damage to the outer hair cells (OHCs) produces a linearization of the growth of the $2f_1$-$f_2$ OAE (e.g., Neely *et al.*, 2003) and so would produce an alteration in the shape of the Hilbert envelope, the Hilbert envelope for an amplitude-modulated OAE varying in amplitude over time in a stimulus-level dependent manner. Therefore, comparison of the correlation coefficient of two OAE responses pre-acoustic trauma to the base of the cochlea versus the correlation coefficient pre-versus post-acoustic trauma should be sensitive to changes in the OAE associated with changes to the region/s of the cochlea responsible for generation of the $2f_1$-$f_2$ OAE. Expressing the Hilbert envelope of the OAE in logarithmic terms before calculating the correlation coefficient gives greater weighting to OAE obtained at lower stimulus levels or that part of the OAE that will be affected more significantly by changes to the gain of the active process (the log of the Hilbert envelope tends to weight the responses at all stimulus levels more equally).

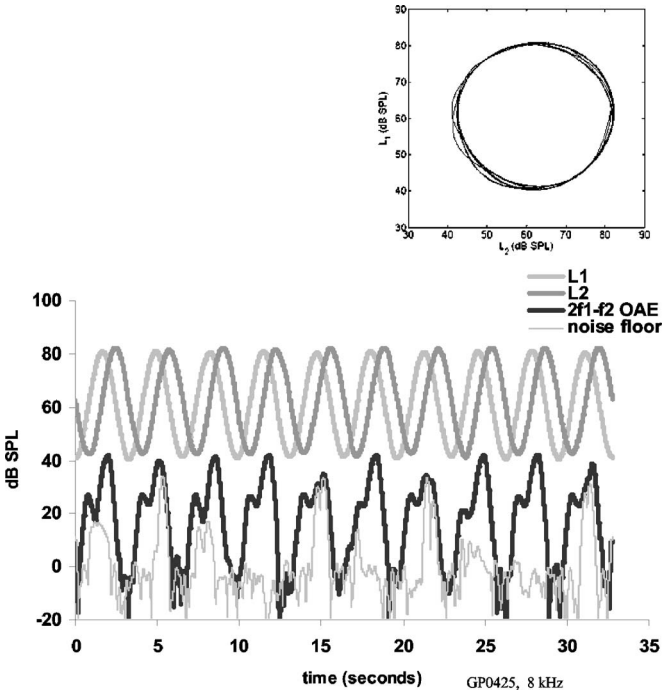


FIG. 3. The log of the Hilbert envelopes of two amplitude-modulated stimulus tones and the $2f_1$-$f_2$ OAE, obtained from analysis of the ear canal recording. Also shown in the top right corner is the stimulus input-output function.

## III. RESULTS

Figure 3 shows an example of the log of the Hilbert envelope of the amplitude-modulated stimulus tones and the $2f_1$-$f_2$ OAE. Stimulus level in Fig. 3 ranges from ~40 to 80 dB SPL, producing an OAE that varies in amplitude over time in a stimulus-level dependent manner. A stimulus-level range of ~40–80 dB SPL is representative of all the stimuli used in this study. In this particular example, the two stimuli are amplitude modulated at the same rate (~0.3 Hz) with a 90° phase difference or 0.83 s difference between amplitude-modulated stimulus envelopes. An identical amplitude modulation rate with no phase difference is equivalent to co-varying stimulus intensity level. By introducing a 90° phase difference, an input-output function is obtained that has an $L_1$-$L_2$ space that is an ellipse (Neely *et al.*, 2005), i.e., for any given $L_1$ it does not have a wide range of $L_2$ values (see inset, Fig. 3) but nevertheless examines the $2f_1$-$f_2$ OAE over a range of stimulus levels. To obtain a $2f_1$-$f_2$ OAE that arises from a more complete two-dimensional stimulus level space requires that the amplitude modulation rate of the two stimuli differ (see Neely *et al.*,

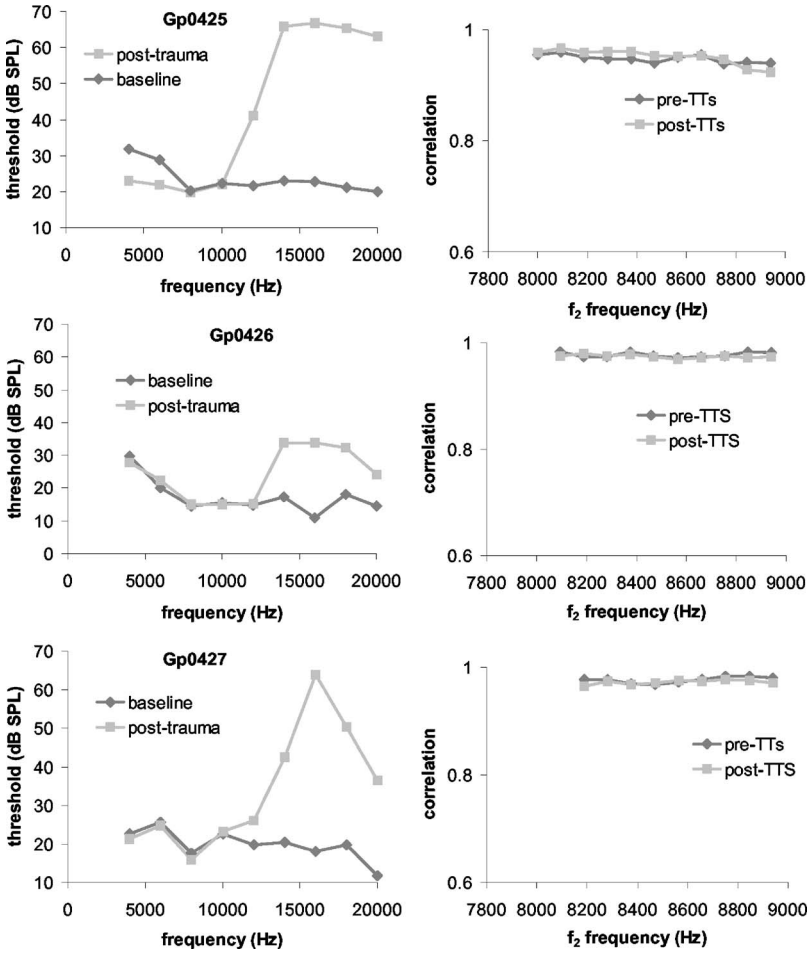


FIG. 4. CAP audiograms pre- vs post-trauma and the correlation of the OAE obtained in two pre-trauma measurements and pre- vs post-trauma when the change in CAP thresholds produced by the acoustically traumatizing tone does not extend into the region of the cochlea tuned below 10 kHz. No change is observed in the correlation coefficient of the two pre-TTS OAEs vs the pre- and post-TTS OAEs for all three animals.

2005). It is evident from Fig. 3 that the log of the Hilbert envelope of the $2f_1$-$f_2$ OAE varies reasonably systematically over time for an amplitude-modulated stimulus complex that repeats systematically as would be expected from a memoryless or limited memory system on the time scale we are observing the OAE; note that the recorded response was essentially the real time recording without averaging (number of averages=2), the $2f_1$-$f_2$ OAE extracted from the ear canal recording without the benefit of synchronous averaging by narrow-band windowing the response about the center frequency of interest using a Blackman window. The $2f_1$-$f_2$ OAE time waveform shows a double peak within each cycle of modulation of the stimuli, such an amplitude variation or notch being consistent with a vector cancellation of two OAE components (Brown, 1987; Whitehead *et al.*, 1992; Mills and Rubel, 1994) or shifts in outer hair cell operating point (Lukashkin *et al.*, 2002). Also shown in Fig. 3 is the noise floor which is calculated as the difference between the first and last half of the recordings (Sec. II E step iv of the Method section but taking the difference rather than the average); surges or peaks in the noise floor are evident coincident with peaks in the OAE response and presumably reflect incomplete cancellation of the OAE rather than noise, implying a recorded response that is not completely stationary over time, although there is no suggestion that such incomplete cancellation is systematic.

To examine the role that the basal region of the cochlea has on the generation of the $2f_1$-$f_2$ OAE, the base was damaged using an acoustically traumatizing 12 kHz, ~100–105 dB SPL presented for approximately 10 min, the 12 kHz tone presented with the intention of producing damage to the region of the cochlea basal to the initial nonlinear generation region of the $2f_1$-$f_2$ OAE. Figure 4 shows three examples of results obtained when the change in CAP thresholds produced by the acoustically traumatizing tone did not extend into the region of the cochlea tuned below 10 kHz, where the initial nonlinear ($f_2$) region of generation of the $2f_1$-$f_2$ OAE extended from 8 to 8.94 kHz. The left panels show CAP thresholds before and after acoustically traumatizing the base of the cochlea. The right panels show correlation coefficients obtained at each $f_2$ stimulus frequency before and after the TTS. CAP thresholds are unaltered up to 10 kHz but significant change in CAP thresholds above 10 kHz is apparent, including the 13.3–14.9 kHz region from whence a harmonic distortion mechanism would produce $2f_1$-$f_2$ as a quadratic distortion tone, i.e., where the second harmonic of the BM response to the $f_1$ stimulus, $2f_1$, interacts nonlinearly with the BM response to the $f_2$ stimulus to generate $2f_1$-$f_2$. No change is observed in the correlation of the two pre-TTS OAEs versus the pre- and post-TTS OAEs for all three animals, suggesting no change in $2f_1$-$f_2$ OAE amplitude for $L_1$ and $L_2$ varying from approximately 40–80 dB SPL, after damaging the basal region of the cochlea. This analysis does not support the region of the cochlea basal to the initial nonlinear generation region, $f_2$, contributing to the generation of the $2f_1$-$f_2$ OAE.

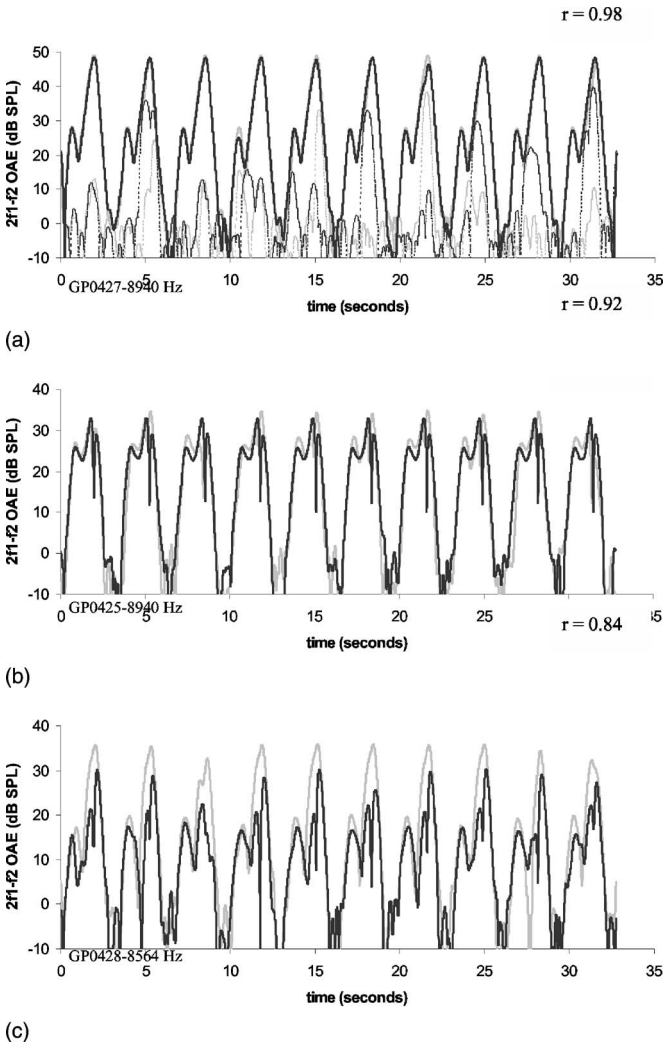


FIG. 5. Examples of $2f_1$-$f_2$ OAE Hilbert envelopes with correlation coefficients of 0.98, 0.92, and 0.84. The waveform envelopes in panel (a) for a correlation of 0.98 are basically identical, in panel (b) for a correlation of 0.92 the discrepancy is small and confined to the peak of the responses, and in panel (c) for a correlation of 0.84 the discrepancy is larger than in (b) although the wave forms retain somewhat similar shapes.

To illustrate the differences in waveform morphology that produce various correlation coefficients, Fig. 5 shows examples of $2f_1$-$f_2$ OAE Hilbert envelopes with correlation coefficients of 0.98, 0.92, and 0.84. The waveform envelopes in panel (a) for a correlation of 0.98 are basically identical, in panel (b) for a correlation of 0.92 the discrepancy is small and confined to the peak of the responses, and in panel (c) for a correlation of 0.84 the discrepancy is larger than in (b) although the general waveform envelope shape is retained.

Figure 6 shows two examples of results obtained when the change in CAP thresholds produced by the acoustically traumatizing tone *does* extend into the region of the cochlea tuned below 10 kHz. The left panels show CAP thresholds to have been affected within the $f_2$ region that generates $2f_1$-$f_2$ as a cubic distortion tone subsequent to exposure to the acoustically traumatizing tone. The OAE correlations corresponding to the left panels show changes in the OAE occurred post TTS, correlation coefficients reducing after acoustically induced damage to the cochlea.

Figure 4 illustrates that $2f_1$-$f_2$ OAEs before and after damage induced basal to the initial nonlinear generation region of the $2f_1$-$f_2$ OAE are highly correlated while Fig. 6 illustrates that when such damage extends to include the initial nonlinear generation region of the $2f_1$-$f_2$ OAE, changes to the OAE are quantifiable in terms of changes in waveform morphology expressed in terms of a correlation coefficient. But does this discount a significant source of $2f_1$-$f_2$ basal to $f_2$, either through a harmonic mechanism or simply a basalward shift in the region of generation, as might occur with increasing stimulus level, contributing to the generation of the $2f_1$-$f_2$ OAE? To explore this possibility, the $2f_1$-$f_2$ OAE was examined during that part of the time domain recording where $L_1 > 60$ dB SPL, based on the premise that $L_1$ stimulus level dependence is greater than $L_2$ stimulus level dependence because the amplitude of the $2f_1$-$f_2$ OAE is determined by the square of the BM response to the $L_1$ tone multiplied by the BM response to the $L_2$ tone (Withnell and Yates, 1998). Figure 3 seems to support this assumption,

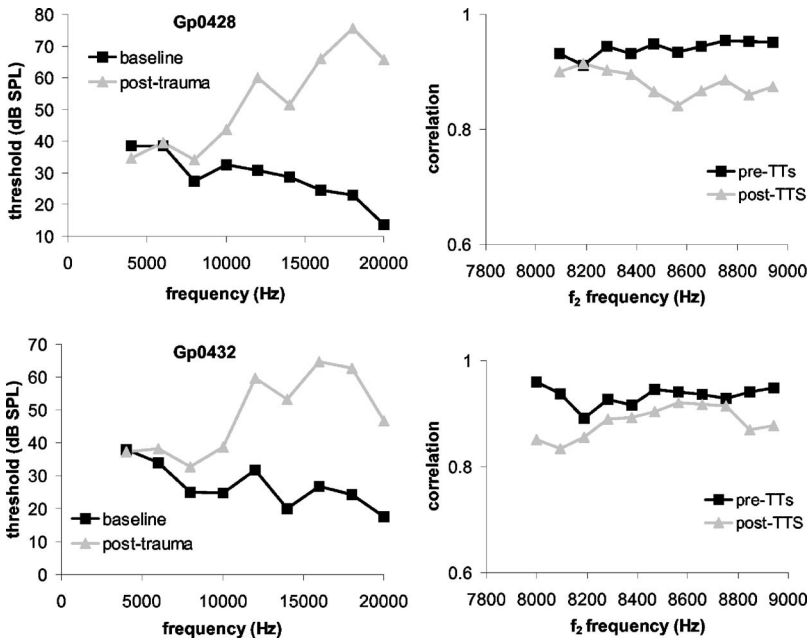


FIG. 6. CAP audiograms pre- vs post-trauma and the correlation of the OAE obtained in two pre-trauma measurements and pre- vs post-trauma when the change in CAP thresholds produced by the acoustically traumatizing tone *does* extend into the region of the cochlea tuned below 10 kHz. The OAE correlation coefficients show changes in the OAE occurred post TTS, correlation coefficients reducing after acoustically induced damage to the cochlea.

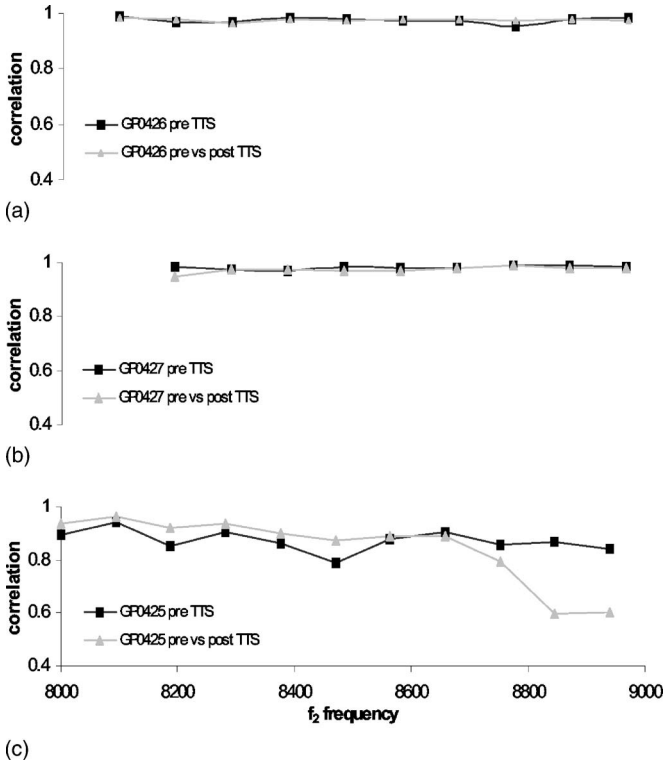


FIG. 7. A comparison of the correlation coefficients of the two pre-TTS OAEs vs the pre- and post-TTS OAEs obtained when $L_1 > 60$ dB SPL (from the data set reported in Fig. 4). It is evident that the correlation coefficients do not differ except above 8.7 kHz in panel (c), arguing against a significant source of $2f_1$-$f_2$ basal to $f_2$ contributing to the generation of the $2f_1$-$f_2$ OAE when $L_1 > 60$ dB SPL.

with OAE amplitude a maximum when $L_1 = L_2$[3] and then rolling off rapidly as $L_1$ decreases, even though $L_2$ continues to increase in amplitude for approximately 0.5 s after $L_1 = L_2$. Figure 7 compares the correlation coefficients of the two pre-TTS OAEs versus the pre- and post-TTS OAEs obtained when $L_1 > 60$ dB SPL from the data set reported in Fig. 4. It is evident that the correlation coefficients do not differ except above 8.7 kHz in panel (c), arguing against a significant source of $2f_1$-$f_2$ basal to $f_2$ contributing to the generation of the $2f_1$-$f_2$ OAE when $L_1 > 60$ dB SPL.

Measurement of the $2f_1$-$f_2$ OAE using amplitude-modulated stimuli provides for the generation of an $L_1$-$L_2$ cubic distortion tone (CDT) space input-output function that should be a very sensitive measure of the effects of induced cochlear damage. The data in Figs. 4 and 6 suggest that the amplitude of the $2f_1$-$f_2$ OAE is only altered when cochlear damage extends into the $f_2$ region that generates $2f_1$-$f_2$ as a cubic distortion tone. It does not support $2f_1$-$f_2$ being generated as a quadratic distortion tone at the cochlear $2f_1$ location or as a result of broadened stimulus-related BM excitation patterns with basal-ward shifts in the maximum BM responses extending the region of significant nonlinear interaction basal to $f_2$.

## IV. DISCUSSION

The traditional notion for the generation of intermodulation distortion product OAEs, a two source interference model (e.g., Talmadge *et al.*, 1999; see Fig. 1), was brought into question with the findings of Martin *et al.* (1999) that showed, with the addition of a third (suppressor) tone, iso-suppression/enhancement contours with changes in the $2f_1$-$f_2$ OAE for a suppressor tone more than one octave above the stimulus tone frequencies. That is, the suppressor tone, at a frequency an octave above $f_2$, produced a significant reduction in $2f_1$-$f_2$ OAE amplitude. The findings of Martin *et al.* included results to an experiment analogous to that reported in this paper, i.e., the use of a traumatizing tone to damage the cochlea basal to the $f_2$ region in a rabbit [see Fig. 8 of Martin *et al.* (1999)); after exposure, the alteration to the $2f_1$-$f_2$ OAE amplitude observed with addition of the suppressor tone was removed. Martin *et al.* (1999) interpreted findings such as this to be consistent with the acoustically traumatizing tone having affected source/s of $2f_1$-$f_2$ OAE basal to $f_2$. Fahey *et al.* (2000) posited that one of two mechanisms could account for this phenomenon, a catalyst mechanism and/or a harmonic distortion mechanism. The harmonic mechanism, as described by Fahey *et al.* (2000), would not be expected to operate at intermediate and high frequencies where the high-frequency cutoff of the basilar membrane response is sharp, i.e., harmonics do not achieve sufficient amplitude at their own characteristic frequency place to generate sufficient nonlinear interaction quadratic distortion tone (QDT) to produce $2f_1$-$f_2$. Martin *et al.* (1999) used an $f_1$ of 2.53 kHz for this experiment, representing the mid or intermediate frequency region where the high-frequency cutoff is steep (Borg, 1988) and so a harmonic mechanism would not be expected to play a role as a basal to $f_2$ source of $2f_1$-$f_2$. In contrast, the catalyst mechanism, where $2f_1$-$f_2$ OAE arises out of a concatenation of nonlinear interaction of the three stimulus tone BM responses, their harmonics, and intermodulation distortion products is predicted to generate the response pattern observed (Fahey *et al.*, 2000). The absence in this paper of evidence for a harmonic mechanism in the generation of the $2f_1$-$f_2$ OAE for 8 kHz $\leqslant f_2 \leqslant$ 8.94 kHz (at least up to a stimulus level of 80 dB SPL) supports the prediction of Fahey *et al.* (2000).

### A. Origin of the $2f_1$-$f_2$ OAE

Damage to the cochlea basal to the $f_2$ place does not alter the $2f_1$-$f_2$ OAE, a finding that establishes the initial generation site of the emission as being in the region of $f_2$ for the stimulus levels used. That is, while the region that generates the distortion is expected to be distributed over some length of the cochlea, it predominantly arises initially from the locus of $f_2$ for stimulus levels up to ~80 dB SPL. Acoustically traumatizing the base of the cochlea or preexisting basal damage provides an alternate means of examining the spatial extent of generation of the $2f_1$-$f_2$ OAE without the addition of a third (suppressor) tone that, in a nonlinear, active cochlea, may complicate examination of the origin of the $2f_1$-$f_2$ OAE.

The recent development of a scanning laser interferometer that provides for basilar membrane vibration to be measured as a function of longitudinal location (Ren, 2004) affords the possibility that the spatial extent of generation of $2f_1$-$f_2$ intermodulation distortion on the basilar membrane

can be examined directly in the future. It is evident though from this study that, in the absence of a third tone, the $2f_1$-$f_2$ OAE generated by a two-tone stimulus complex predominantly has an initial generation site solely in the region of $f_2$.

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[1]Note that apical to the resonant place for $2f_1$, the BM response at the frequency $2f_1$ will be negligible, regardless of the stimulus level of $f_1$ (Lighthill, 1991).

[2]The experimental paradigm involved varying only $L_2$ with $f_2/f_1=1.6$

[3]It is interesting to note from Fig. 3 that the $2f_1$-$f_2$ OAE has a maximum amplitude when $L_1=L_2$ in guinea pig for $f_2/f_1=1.2$, in contrast to previous findings in humans and not supportive of the contention that the amplitude of the OAE is maximized when the BM responses to the two stimulus tones at the $f_2$ place are equal (when $L_2/L_1 \approx 10$ dB).

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