

# Otoacoustic emission latency and cochlear mechanics

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## 1 Introduction

Measuring the latency of transient-evoked otoacoustic emissions (TEOAEs), defined as the time delay between the click stimulus and the maximum of each frequency component of the OAE response, provides information about the cochlear mechanisms.

From an experimental point-of-view, the latency is a decreasing function of frequency and of the stimulus level, as expected from basic theoretical considerations. Accurate experimental estimates of the TEOAE latency have been obtained using the time-frequency analysis of the TEOAE waveforms. Wavelet analysis was used for TEOAE latency estimates [1, 2]. Matching Pursuit (MP) algorithms [3] are also capable of identifying the resonant modes dominating the TEOAE waveform [4, 5]. Another time-frequency approach, based on an optimized bilinear algorithm, was used in DPOAE and SFOAE studies [6] to test OAE generation models.

According to transmission-line models, the latency is given by the round-trip delay associated with the forward transmission of each frequency component of the acoustic stimulus along the basilar membrane (BM) from the base to its own resonant place, and the backward transmission of the corresponding OAE component. The forward transmission of the stimulus is quite generally assumed to consist of a transverse displacement traveling wave on the BM, whereas no general consensus has been found yet on the nature of the backward OAE propagation mechanisms.

A class of transmission-line cochlear models [7, 8] assumes that a slow backward TW packet is generated for each frequency near its resonant place, transmitted back to the cochlear base and recorded as an OAE in the ear canal. Other models, supported by recent measurements of the phase of the BM motion at the distortion product frequency [9], explain the OAE backward propagation with fast longitudinal compression waves in the fluid [10, 11]. In this case, the backward transmission delay would be negligible. In any case, the forward transmission delay, which would amount, in the two cases, to half or to the total experimental OAE latency, is predicted to be intrinsically dependent on frequency due to the nature of the tonotopic map relating resonance frequency to cochlear position [12], which implies a longer path for lower frequencies.

The dependence on stimulus level is also expected because the slowing-down of each frequency component approaching its resonant place is more relevant as the quality factor of the resonance increases. Therefore, as the cochlear quality factor is well-known to be a decreasing function of the stimulus level, shorter latencies are expected as the stimulus increases. Another characteristic time that can be estimated from the OAE analysis is the phase-gradient delay (often named group delay), experimentally defined as the negative slope of the OAE phase-frequency relation. A few studies have measured TEOAE phase-gradient delays in humans [13, 14] and guinea pigs [15]. Several authors have reported measurements of the stimulus-frequency OAE (SFOAE) [16, 17] and of the distortion product OAE (DPOAE) phase-gradient delay [18, 19] in humans and small mammals.

The comparison between OAE phase-gradient delay and latency is not obvious. On one hand, if the system is approximately linear, a correspondence between the two delays is expected. On the other hand, the phase behavior of the OAE response is generally assumed to depend on the place-fixed or wave-fixed nature of the OAE generation mechanisms involved. A place-fixed mechanism is the linear reflection by random irregularities predicted by the coherent reflection filtering (CRF) theory [7, 8, 20, 21]. The most popular wave-fixed OAE source is nonlinear distortion. For a scale-invariant cochlea, a slowly rotating OAE phase (and therefore, little or no phase-gradient delay) is expected for wave-fixed mechanisms, whereas a fast rotating phase (with phase-gradient delay of the order of the OAE latency) is expected for place-fixed mechanisms [14]. Both linear reflection and nonlinear distortion are possible OAE sources, with different weights at different levels of stimulation, and the different predicted phase behavior has been used to disentangle their contributions by time-domain filtering, for SFOAEs and DPOAEs. For short transient stimuli, comments by Talmadge et al. [22] suggest that it is not clear if a null phase-gradient delay should be really expected for wave-fixed components. Time-frequency analysis of the TEOAE waveforms provides a direct estimate of the physical round-trip delay of multiplecomponent OAE responses, independently of the placefixed or wave-fixed nature of the OAE source.

From TEOAE latency estimates, objective (even if modeldependent) estimates of cochlear tuning have also been obtained [2, 23, 24, 25], which can be compared with other OAE-based or behavioral estimates of cochlear tuning.

Intrinsic cochlear phenomena, such as the presence in the TEOAE response of contributions from intermodulation distortion, long-lasting and spontaneous OAEs (SOAEs) and multiple intra-cochlear reflections, as well as extrinsic effects, such as noise, and the waveform deformation due to the acquisition window, may bias the TEOAE latency estimates. It is important to evaluate correctly these uncertainties, to get meaningful interpretations of the experimental findings [2, 5].

#### 2 Methods

TEOAE data have been analyzed, recorded with the ILO 292 Echoport system (Otodynamics, Ltd.), using the nonlinear acquisition paradigm. The stimulus level was varied between 60 and 90 dB pSPL in 5 dB steps.

A time-frequency analysis technique based on the continuous wavelet transform (CWT) was used to estimate the OAE latency as a function of frequency [1, 25]. Integrating the wavelet coefficients over 500 Hz frequency bands, seven wavelet band coefficients relative to bands centered between 1 and 4 kHz have been obtained. At lower and higher frequencies, the estimates of the relation between latency, frequency and stimulus level are affected by systematic errors associated with the cut-offs introduced by the acquisition window. The spectral latency relative to a given frequency band may be defined as the time for which the coefficient of that band reaches its maximum absolute value.

#### **3** Cochlear Modeling

The linearized equations in the frequency domain for a 1-d transmission line model can be written as:

$$\frac{\partial^2 P_d(x,\omega)}{\partial x^2} + k^2 P_d(x,\omega) = 0 \tag{1}$$

where:  $\omega$  is the angular OAE frequency, k is the wave vector,  $P_d$  is the differential pressure applied to the BM, and x is the longitudinal coordinate measured on the BM from the base. The resonance frequency is related to the cochlear position x along the BM by the Greenwood tonotopic map [12]:

$$\omega_0(x) = \omega_1 + \omega_{\max} e^{-k_\omega x}, \qquad (2)$$

where  $\omega_{max} = 2\pi \cdot 20655$  rad/s,  $\omega_l = 2\pi \cdot 145$  rad/s, and  $k_{\omega} = 1.382$  cm<sup>-1</sup>.

The wave vector is tonotopically resonant:

$$k^{2}(x,\omega) = \frac{k_{0}^{2}\omega^{2}}{\Delta(x,\omega)}.$$
 (3)

 $\Delta(x, \omega)$  is a resonant denominator that may include delayedstiffness terms [7, 20, 26], which effectively act as additional damping and anti-damping terms.

For a tonotopically resonant 1-d transmission line cochlear model, the roundtrip delay of a particular Fourier component of the OAE response to a pulse stimulus is given by the path integral of the inverse of the traveling wave velocity:

$$\tau_{OAE}(\omega) = 2 \int_{0}^{x} \frac{\partial Re(k)}{\partial \omega} dx$$
 (4)

This is the physical delay associated with the roundtrip path of a traveling wave packet centered at frequency  $f=\omega/2\pi$ propagating along the BM from the base to its resonance place and back. For each frequency component, the propagation velocity is determined by the group velocity, associated with the local relation between wave vector and frequency. The steeper the dependence on frequency of the wave vector, the slower the propagation. Due to the resonant nature of the dispersion relation Eq.(3), a significant contribution to the total latency comes, for each frequency component, from the part of the cochlear path close to the resonance place, where the relation between the wave vector and the frequency is the steepest.

The wavelet latency  $\tau_{OAE}(f)$  defined is a direct measure of this physical delay, because it measures the time interval between the click stimulus and the arrival of the most intense OAE wave packet at frequency *f*.

Using the scale invariance hypothesis, it is possible to get an approximate and model-dependent expression for the round-trip latency as a function of the quality factor of the resonance, where  $\beta \approx 0.9$  is a dimensionless constant [2]:

$$\tau_{OAE}(\omega) \cong \frac{k_0}{\omega k_\omega} \left( \beta \sqrt{Q} - \frac{2\omega}{\omega_0} \right) \tag{5}$$

By inverting this relation, it is possible to get a modeldependent estimate of cochlear tuning from latency measurements:

$$Q(\omega) = \frac{1}{\beta^2} \left( \frac{\omega k_{\omega}}{k_0} \left( \tau(\omega) - \tau_{nc} \right) + \frac{2\omega}{\omega_0} \right)^2 \quad (6)$$

From Eq.(5), it is evident that latency is an increasing function of tuning, because high tuning means steep relation between wave vector and frequency and slow propagation speed.

It is well-known that tuning decreases at high stimulus levels, as demonstrated by psychoacoustical measurements and by the analysis of DPOAE suppression tuning curves. This effect may be modeled by assuming that the damping function of the BM is described by a nonlinear function of the BM transverse displacement  $\xi$ . A cubic nonlinearity, due to a damping coefficient proportional to  $\xi^2$  explains a number of OAE and psychoacoustical observations. Van der Pol oscillators assume a quadratic damping term along with constant anti-damping, while other oscillator models [22, 27] assume damping terms made up of a quadratic term and a constant term, both positive. Studying the experimental relation between latency and stimulus level may provide information about these details of the nonlinear damping function.

The auditory brainstem response (ABR) latency is made up of a constant neural delay, of order 5 ms and largely independent of frequency and stimulus level, and of a cochlear forward transmission delay, which is frequency and level dependent [28, 29, 30, 31]. If we subtract from the ABR latency the neural contribution, we get an ABR estimate of the forward transmission delay, which can be compared with the OAE latency. Assuming that OAE propagate back via fast longitudinal compression waves in the fluid, the two delays should be equal, whereas, if the OAE transmission is associated with a backward TW on the BM, the OAE latency [32]. The accuracy of both measurement techniques is amply sufficient to discriminate between the two hypotheses.

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#### 4 **Results and Discussion**

#### 4.1 OAE latency and stimulus level

The experimental relation between average latency, frequency and stimulus level is shown in Fig.1. The expected decrease of latency with increasing frequency and stimulus level is clearly visible [2]. The latency estimates at frequency higher than 3-4 kHz cannot be fully trusted, because the acquisition window cuts part of the high-frequency response, causing systematic overestimates of the average latency.



Fig.1 Experimental relation between average latency, frequency and stimulus level. Average latency is defined here as the time of the maximum of the band wavelet coefficient averaged over the examined ears.

#### 4.2 OAE latency and cochlear tuning

From the latency data shown in Fig. 1 it is possible, using Eq.(6), to get the estimate of cochlear tuning, as a function of frequency and stimulus level, shown in Fig.2. Tuning increases with frequency and decreases with the stimulus level, which is in qualitative agreement with other OAE-based and psychoacoustical estimates [2].



Fig.2 Cochlear tuning as a function of frequency and stimulus level, obtained from the TEOAE latency data shown in Fig. 2, using Eq. (6). The expected decrease of tuning with increasing stimulus level can be observed, while the dependence on frequency is more irregular.

# 4.3 OAE latency and cochlear nonlinearity

The observed decrease of tuning with increasing stimulus level is associated with the nonlinearity of the cochlear amplifier. Assuming proportionality between the OAE pressure and the local transverse BM displacement  $\xi$ , these data may be used to test different models of the cochlear amplifier nonlinearity. A damping function  $\Gamma = \omega/Q$ , made up of a quadratic nonlinear term plus a positive constant term, representing the asymptotically linear response at low stimulus levels [22, 27], fits well the experimental data, whereas a Van der Pol oscillator model, which would have predicted in Fig.3 a negative intercept with the vertical axis, seems to be ruled out by these results [2].



Fig.3 Damping coefficient at 1, 2, 3, and 4 kHz, plotted against the square of the OAE spectral amplitude in that frequency band.

# 4.4 OAE latency and phase-gradient delay

The comparison between individual wavelet latency values and phase-gradient delays shows a good correlation between the two measurements [14].



Fig.4 Wavelet latency and phase-gradient delay at stimulus levels between 60 and 90 dB pSPL. Both delays decrease with increasing stimulus level, maintaining good correlation between them.

In Fig. 4 we show this comparison in the frequency band 2.5 kHz, where the data obtained at all stimulus levels are all plotted together, to show the correlated dependence on stimulus level of both characteristic times. This correspondence seems to favor phase-fixed mechanisms for the OAE generation [14], but the interpretation of these results needs further theoretical studies.

#### 4.5 OAE and ABR latency

A comparison between the OAE wavelet latency and the estimates of the cochlear latency obtained from ABR latency measurements available in the literature [28, 29, 30, 31] is shown in Fig.5. From the ABR latency a constant neural contribution of 5 ms (independent of stimulus level and frequency) was subtracted to obtain an estimate of the cochlear latency associated with the forward transmission of the acoustic stimulus along the BM. This estimate of the forward cochlear latency is multiplied by a factor two in Fig. 5, to compare it with the OAE latency. The agreement shown in Fig.5, as regards both the frequency dependence and the absolute latency value, suggests that the OAE backward transmission is well explained by slow TWs on the BM, and seems to contradict the hypothesis of a fast OAE backward transmission via longitudinal compression waves in the fluid [32]. In this case, the OAE latency should have been equal to (and not twice) the ABR estimate of the forward cochlear latency. The large vertical dispersion is due to the dependence on stimulus level, which is the same for both measurements.



Fig.5 OAE latency estimates (solid lines), compared to twice the ABR estimates of the forward cochlear delay (dotted lines). The agreement suggests a symmetry between the forward and backward propagation mechanisms.

### 5 Conclusion

The results reviewed here show the important contribution to the study of the cochlear physiology that may come from accurate experimental estimates of the OAE latency, if coupled to theoretical modeling concepts. Further efforts, both in the development of more accurate experimental and data analysis setups and in the refinement of theoretical models, are strongly encouraged to improve the usefulness of latency measurements, also for diagnostic purposes.

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