# *The influence of contralateral stimulation on the frequency width of spontaneous otoacoustic emissions in humans*

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**Abstract**

***Spontaneous otoacoustic emissions (SOAEs) were measured in humans under different auditory contralateral stimulus conditions. Different intensities of broadBand noise stimuli where used to examine the effects on upwards shifts of the SOAE and changes in the spectral width of the SOAE. These effects were compared to several other characteristics of the peak. Correlations were found between the intensities of the stimuli and the peak frequency width. The results also confirmed the relation between peak amplitudes and frequency width found in earlier research that used ear canal pressure stimuli instead of acoustic stimuli. A variety of data indicate that SOAEs are part of a system that helps to reduce acoustic trauma and lessen the masking of transients by background noise. However, much remains unknown about the system that produces these SOAEs. More research into SOAEs may not only explain more about human auditory perception, but could also provide clinical benefits.***

## 1. Introduction

*Spontaneous oto-acoustic emissions (SOAEs) are weak acoustic signals emitted from the inner ear. They have been recorded from amphibians, lizards, birds and mammals (Manley &Van Dijk, 2008). They are believed to be caused by instabilities in the hair cell epithelium in the inner ear. By active mechanical feedBack, hair cells improve the detection threshold and frequency selectivity of the ear.*

*While SOAEs seem to be partly a local mechanism, there are clear indications that efferent connections from the central nervous system have a significant influence. (Buno, 1978; Murata et al., 1980)*

*A better understanding of SOAEs will not only improve applications in hearing diagnostics, where otoacoustic emissions are broadly used. It will also improve existing cochlear models and aid computer hearing applications. (Slaney & Lyon, 1993)*

*In most research, these influences are examined by presenting a sound in the contralateral ear and carefully measuring the changes in the various types of oto-acoustic emissions in the ipsi lateral ear. This experiment followed a similar line. There are a number of effects that can influence the measurements on the contralateral ear aside from central efferents most notably the changes in middle ear transduction (stapedius reflex) and transcranial transmission of the signal. Both of these reduce the amplitude and increase the frequency in SOAEs.*

*In Owls it has been shown that when nerve bundles responsible for the stapedius reflex are removed,the effects of contralateral stimulation persist (Manley, Taschenberger, Oeckinghaus, 1999) In humans, the influence of changes in middle ear transduction seem to be only minor, especially in the higher frequency ranges. Recent research using differences in ear canal pressure levels instead of auditory stimulation indicated that in humans the frequency width and the chance in amplitude have a strong relation (Manley & Van Dijk, 2008). Our experiment will attempt to verify this using an auditory stimulus.*

*Since the middle ear transduction is a passive filter. it will have certain statistical properties that we can observe in the changes of an individual SOAE. A filter such as this will dampen and possibly shift the signal but will not change the width of a SOAE. The possibility of sound beingtransmitted transcranially is somewhat more difficult to eliminate.*

*Our expectation is that in all but the control conditions, the SOAE peak will shift upwards in frequency and downwards in height. It is also expected that the width of the SOAE peak will increase and the amplitude of the peak (here defined as the surface of the peak) will decrease.*

*This leads (us) to the following hypothesis:*

*Reduction of the SOAE amplitude by contralateral stimulation is caused by reduction of the amplitude of intra-cochlear emission source signal.*

# 2. Materials

*The setup was developed with the aim of minimizing noise and maximising sensitivity in the areas where SOAEs are most common (1000Hz - 6000 Hz) Two microphones were used: one for receiving, and another for providing stimuli (if any). The preamplifier of the receiving microphone was always turned on, whereas/ while the other preamplifier was turned off, because the stimuli were generated elsewhere and the reception of the microphone was not required.*
*The microphones were fitted with custom-built tips to allow the fitting of reusable earpieces. The receiving microphones were powered by batteries in order to prevent any noise from the power supply. The signal from the listening microphone was passed to the SR560, which was used as a bandpass filter from 300 Hz to 10000 Hz. The high-pass was used in order make sure that we did not overload the A/D-converter, the low-pass in order to prevent allaising. Because the A/D converter sampled at 40.000 Hz theoretically the low-pass could have been set to 20.000 Hz. However, experience has shown that most signals above 10.000 Hz are riddled with artifacts. The SR560 was also used to amplify the signal by a factor 2 to 20 in order to maximise the use of the dynamic reach of the A/D-converter. Signals were later digitally scaled back to their original level.*
*The oscilloscope and SR 760 were used to check if the signal came through properly during the tests.*
*The computer's AD converter was used to record the actual signal for later processing. The 600Ohm resistor was added because the SR560 requires a 600Ohm exit resistance in order for its gain to be correctly calibrated. Since the other parallel components have resistances of 1MOhm and higher, these have little influence.*
*The stimulus microphone preamplifier was turned off as the stimulus was generated by the DS360. The DS360 was set to unbalanced, white noise burst output. Our stimulus strengths ranged from 0V to 3V. Again the oscilloscope was only used to monitor the signal during the tests.*

## 2.1. Overview of the setup

*In the experiments the following equipment was used:*

- *2 Etymotic Research Illinois ER-10C DPOAE microphones*
- *Probes with corresponding preamplifiers*
- *Stanford Research Systems Model SR560 Low-Noise Preamplifier*
- *Stanford Research Systems Model DS360 Ultra Low Distortion Function Generator*
- *Stanford Research Systems Model SR760 FFT Spectrum Analyzer*
- *Hameg HM 203-6 20MHz oscilloscope*
- *A G5 Apple computer with built-in AD converter*
- *And finally a custom build 600 Ohm terminating resistor.*

*The experiments were carried out in a sound dampening lab in the University Hospital in Groningen. The test subjects resided in a silent chamber, which has extremely high sound-dampening properties, while the signals were processed and recorded in another room.*

*This was organized in the following setup:*

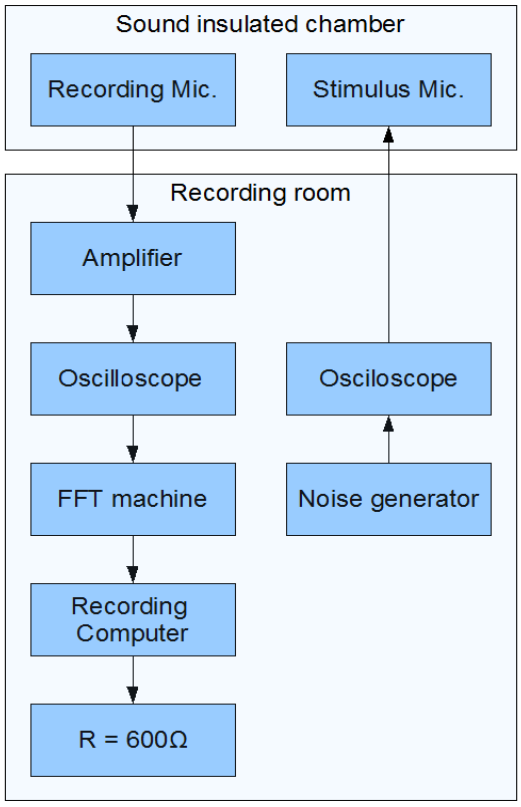


*Fig.1 Graphical representation of the measuring setup*

# 3. Methods

*Test subjects were seated in the 'silent chamber'. They were briefly informed of the purpose of the experiments and were told which procedures would be followed.*

*First there were two initial measurements under a no stimulus condition,in order to determine which ear had the most SOAEs. Measurements where continued on the ear with the most SOAEs. The measurement already taken from that ear was used as the initial 0V condition (further on referred to as 0V1)*

*Measurements were done under different noise conditions ranging from 0V to 3V. We started with a 0V condition. Then a fast build-up to a 3V condition, followed by 5 increasingly weaker conditions, decreasing to 0V. Then, using the same conditions, we built back up to 3V and we finished with another 0V condition. This makes 13 conditions in total. When we wanted to compare different effects of the stimuli the deltas were always compared to the average of all three 0V conditions.*

*After the normal measurements we determined the threshold of the stapedius reflex on a thypathic measuring setup using the contralateral broad band noise setting (since this agrees most with our test setup)*

*Finally, a standard audiometric setup was used to ensure that all test subjects had normal hearing.*

## 3.1. Data Processing

*Each recording was separately processed into an periodogram. This was done according to following procedure:*

*The signals were first sliced into fragments. An FFT is preformed on each fragment and fragments that are deemed to contain artifacts are then removed. The average is taken over all non-reject spectra to create a periodogram. The peaks are fitted with Lorentzian curve augmented by two terms to model a locally linear noise floor. This results in the following formula:*

$$A = \frac{B}{(f - F_{mid})^2 + \frac{F_{fwhm}^2}{4}} + N_{dc} + N_l * f$$

*(derived from Cauchy distribution in appendix)*

*The peaks where fitted with the fminsearch function in Matlab 2008b witch tried to minimize the summed squared error over a fragment of 75 Hz around the peak maximum:*

$$Error = \sum (A - A_{fitted})^2$$

*From these fits we extracted the following data:*

1. *Peak height, defined as:*

$$\frac{4B}{F_{fwhm}^2}$$

2. *Peak amplitude, or the surface under the curve minus the noise element, defined as:*

$$\frac{B}{F_{fwhm}} 2\pi$$

3. *Fmid which is a parameter of the fit*
4. *Ffwhm which is a parameter of the fit*

# 4. Results

*Our test subject pool consisted of 10 subjects aged 21 to 25 (mu = 22.1, s = 1.14, 6 female, 4 male).*

*We measured 5 subjects on the right ears and 5 on their left ear. After analysis of the data 6 subjects were found to have measurable SOAEs. One had 8, and 5 others had 1. bringing the total number of peaks analyzed to 13.*

*A portion of our measured data was dropped from our final analysis data. This happened for four different reasons*

*First of all, the data from the initial measurement of the contralateral ear was not used. Since there were only measurements under a single condition of that ear, no useful information concerning the effects of a contralateral stimulus could be obtained from that data.*

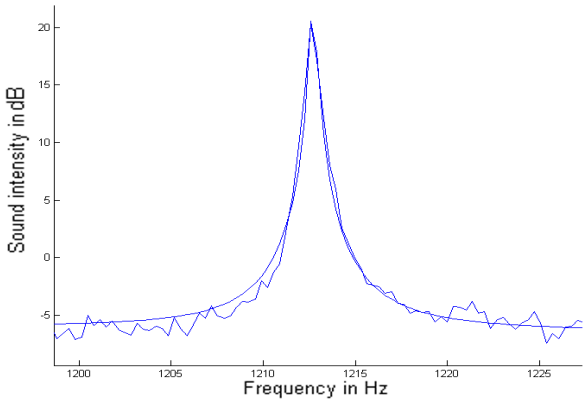


*Fig. 2 Example of what a fit of an SOAE looks like*

*Secondly, all frequency data outside the 300 Hz – 10 KHz band was dropped. Although this data was generated by the FFT of the original signal this data was filtered by our hardware bandpass to accommodate the A/D converter and as such it could not be used.*

*Thirdly, there were several peaks that were characteristic for measurement artifacts that were identically present in all data. As these where clearly not produced by the ear but by our equipment, these were also not analysed.*

*Finally, some of the peaks with smaller amplitudes became indiscernible from the noise floor under the 1V/3V conditions and no fit with informative parameters could be obtained. These fits where also dropped from our further analysis. Therefore we have more data under the lower noise conditions than under the higher noise conditions. This bias in our data is addressed later.*

| *Condition number* | *Condition name* | *dB Spl.* | *Occurrence* |
|---|---|---|---|
| *1* | *0V 1* | *0* | *13* |
| *2* | *3V 1* | *73* | *8* |
| *3* | *1V 1* | *63.45* | *10* |
| *4* | *0.3V 1* | *53* | *10* |
| *5* | *0.1V 1* | *45* | *10* |
| *6* | *0.03V 1* | *33* | *13* |
| *7* | *0V 2* | *0* | *13* |
| *8* | *0.03V 2* | *33* | *13* |
| *9* | *0.1V 2* | *43.45* | *11* |
| *10* | *0.3V 2* | *53* | *10* |
| *11* | *1V 2* | *63.45* | *10* |
| *12* | *3V 2* | *73* | *9* |
| *13* | *0V 3* | *0* | *12* |

*Table 1: An overview of the final dataset, the sequence of stimuli is supplemented with a column that shows the measured sound intensity of the stimulus. The final column indicated the number of successful fits under that condition. Only 8 of the 13 examined peaks could be fitted under all conditions. Because as earlier indicated it is sometimes hard to make a parameter fit that holds useful information under the greater stimulus conditions.*

*The third column is the result of a measurement of the stimulus in a Zwislocki-coupler although the spectra were not uniformly distributed. It was close enough to assume it did not influence our measurements.*

*The first comparison of our data to that of an earlier study (van Dijk & Manley, 2009) that shared its mathematical model and measurement procedures with this one. The study in question found that a strong intra subject relation existed between the width of an SOAE and its amplitude.*

*This study used a modification of ear canal pressure instead of broad-band noise. Our results, however, were quite similar. Additionally the parameters found for this relation held quite strongly in our data as well even though our stimulus was quite different.*

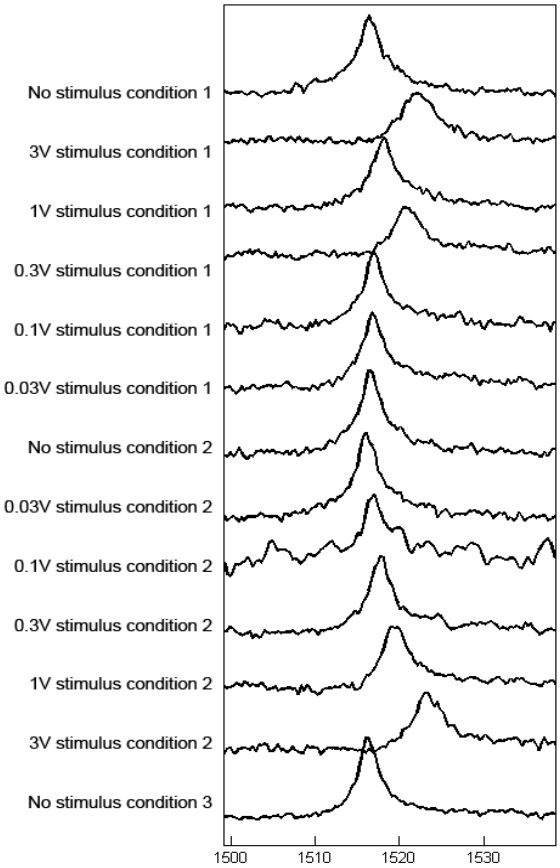


*Fig. 3 An example of the shift o a single under different stimulus conditions. Spectra are offsetted horizontally in order to increase comparability.*

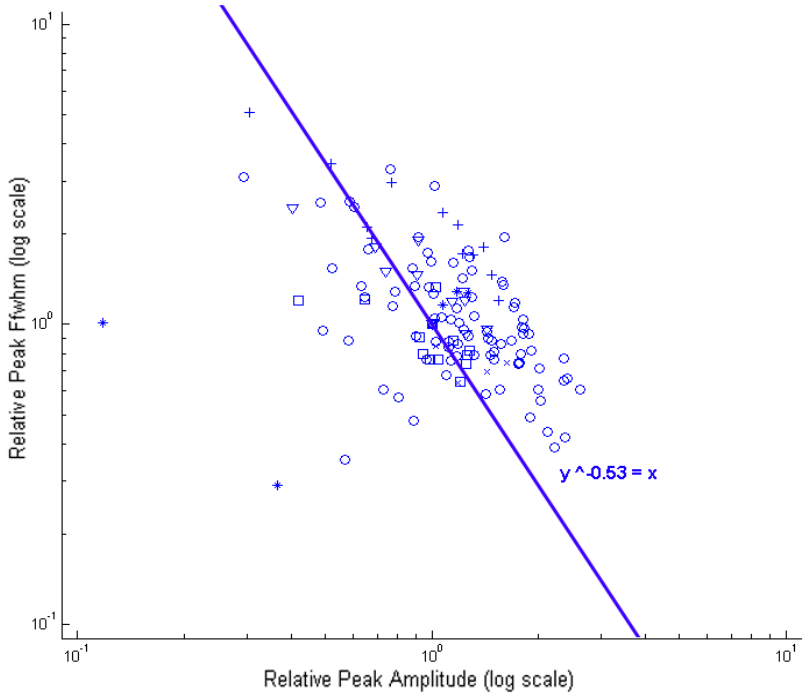


*Fig. 4 The relation between width and height shown in a scatter plot. All peak where normalised to allow comparison for within subject data. Different symbols for data points correspond to different test subjects.*

*To test our main hypothesis we also correlated the relative peak width (relative compared to the average of all three 0V conditions) with the stimulus strength in dB and found a very strong correlation.*

*A correlation over all data produced:*
*Rho = 0.2953, ( p = 0.36 * 10^-4)*
*However, this statistic is biased since we have more fits of lower widths and stimulus strength increasing variance over both variables while increasing the number of examples.*
*If we correlated only over the complete data (peaks of which fits where made under all conditions) we got:*
*rho = 0.5344, (p = 6.09 * 10^-9)*
*The relation is nevertheless far from linear even if the sound level is expressed in dB.*

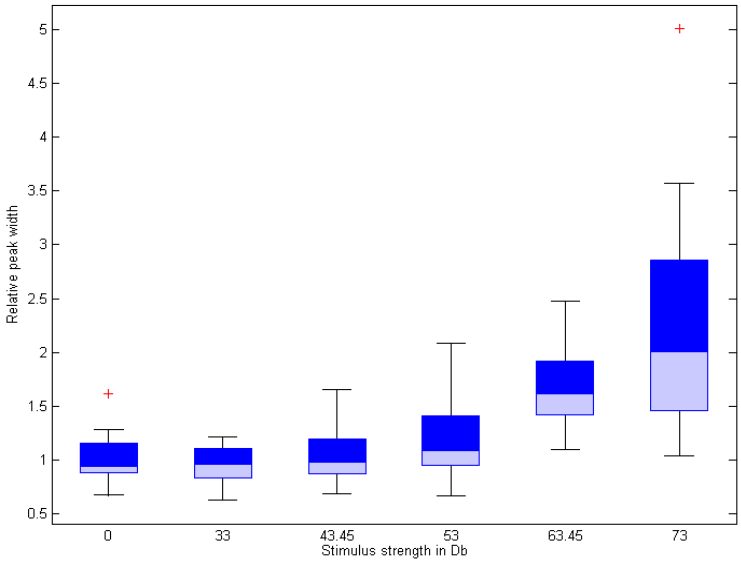


*Fig 5. A box plot of the unbiased data. Clearly shown is the non linear relation between Stimulus strength and peak width.*

# 5. Discussion

*We examined the relation between stimulus strength and the increase in peak width caused by contralateral suppression and a found significant correlation. Our data is also in line with the findings of an earlier experiment with the same measurement setup but a different kind of stimulus.*
*The (contralateral) suppression of SOAEs has been examined under a host of different conditions, including, but not limited to:*

- *body temperature*
- *ear canal pressure*
- *many variations of auditory stimuli*
- *subject orientation*

*In the greater majority of all cases the response of the peak is the same: amplitude decreases as with and a frequency increases. In some rare cases the peak frequency decreases instead of increases.*
*There are many models that explain parts of SOAE's and how they respond external influences. However, no model exists that encompasses the entire field of available data. Most models either try to explain what causes SOAE's to arise in the first case. Or they try to explain a limited scale of the reactions that SOAEs show in response to stimuli.*
*There are at least three generally different models that try to explain what SOAEs really are (Shera, 2003):*

*The point-source model (PS-model)*
*This model dates back to the work of Gold. It supposes that SOAEs arise through the local, autonomous oscillation of some cellular constituent of the organ of Corti.*
*The passive standing-wave model (PSW-model)*
*This model assumes SOAEs are biological noise, passively amplified by cochlear standing-wave resonances acting as nonlinear narrow-band filters.*
*The active standing-wave model (ASW-model)*
*SOAEs are standing-wave amplitudes are maintained by coherent wave amplification within the cochlea.*

*Each of these models is mutually exclusive at first glance. However, in closer analysis we find that there are many overlaps between them. Additionally it is apparent that the mechanisms that cause SOAEs in mammals are quite different from those in reptiles, bird or other species. Therefore, not all experiments generalize to the whole of SOAE research.*

## 5.1. The PS-model

*The PS-model has many problems in its original form. It does not account for collateral suppression, it demands that a very small part of the organ of Corti delivers a rather great amount of acoustic energy and it does not explain why the frequency shifts of a SOAE is upward in most cases.*
*But even updated versions of the point source model that include the use of the Medial Olival Cochlear cells efferents to Outer Hair Cells (OHC) (Veuillet et al., 1991) lack in explaining power compared to the other two models. They do give a very good explanation for the shape of most SOAEs: a single source can be assumed to be oscillator that varies in frequency and amplitude resulting in the measured Lorentzian shaped peaks.*

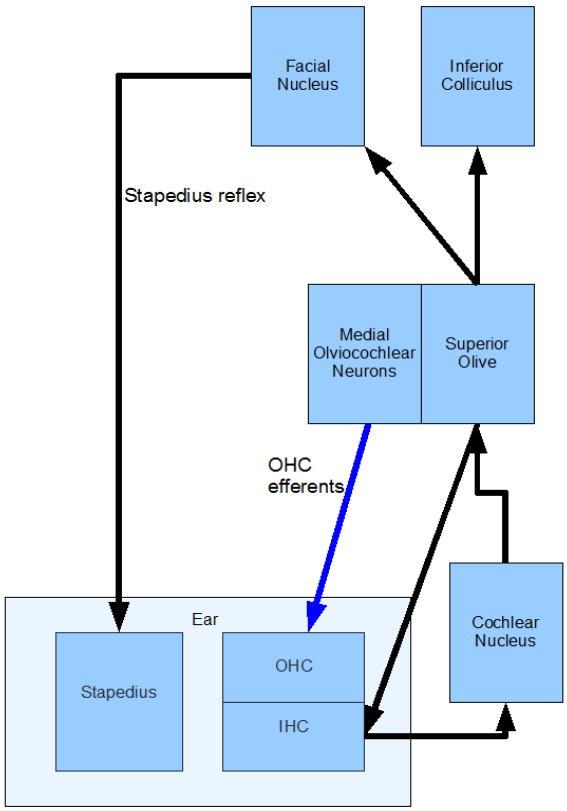


*Fig. 6 A simplified representation of the different routs by which stapedius reflex and OCH are controlled*

### 5.2. The standing wave models

*PSW-model and ASW-model are quite similar in many ways. Both assume that forward traveling waves are reflected of off? randomly distributed perturbations in the mechanics of the cochlea. The reflection results in the forming of standing waves in the cochlea that are in some cases measurable in the ear canal as SOAE's. However when all this is powered by random noise and amplification there is no way to explain why recordings of oto-acoustic signals are not littered with all kinds of standing wave signals.*

*This is where the models start to diverge.*

*The PSW-model explains the frequencies specificity by introducing a filter function that filters all standing wave components except for certain narrow band frequencies.*

*The ASW-model puts that only coherent waves are amplified and that therefore only certain waves receive enough amplification on a round trip to sustain themselves.*

*While in an ordinary spectral representation these would look the same, there are some key predictions that contrast the PSW-model and ASW-model. Using and extended version of the Bialek Wit analysis (Bialek & Wit, 1984) It has been shown that some of the deeper statistical properties of SOAE's do not agree with those predicted by the PSW-model.*

*(Shera, 2003)*

*Even though the ASW-model concurs most with the measured data, it is not conclusive as to what really causes contralateral suppression. There is in fact a multitude of ideas as to how exactly contralateral suppression takes place. Most of these can be combined with more than one of the models that describe SOAEs themselves.*

*Initially it was thought that contralateral suppression was entirely by the stapedius reflex (REF) it was a logical assumption, as at that time there was no knowledge of other ipsi/contra-lataral mechanics in the ear. However, since the discovery of OHC efferents this has become ever more unlikely.*

*For instance it has been found that the SOAEs respond most to frequencies that are slightly lower than their own. (Mott et al. 1988) Because the stapedius reflex is rather unspecific for frequency, and certainly not to?the extent shown in contralateral suppression this makes the idea that the stapedius reflex is the main cause of contralateral suppressionn inconclusive at best.*

*But as put mentioned? Stated? earlier, none of these models are truly mutually exclusive. For standing waves it has been suggested that the slight change in impedance that could result from partial contraction of the stapedius muscle that would not be detected by conventional impedance measurements. (Berlin et al, 1993)*

*And indeed models using this prediction can give some predictions as to how big a relative shift will be*

*However, a test with patients with severed or paralyzed stapedius reflexes has shown that these people still exhibit completely normal contralateral suppression. (Veuillet et al. 1991)*

*There is also the possibility of transcranial transmission. Sound conducted by the cranial bones could locally influence SOAE generators or indirectly affect impedances in the inner ear. Cranial bones only conduct lower frequencies properly, which would work in case of our broad band noise stimulus but not in case of higher pure tone stimulus such as used by Mott (Mott et al. 1988)*

*Then there are the OHC efferents. Active control of the OHCs can either decrease or increase local impedance? or decrease or increase amplification of some or all frequency bands. OHCs could even perform some intrinsic oscillation as implied by the point source model. This oscillation could then in turn be modulated by the OHC efferents to cause contralateral suppression.*

*There is as of yet only highly circumstantial evidence as to with of these functions the OCHs perform. What is known is that the path that innervates OCHs is rather different than the one that controls the stapedius reflex(see Figure 4 for more details). An extensive coverage of the anatomy surrounding the OHC efferents can be found in the Guinan review of the subject (Guinan, 2006).*

*Our model mostly avoids these discussions by providing a purely mathematical framework within which we examine empiric data. It does however make one important assumption: SOAE's are caused by a single second order oscillator which varies in frequency and amplitude. This oscillator is referred to as the SOAE generator.*

*It is interesting that we find the same relation with different kinds of stimuli because this makes it likely that they influence the SOAE generator in the same fashion.*

*In our model, the width increase is explained by a growing instability in the SOAE generator. As the amplitude of a SOAE decreases under the influence of*

*some yet to determine suppressing factor the background noise begins to have more influence on the frequency of the SOAE generator. This in turn increases its frequency variance making the peak wider and decreases the height even further.*

### 5.3. Summary

*Our findings were consistent with earlier studies that used a different type of stimulus. This concurs with the idea that contralateral suppression is the result of processes inside the cochlea where in all likelihood SOAE generators are located. It is also apparent that there is a non linear relation between the strength of the stimulus and the width increase of SOAE peaks, something that cannot be performed by a simple passive filter sutch as the stapedius. It is however in no way clear how exactly contralateral stimulation works. The stapedius reflex and transcranial transmission seem ever more unlikely to be important factors but still cannot be completely excluded. Especially since the impedance mismatch at the cochlear boundary with the middle ear has a crucial role in both standing-wave models.*

*Of all the models that try to explain the nature of SOAEs the actively amplified standing-wave model seem to agree best with modern findings.*

*It still offers no real answer to the how and why of contralateral suppression. Even if it can predict how SOAEs will change under influence of a middle-ear impedance change, it has been shown that contralateral suppression also takes place when the impedance changes are impossible. Recent advances in neuroscience have shown that the controlling mechanisms of OHCs are far more complex than previously believed and indeed some answers might be found in that direction.*

*The research of spontaneous oto-acoustic emissions stands on the limits of what is known about the human hearing mechanism. There are extensive models of the middle ear cochlea that are incorporated in all kinds of smart sensory or sensory modeling systems.(Slaney & Lyon, 1993) But still very little is known about the early integration of auditory information which occurs in the very systems that are probably causing SOAEs as well. A greater understanding of this mechanism and its limitations allows us to further improve these systems and better understand the limits of human perception.*

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## 7. Appendix: Model

*Our model assumes that that every SOAE is generated by a single oscillate that has a distinct variance in frequency and amplitude. Generalized over time this can be seen Van Der Pol oscillator of the form:*

$$\ddot{x}+u(x^2-1)\dot{x}+x=0$$

*If we assume*

$$v=\dot{x}$$

*Then this can be formulated as*

$$\begin{bmatrix}\dot{x}\\ \dot{v}\end{bmatrix}=\begin{bmatrix}0 & 1\\ -1 & -\mu(x^2-1)\end{bmatrix}*\begin{bmatrix}x\\ v\end{bmatrix}$$

*Adding noise terms to a system such as this and setting initial values allows us to form a solution to this dynamic system of the form:*

$$x=(A_0+A_{noise})\cos(\omega_0 t+\omega_{noise}t+\psi_0)$$

*Note that both noise terms are not constants but rather functions of the time in their own right. It is known that the spectra of systems such as these are distributed according to the Chaucy or Lorenzian distribution:*

$$f(x;x_0,\gamma)=\frac{1}{\pi}\left(\frac{1}{(x-x_0)^2+\gamma^2}\right)$$

*This is of course a normalized form probability distribution, substituting $\gamma$ for Ffwhm/2 , all the x's for frequencies and multiplying the whole with B*$\pi$ yields:*

$$h(f;B,F_{mid},F_{Fwhm})=\frac{B}{(f-F_{mid})^2+\frac{F_{Fwhm}^2}{4}}$$

*Which is the basic curve we use to fit our data*
*If we amply the same transformation to its cdf:*

$$F(x;x_0,\gamma)=\frac{1}{\pi}\arctan\left(\frac{x-x_0}{\gamma}\right)+\frac{1}{2}$$

*And integrate over all f we receive surface under the entire curve:*

$$\frac{B}{F_{fwhm}}2\pi$$

*This is also the amplitude of the original oscillator if no noise existed; hence this expression is often referred to as the amplitude of the SOAE.*

*If we add a linear and a direct current noise term we receive our final fitting formula:*

$$A=\frac{B}{(f-F_{mid})^2+\frac{F_{fwhm}^2}{4}}+N_{dc}+N_l*f$$

*This model can explain the widening of the peak as the amplitude decreases and the shape of the peak.*
*If the noise component increases so does the variance of the frequency of the oscillator because it keeps being pushed out of its stable limit cycle. If the amplitude of the oscillator increases so does the influence of the dampening constant μ. Therefore peaks with greater amplitudes are more stable and therefore have a smaller frequency.*

*However this model cannot explain the upward shift in frequency that occurs under the influence of contralateral stimulation. Perhaps this can be remedied by adding an extra term to the model. But there is no reliable mathematical model of the frequency shift with which to do so. Also we must be careful not to modify the distribution shape that SOAE follow so well.*