



## Introduction

There are relatively few data concerning the influence of stimulus level and hearing status (normal or sensorineural hearing loss, SNHL) on the latencies of stimulus frequency otoacoustic emissions (SFOAE), and few data comparing SFOAE and distortion product otoacoustic emission (DPOAE) latencies. In the limit of low stimulus levels, the linear coherent reflection emission model of SFOAE generation [1, 2] predicts that the spectrum of a SFOAE response, apart from fine-structure effects, resembles the spectrum of the evoking stimulus. Experimental evidence at low and moderate levels shows a more complex relationship [3], which may be due to intermodulation distortion and spatial variation in the reflectance magnitude [4, 5, 6], or two-tone suppression effects on the basilar membrane [3].

This model further predicts that SFOAE latency is a measure of the round-trip travel time between the ear canal and the tonotopic-place region on the basilar membrane [7], and that this latency is proportional to the local value of the auditory filter bandwidth [1]. In particular, auditory filter bandwidths have been estimated from frequency-domain measurements of SFOAE group delay. The resulting equivalent rectangular bandwidth ( $Q_{\text{erb}}$ ) from SFOAE measurements predicts behavioral measurements of frequency tuning in normal-hearing humans [8]. A relationship between emission latency and stimulus level or hearing status is expected, but has not yet been shown, although a SFOAE auditory tuning bandwidth has been shown from frequency-domain measurements to decrease with increasing self-suppression level [9].

The present report extends our previous work [3] by evaluating effects of stimulus level on transient-evoked SFOAE and DPOAE in groups of normal and impaired individuals. SFOAE latencies were measured directly in the time domain. Responses were examined in relation to the evoking stimuli, which included tone pip (pp) pairs, gated tone (gg) pairs, and for DPOAE, continuous and gated tone (cg) pairs. The aims of this study were to: (1) examine effects of stimulus level on time-domain recorded OAE latency; (2) determine whether a correlation exists between cochlear tuning estimates ( $Q_{\text{erb}}$ ) and hearing sensitivity; (3) test whether two-tone suppression acts differentially at higher and lower frequencies within the bandwidth of a short-duration stimulus used to evoke SFOAEs; and (4) if a two-tone suppression effect exists, determine whether the effect varies with hearing status.

## Methods

We re-analyzed data reported in Konrad-Martin and Keefe [3], which describes the detailed methods used to acquire the SFOAE and DPOAE responses. Synchronous spontaneous otoacoustic emissions (SSOAE) also were recorded in order to assess their contribution to SFOAEs and DPOAEs. Each OAE response was recorded in the time domain, and the response at the OAE frequency was extracted using a narrow-band filter.

**Subjects.** OAEs were measured in 19 normal-hearing (pure-tone thresholds 20 dB HL or better at half-octave frequencies between 0.25 and 8.0 kHz) subjects and 11 subjects with impaired hearing. All 11 hearing-impaired subjects had pure-tone thresholds > 20 dB HL at 4 kHz; 10 hearing-impaired subjects had pure-tone thresholds > 20 dB HL at 3 kHz. All subjects had normal 226-Hz tympanograms at the time of testing.

**Stimuli.** Stimulus classes for SFOAE and DPOAE were continuous tones, gated tones, and brief tone pips. For SFOAEs, pairs of tone pips (pp) and gated tones (gg) were presented at equal levels at center frequencies of 2.7 and 4 kHz. DPOAEs were obtained for primary frequencies  $f_1 = 3.34$  kHz and  $f_2 = 4$  kHz, which were presented at unequal levels  $L_1 = L_2 + 10$  dB. The primary DPOAE stimuli were a continuous  $f_2$  paired with a gated  $f_1$  (cg), or a gated  $f_2$  paired with a gated  $f_1$  (gg).

**Procedures.** Temporal envelopes of stimulus and OAE waveforms were obtained by narrow-band filtering at the SFOAE or DPOAE frequency. The pp responses were filtered using the same Hamming filter centered at the OAE frequency that was used to construct the tone-pip stimuli (window duration 10.5 periods of the center frequency). The gg and cg responses were filtered using a Remez finite-impulse response filter centered at the OAE frequency (pass band of 20 Hz, reject band of 50 Hz).

Each OAE was classified as present or absent based on a 6-dB ratio of time-domain signal and noise energies, and OAE latencies were calculated only for those OAEs classified as present. pp latency was the time between the peak SPL in the pip and OAE. gg latency was the pip to OAE onset-time difference taken at the 3-dB down-point from the respective steady-state levels. Each measured OAE latency was accepted as valid if it exceeded a minimum criterion latency  $T_{\text{min}}$ , equal to one-half the duration of the pip stimulus.  $T_{\text{min}}$  was 1.94 ms at 2.7 kHz, and 1.31 ms at 4.0 kHz. This lower bound controlled for time-domain artifact during the time that the pip stimulus was on. The same criterion was used for the gg and cg conditions to maintain consistency with the pp condition, even though the stimuli differed slightly in their onset and decay times.

The ppSFOAE responses were analyzed in the frequency domain for the presence of two-tone suppression within the bandwidth of the pip stimulus. Based on limited data, Konrad-Martin and Keefe [3] hypothesized that since high-frequency tones are more effective than low-frequency tones at suppressing SFOAEs [10], an asymmetry in the SFOAE spectrum might be produced with larger SFOAE energy in the high-frequency half of the pip spectrum than in the low-frequency half. The SFOAE energy was separately summed over the upper- and lower-frequency halves of the main lobe of the pip spectrum, and each was converted to SPL. This asymmetry was separately assessed in normal and hearing-impaired ears.

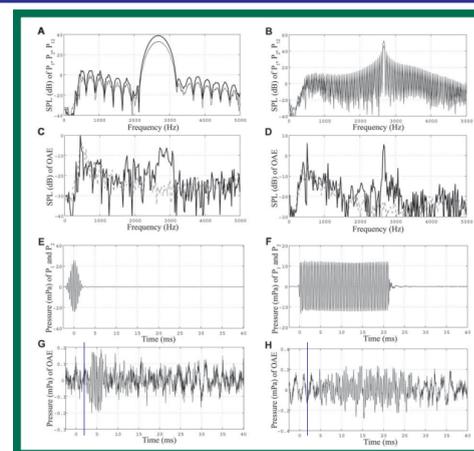
Shera et al. [8] showed that the SFOAE round-trip latency  $T(f)$  at frequency  $f$  predicts the behavioral  $Q_{\text{erb}}$  at the same center frequency  $f$  by  $Q_{\text{erb}} = (k/2) T f$ , in which  $k(f)$  also varies with  $f$ . Their estimated means and confidence intervals for  $Q_{\text{erb}}$  and  $k$  are shown below (also see Fig. 3).

Our measurements of round-trip latency  $T$  were converted into estimates of  $Q_{\text{erb}}$  using the above formula based on the mean value of  $k$ . We assume that their value of  $k$ , which Shera et al. calculated based on measurements in normal ears at a SFOAE probe level of 40 dB SPL, is also applicable for deriving estimates of  $Q_{\text{erb}}$  in ears with hearing loss and over a range of probe levels. Thus,  $k$  is assumed to be a frequency-dependent constant such that changes in round-trip latency due to probe level or hearing status are proportional to changes in mechanical tuning bandwidth on the basilar membrane.

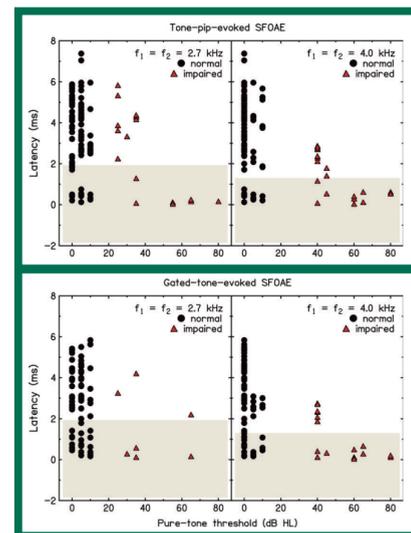
	$f$ (kHz)	Mean	95% Confidence Interval
$Q_{\text{erb}}$	2.7	17.1	16.1-18.2
	4	19.2	17.7-20.9
$k$	2.7	2.15	1.88-2.44
	4	2.09	1.79-2.43

## Results

**Figure 1. Example of a Pip-evoked (pp) SFOAE (Left column) and a Gated-tone-evoked (gg) SFOAE (right column) in a normal ear.**  $f_1, f_2 = 2.7$  kHz.  $L_1, L_2 = 53.5$  dB SPL. (A, B) The stimulus pressure spectra. (C, D) The SFOAE spectrum. (E, F) The stimulus waveforms. (G, H) The SFOAE waveform.



- The SFOAE spectra resembled the stimulus spectra, except that for the ppSFOAE (Fig. 1A), the lower frequencies in the stimulus spectrum were reduced or absent in the SFOAE spectrum (compare Fig. 1C to Fig. 1A).
- The blue line in panels G and H indicate  $T_{\text{min}}$ .



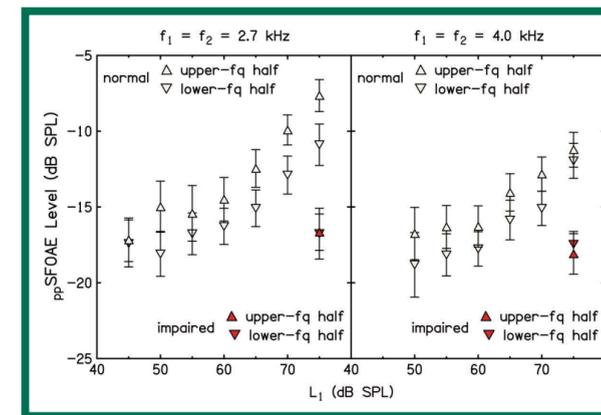
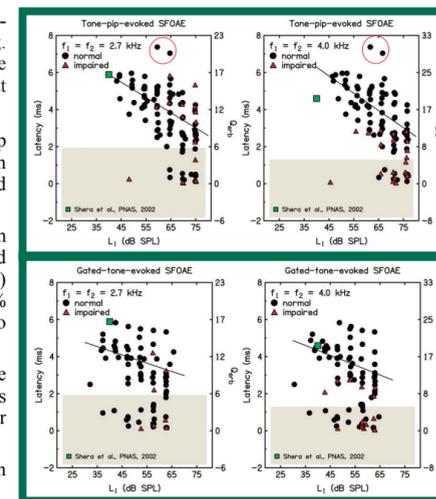
**Figure 2. Effect of hearing status on SFOAE latency at two frequencies.** SFOAE at 2.7-kHz (left column) and 4.0-kHz (right column) are plotted as a function of pure-tone threshold at the test frequency (3-kHz and 4-kHz, respectively). Data are collapsed across stimulus level. Rows correspond to the particular class of stimulus used (top row = ppSFOAE data; bottom row = ggSFOAE data), which are depicted in Fig. 1. Black symbols represent data from normal ears, red symbols represent data from impaired ears, and the shading identifies  $< T_{\text{min}}$ .

- Latencies were obtained in most subjects with thresholds below 45 dB HL. Subjects in our data set with impaired hearing and valid latencies had mild hearing loss (25 to 35 dB HL) at 2.7 kHz, and moderate hearing loss (40 to 45 dB HL) at 4.0 kHz.
- Latencies measured in impaired ears at 4.0 kHz were at the lower bound of the normal data.
- The proportion of subjects with valid latencies is given in the table below. It is likely that ppSFOAEs were present more often than ggSFOAE in impaired (and normal) ears because the pp stimuli were higher in peak SPL.

	ppSFOAE		ggSFOAE	
	2.7 kHz	4.0 kHz	2.7 kHz	4.0 kHz
Normal	18/19	18/19	15/19	15/19
Impaired	5/10	5/11	3/10	2/11

**Figure 3. Effect of stimulus level on SFOAE latency (left-hand axis) and  $Q_{\text{erb}}$  (right-hand axis).** Latency data from Fig. 2 are re-plotted as a function of stimulus level. Linear fits are shown for normal data that meet latency criteria ( $> T_{\text{min}}$ ). Format is the same as for Fig. 2.

- Increasing  $L_1$  significantly decreased ppSFOAE latencies (top row) and ggSFOAE latencies (bottom row), consistent with previous time-domain measurements of tone-burst-evoked SFOAE [11].
- With the assumption that cochlear transmission is minimum phase,  $Q_{\text{erb}}$  (with values on the right-hand axis) also decreased with increasing  $L_1$ . For ppSFOAE, latency (and, thus  $Q_{\text{erb}}$ ) decreased (over a 30-dB range from 45 to 75 dB SPL) by 45% and 48% at 2.7 and 4.0 kHz, respectively based on linear fits to the data.
- At 4 kHz, ppSFOAE and ggSFOAE  $Q_{\text{erb}}$  in normal ears were significantly larger than latencies in impaired ears, and it was usually not possible to measure latencies in impaired ears at lower  $L_1$ .
- High latency values (outliers in Fig. 3, top panels circled in red) were obtained in ears with strong SSOAE at 2.7 kHz.



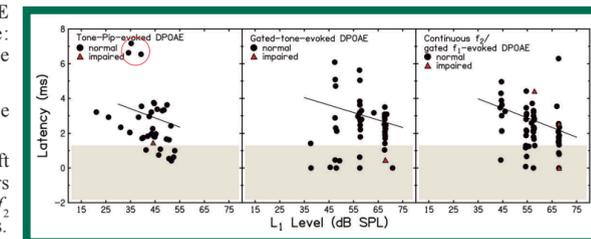
**Figure 4. Effect of stimulus level on SFOAE asymmetry at two frequencies.** The SPL in the lower- (down arrows) and upper-frequency halves (up arrows) of the pip stimulus spectrum is plotted at 2.7 kHz (left) and 4.0 kHz (right) as a function of  $L_1$  for normal (open symbols) and impaired ears (closed symbols).

- In normal ears, the ppSFOAE spectrum is more narrow than the eliciting stimulus, and response components correspond primarily to the upper-frequency portion of the stimulus pass-band (also see Fig. 1C).
- The SPL of the upper-frequency half of the ppSFOAE spectrum is significantly larger than the SPL of the lower-frequency half. This is consistent with the hypothesis that two-tone suppression acts within the

bandwidth of the pip, with greater suppression by the higher frequencies over lower-frequency components.

• While intermodulation distortion may also play a role in transient OAEs in some species [4, 6], we found little direct evidence of intermodulation distortion in these human data [3].

**Figure 5. Effect of stimulus level on DPOAE latency.** Left: ppDPOAE. Middle: ggDPOAE. Right: cgDPOAE. Format is the same as for Figs. 2 and 3.



- Level-dependent latency changes were significant for ggDPOAE and cgDPOAE.
- High latency values (outliers in Fig. 5, left panel circled in red) were obtained in ears with strong SSOAE (near DPOAE or  $f_2$  frequencies) or multiple internal reflections.
- DPOAE latency data were sparse, and often shorter in impaired ears relative to normal ears at equal SPL.
- Impaired ears were less likely to have DPOAEs present than SFOAEs present (see table).

	ppDPOAE	ggDPOAE	cgDPOAE
Normal	12/19	18/19	18/19
Impaired	1/11	1/11	2/11

## Summary and Conclusions

- (1) SFOAE latencies in normal ears at 2.7 and 4.0 kHz decreased with increasing stimulus level. This implies that  $Q_{\text{erb}}$  also decreases with increasing stimulus level.
- (2) SFOAE latencies at 4.0 kHz were significantly larger in normal compared to impaired ears. Latency variation with level and hearing status in the present report suggests that transient-evoked SFOAE provide a rapid, non-invasive measure of cochlear tuning.
- (3) Two-tone suppression effects were observed in time-varying SFOAEs, which may have significance for the cochlear encoding of complex sounds.
- (4) DPOAE latencies decreased with increasing stimulus level. When DPOAEs were present in impaired ears at higher stimulus levels, their latencies often were less than those in normal ears.
- (5) SFOAE and DPOAE latencies were increased in some ears with high reflectance as indicated by strong SSOAE and/or multiple internal reflections.

## References

- [1] Zweig G and Shera C (1995). The origin of periodicity in the spectrum of evoked otoacoustic emissions. *J. Acoust. Soc. Am.* 98, 2018-2047.
- [2] Talmadge CL, Tubis A, Long GR, Piskorski P (1998). Modeling otoacoustic emission and hearing threshold fine structures. *J. Acoust. Soc. Am.* 104, 1517-43.
- [3] Konrad-Martin D and Keefe DH (2003). Time-frequency analyses of transient-evoked stimulus-frequency and distortion-product otoacoustic emissions: Testing cochlear model predictions. *J. Acoust. Soc. Am.* 114, 2021-2043.
- [4] Yates GK and Withnell RH (1999). The role of intermodulation distortion in transient-evoked otoacoustic emissions. *Hear. Res.* 136, 49-64.
- [5] Talmadge CL, Tubis A, Long GR, Tong C (2000). Modeling the combined effects of basilar membrane nonlinearity and roughness on stimulus frequency otoacoustic emission fine structure. *J. Acoust. Soc. Am.* 108, 2911-2932.
- [6] Goodman SS, Withnell RH, De Boer E, Lilly DJ, Nuttall AL (2003). Cochlear delays measured with amplitude-modulated tone-burst-evoked OAEs. *Hear. Res.* in press.
- [7] Kemp DT (1980). Towards a model for the origin of cochlear echoes. *Hear. Res.* 2, 533-548.
- [8] Shera CA, Guinan JJ, and Oxenham AJ (2002). Revised estimates of human cochlear tuning from otoacoustic and behavioral measurements. *Proc. Natl. Acad. Sci.* 99, 3318-3323.
- [9] Lineton B and Lutman ME (2003). The effect of suppression on the periodicity of stimulus frequency otoacoustic emissions: Experimental data. *J. Acoust. Soc. Am.* 114, 871-882.
- [10] Brass, D and Kemp DT (1993). Suppression of stimulus frequency otoacoustic emissions. *J. Acoust. Soc. Am.* 93, 920-939.
- [11] Norton SJ and Neely ST (1987). Tone-burst-evoked otoacoustic emissions from normal-hearing subjects. *J. Acoust. Soc. Am.* 81, 1860-1872.

## Acknowledgements

Work supported by the Department of Veteran Affairs Rehab. R&D Service (E3239V) and the NIH (R01 DC03784).