Research Article

Aftereffects of Intense Low-Frequency Sound on Spontaneous Otoacoustic Emissions: Effect of Frequency and Level

LENA JEANSON,¹ LUTZ WIEGREBE,¹ ROBERT GÜRKOV,^{2,3} EIKE KRAUSE,^{2,3} AND MARKUS DREXL^{2,3}

¹*Division of Neurobiology, Department Biology II, University of Munich, 82152, Martinsried, Germany*

²*German Centre for Vertigo and Balance Disorders (IFB), Grosshadern Medical Centre, University of Munich, 81377, Munich, Germany*

³*Department of Otorhinolaryngology, Head and Neck Surgery, Grosshadern Medical Centre, University of Munich, 81377, Munich, Germany*

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ABSTRACT

The presentation of intense, low-frequency (LF) sound to the human ear can cause very slow, sinusoidal oscillations of cochlear sensitivity after LF sound offset, coined the “Bounce” phenomenon. Changes in level and frequency of spontaneous otoacoustic emissions (SOAEs) are a sensitive measure of the Bounce. Here, we investigated the effect of LF sound level and frequency on the Bounce. Specifically, the level of SOAEs was tracked for minutes before and after a 90-s LF sound exposure. Trials were carried out with several LF sound levels (93 to 108 dB SPL corresponding to 47 to 75 phons at a fixed frequency of 30 Hz) and different LF sound frequencies (30, 60, 120, 240 and 480 Hz at a fixed loudness level of 80 phons). At an LF sound frequency of 30 Hz, a minimal sound level of 102 dB SPL (64 phons) was sufficient to elicit a significant Bounce. In some subjects, however, 93 dB SPL (47 phons), the lowest level used, was sufficient to elicit the Bounce phenomenon and actual thresholds could have been even lower. Measurements with different LF sound frequencies showed a mild reduction of the Bounce phenomenon with increasing LF sound frequency. This indicates that the strength of the Bounce not only is a simple function of the spectral separation between SOAE and LF sound frequency but also depends on absolute LF sound frequency,

possibly related to the magnitude of the AC component of the outer hair cell receptor potential.

Keywords: low-frequency sound, cochlea, bounce phenomenon, outer hair cells

INTRODUCTION

It has long been known that the activity of the cochlea temporarily changes after the presentation of intense, low-frequency (LF) sound, a process for which the term “Bounce” phenomenon has been coined. Typically, measures of cochlear activity slowly cycle (over a time course of a few minutes) between states of hyperactivity and hypoactivity. In early experiments, Hirsh and Ward (1952) and Hughes (1954) have shown that pure tone hearing thresholds in human subjects slowly oscillate for several minutes after LF sound offset. The phenomenon is not limited to psychoacoustic measures: Kemp (1986) demonstrated that evoked otoacoustic emissions (OAEs) in humans show similar oscillations in emission level after intense LF exposure. Animal experiments (Kirk and Patuzzi 1997) revealed that LF exposure has a different effect on cubic and quadratic distortion product otoacoustic emissions (DPOAEs), which was later also confirmed in humans (Drexel et al. 2014). In TTX-treated animals, the phenomenon persists despite paralysis of the auditory nerve and the middle ear muscles (Kirk and Patuzzi 1997), indicating that it is entirely of cochlear origin.

Correspondence to: Markus Drexel · Department of Otorhinolaryngology, Head and Neck Surgery, Grosshadern Medical Centre · University of Munich · 81377, Munich, Germany. Telephone: +49 (0)89/2180 74340; email: markus.drexel@med.uni-muenchen.de

Spontaneous otoacoustic emissions (SOAEs) have been shown to be a sensitive detector of the phenomenon (Kemp 1986; Kugler et al. 2014, 2015) and undergo slow, cyclic changes of level and frequency with time courses similar to hearing threshold changes or alterations of evoked OAEs. “New” SOAEs, presumably hidden in the noise floor, and only appearing for a short period of time after stimulation with a LF sound, were also reported (Kugler et al. 2014). A clear, inverse correlation exists between the strength of the Bounce and the SOAE frequency (Kugler et al. 2014), indicating that LF sound is more effective in inducing the Bounce as SOAE frequency decreases.

While the Bounce is not necessarily limited to an exposure to very low frequencies (Kemp and Brill 2009), sounds below 2 kHz seem to be most effective in inducing the Bounce phenomenon. The slow, oscillatory nature of the Bounce suggests that intense LF sound challenges the homeostasis of a presumably biochemical process controlling cochlear activity. This process was successfully modelled by Kemp and Brill (2009) with a quasi non-linear feedback system acting locally on basilar membrane areas. Patuzzi (2011) suggested that the Bounce is due to oscillations in outer hair cell (OHC) Ca^{2+} levels as a result of the large AC component of the OHC receptor potential caused by low-frequency sound, which is less attenuated by OHC membrane filter properties. Ca^{2+} controls the slow motility of OHCs (Dulon and Schacht 1992; Dulon et al. 1990) and, as a consequence, cochlear amplifier activity. According to Patuzzi (2011), very LF sound should be the most effective in eliciting the Bounce as the AC component of OHC receptor potential is large, due to the low-pass filter properties of the OHC membrane. It has been previously shown that SOAEs are a good measure of the Bounce, as they show distinct level and frequency changes after presentation of a 30 Hz, 120 dB sound pressure level (SPL) stimulus (Kugler et al. 2014, 2015). The Bounce threshold and its frequency dependence, however, are not known.

Here, we explore the effect of the frequency and level of the LF stimulus on the Bounce, quantified in terms of SOAE level changes. The aim of this paper is twofold and addresses the following topics: First, a well-defined parametric space for evoking the Bounce is needed to assess the possibility that environmental sounds suffice to induce the Bounce. Second, the nature of the dependence of the Bounce on the frequency of the inducing stimulus is key for a comprehensive understanding of the mechanisms underlying the phenomenon.

METHODS

Subjects

Thirty-nine human subjects were screened for this study. Data from 20 subjects were analyzed for the effect of LF sound level on the Bounce (16 females and 4 males; aged between 19 and 30 years, mean age 23 years); data from 19 subjects were analyzed for the effect of LF sound frequency (15 females and 4 males; aged between 19 and 27 years, mean age 22 years). Fourteen subjects participated in both parts of the study. Every subject underwent a pure tone hearing test to check that their hearing threshold was normal. The subjects were then checked for bouncing SOAEs after LF stimulation (120 dB SPL, 30 Hz, 1.5 minutes). Fourteen subjects were excluded from the study because no bouncing SOAEs could be recorded or because their hearing thresholds were elevated. All experiments took place in a sound-attenuated booth and the subjects were seated in a comfortable recliner. The subjects were advised to remain still and quiet during the measurements. Every subject participated in two sessions: in the first session, it was checked whether the participants met the inclusion criteria and the effect of LF sound level was measured. In a second session (taking place at least 2 weeks later), the effect of LF sound frequency was measured. All experiments were approved by the ethics committee of the University Hospital of the Ludwig-Maximilians-University Munich, Germany, in agreement with the Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans, and all subjects gave their written informed consent. As a precaution for potential acoustic overstimulation, a maximum stimulus level of 80 dB (A) was employed in all experiments, and it was ensured that the sound exposure intensity and duration was within the current daily limits for normal working environments in Germany.

Procedures, Signal Generation and Data Acquisition

Measurement of the Hearing Threshold. All participants underwent an automated pure tone hearing test (Automatic Pure Tone Audiometry V2.28, HörTech, Oldenburg, Germany) with test frequencies ranging from 125 Hz to 16 kHz. The resulting hearing threshold was compared to a mean threshold curve (ISO 389-8:2004 (E), ISO/TR 389-5:1998 (E)). Subjects with hearing thresholds of more than 20 dB HL at any tested frequency were not included in the study.

SOAE screening. Two-minute recordings without any acoustic stimulation in the ear canal of both ears of each subject were carried out and checked for the presence of SOAEs (Fig. 1, first measurement). The

ear showing the most prominent SOAEs was chosen for further experimentation. Subjects were presented with an LF sound (30 Hz sine wave, 120 dB SPL) for 1.5 min, directly followed by 4 min of recording the SOAEs without any acoustic stimulation (Fig. 1, second measurement). A subject took part in the following experiments if Bounce-related SOAE level changes were detected in at least one SOAE.

LF Sound Level and Frequency Dependence. A trial consisted of a prestimulus period of 1 min of SOAE recording followed by 1.5 min of LF sound presentation and a poststimulus period of 7 min of recording (9.5 min in total; Fig. 1, third measurement). To assess the LF sound level dependence of the Bounce in SOAEs, a sequence of six trials where the LF sound was the same in frequency and duration, but changed in sound level (93 to 108 dB SPL, 3 dB step size, random order), was carried out. Data obtained with a sound level of 120 dB SPL during the screening procedure was also included in the analysis. A similar sequence of five trials was conducted where the LF sound was kept at a constant loudness level and changed in frequency (30 to 480 Hz, one-octave steps, 80 phons, corresponding to 110, 99, 90, 84 and 81 dB SPL at the octave frequencies, random order). It has been shown that a correlation exists between the area under the basilar membrane excitation pattern and the loudness level, resulting in the assumption that constant loudness levels cause excitation patterns with constant areas (Chen et al. 2011). Strictly speaking, the LF sound is not low-frequency anymore with frequencies higher than 200 Hz (e.g., Moller and Pedersen 2011), but, for the sake of clarity, the nomenclature is kept throughout the manuscript.

Signal Generation and Data Acquisition. Overall, signal generation and data acquisition is very similar to previous studies from our lab (Kugler et al. 2014, 2015). In short, SOAE measurements were recorded

with an ER-10C DPOAE probe system (Etymotic Research Inc., Elk Grove Village, IL, USA). The LF sound was generated by a separate loudspeaker (NSW1-205-8A, AuraSound, Santa Ana, CA, USA) driven by a RB-971 power amplifier (Rotel, Tokyo, Japan). The loudspeaker was connected to a 140-cm long polyethylene tube (inner diameter 1 mm), the tip of which was fed through the ER10C-14A foam ear-tip of the DPOAE probe so that it faced the tympanic membrane.

An RME Fireface UC audio interface (RME, Audio AG, Haimhausen, Germany) operated at a sampling rate of 44.1 kHz was used for signal generation and data acquisition. For controlling the experiments, custom-written scripts in Matlab R2010b (Mathworks, Natick, MA, USA) and the SoundMexPro sound application (HörTech, Oldenburg, Germany) were employed to use low-latency, multichannel ASIO interfacing in Matlab.

To exclude potential artefacts exceeding the noise floor, the entire recording pathway was checked with an artificial ear (Ear Simulator Type 4157, Brüel & Kjær Sound & Vibration Measurement A/S, Naerum, Denmark). To ensure that the LF stimuli presented in the LF sound frequency dependence measurements corresponded to the 80-phon equal-loudness contour (ISO 226:2003), the amplitude response of the ER10C probe microphone was compared to the amplitude response of the measuring microphone of the artificial ear using a measuring amplifier (type 2610, Brüel & Kjær Sound & Vibration Measurement A/S, Naerum, Denmark) and corrected accordingly, and the stimulus levels were then calibrated in situ and corrected accordingly. At the beginning of every trial, the probe fit was checked in situ with a calibration procedure employing the in-built loudspeakers of the OAE probe to detect acoustic leaks.

FIG. 1. Procedures for the different recordings.

Data Analysis

Details of the SOAE analysis procedures can be found in Kugler et al. (2014); Kugler et al. (2015). In short, the time-domain SOAE signals were divided into consecutive segments of 5-s duration. Five-second windows were further analysed with spectral averaging using Welch's averaged, modified periodogram method employing MatLab `pwelch` function. SOAEs with a signal-to-noise ratio of less than 5.39 (Dobie and Wilson 1996) were excluded. SOAEs with a frequency between 0.5 and 6 kHz were analysed, as SOAEs with very high frequencies do not show the Bounce (Kugler et al. 2014), and the lower-frequency range is limited by the increasing noise floor of the recording system. Transient SOAEs, which appeared after the LF sound for a short period of time before vanishing into the noise floor again (see Kugler et al. 2014), were not included and only permanent (i.e., lasting for the entire recording period) SOAEs were analysed. SOAEs were then tracked throughout the whole recording (8 min; 1-min prestimulus plus 7-min poststimulus period). Data recorded during the LF sound period were not analysed, as the intense LF sound tends to suppress the SOAEs to levels below the noise floor (Bian and Watts 2008). Since small frequency changes of SOAEs occur after LF sound offset (Kemp 1986; Kugler et al. 2014), SOAEs occurring in a frequency window of ± 15 Hz during the entire recording period were considered as variations of one SOAE.

A change detection analysis (Kugler et al. 2014) was carried out to find significant changes in the SOAE level time series. In short, the mean of the level traces was subtracted from the individual data points and then summed up cumulatively. The difference (delta) between the maximum and the minimum of the resulting cumulative sum was then calculated. The SOAE traces were then randomly reordered (10,000 times) using a bootstrap procedure, the aforementioned procedure repeated, and the percentage of the deltas of the bootstrapped data exceeding the delta of the original time series was calculated. A change in the time series was considered significant if less than 5 % of the bootstrapped time series showed a delta larger than in the original time series. The lowest LF sound level reaching the significance criterion was considered the threshold. All analyses, statistics and visualisations were carried out with scripts written in Matlab R2010b (Mathworks, Natick, MA, USA).

RESULTS

SOAEs were recorded in 25 ears from 25 subjects. One hundred one SOAEs from 20 ears were used for

LF sound level dependence measurements, and for 76/101 SOAEs, LF level thresholds could be identified. One hundred twenty-one SOAEs from 19 ears were used for LF sound frequency dependence measurements, and those between 93 and 105 SOAEs (depending on LF sound frequency) were further analysed.

After the offset of the 30-Hz LF sound (120 dB SPL, 1.5 min), SOAE levels show a typical, non-monotonic trace and slowly cycle through phases of sensitisation and desensitisation, as reported in earlier publications from this group (Kugler et al. 2014, 2015). The cycle consists of a SOAE level maximum, followed by a level minimum before returning to baseline values. The level maximum is normally reached about 1 min after LF sound offset (Fig. 2 (1)) before the SOAE level reaches a minimum about 2.5 min after stimulus offset (Fig. 2 (2)). Occasionally, SOAE levels decreased to levels below the noise floor for a certain time period. Typically, after around 4 min, the SOAE level returned to baseline values (Fig. 2 (3)). Additionally, parallel changes in SOAE frequency occur. In the following, only SOAE level changes were analysed as they are more stable, and more pronounced, than are frequency changes.

LF Sound Level Dependence of the Bounce

The level changes of the tested SOAEs after LF sound exposure was positively correlated with the LF sound levels: the higher the LF sound level, the more pronounced was the Bounce. Representative examples of complete Bounce traces of SOAE level are displayed in Figure 3.

In all 76 SOAEs where a LF sound threshold could be determined, the maximum and minimum SOAE levels during the Bounce were analysed as a function of LF sound level. With increasing LF sound level, the

FIG. 2. Typical frequency and level trace of a bouncing SOAE after the offset of an LF sound. The SOAE level and frequency reached a maximum (1), a minimum (2) and returned to base line values (3).

FIG. 3. Representative examples of SOAE level traces after LF sound offset (30 Hz sine wave, 1.5 min) depending on LF sound level (dB SPL, colour-coded, see legend). SOAE frequency displayed for each subject. Note that the traces are very similar between subjects, i.e. SOAE level maxima and minima can be found at approximately the same time after LF sound offset. The Bounce magnitude was

positively correlated with LF sound level but can vary greatly between individuals. *Gaps* in the traces indicate rejected data points due to insufficient signal-to-noise ratios. SOAEs after presentation of a sound level of 120 dB SPL were tracked for 4 min.

median of SOAE level maxima increased, and, to a slightly lesser extent, the minima decreased (Fig. 4a). The maxima and minima of SOAE level changes were strongly correlated (Pearson's linear correlation coefficient, $R^2 = 0.946$, $p < .001$ and $R^2 = 0.689$, $p = .0208$, respectively) with the LF sound level.

The lowest LF sound level where a significant SOAE level change could be detected (i.e. the Bounce threshold, see the "Methods" section) varied across subjects and also across SOAEs within the same subject. The median Bounce threshold for all SOAE level changes was 102 dB SPL (first quartile 96 dB SPL, third quartile 105 dB SPL). The distribution of thresholds is shown in Figure 4b. Due to our LF sound level constraints, the lowest threshold was detected at 93 dB SPL and the highest at 120 dB SPL. Higher and lower thresholds are of course possible and would not

have been detected with the limited set of stimulus parameters in this study, but in an attempt to avoid cumulative overexposure of the participants, the number of level steps were kept to a minimum. In nine subjects, at least one SOAE bounced at the lowest LF sound level used (93 dB SPL).

LF Sound Frequency Dependence of the Bounce

At each LF sound frequency, the SOAE level maxima and minima appeared at similar points in time (at about 1 and 2.5 min after LF offset, respectively; see Figure 5 for representative examples). While different LF sound frequencies did not change the timing of the Bounce, the effect of the LF sound frequency on the Bounce was quite varied across individuals (Fig. 5). The trials

FIG. 4. **a** Median of 76 bouncing SOAEs level maxima (blue) and minima (red) and third and first quartiles (black lines), respectively, as a function of LF sound level. All data are expressed relative to the corresponding SOAE preexposure levels

(mean of 1 min pre-LF recordings). **b** Distribution of LF sound levels sufficient to induce the Bounce phenomenon (= threshold), derived from the data shown in **a**.

FIG. 5. Representative examples of SOAE traces after stimulation with an LF sound (80 phons, 1.5 min) at various frequencies (between 30 and 480 Hz) from five different subjects. The frequency of the LF sound (in Hz) is colour-coded, see legend below top panels. SOAE frequencies are shown for individual subjects.

with low LF sound frequencies caused a clear Bounce with distinct phases of sensitisation and desensitisation, but, in most cases, the bounce became less pronounced at higher LF sound frequencies (Fig. 5). In particular, SOAE level minima became less pronounced with increasing LF sound frequency while SOAE level maxima were less affected by an increasing LF sound frequency. Occasionally, this resulted in an almost monophasic Bounce at the highest tested LF sound frequency (480 Hz, Fig. 5). A quantitative analysis of all SOAE level changes (expressed as the SOAE level peak-to-trough changes) revealed that with increasing SOAE frequency, the SOAE level maximum-to-minimum changes decreased (Fig. 6), in accordance with the literature (Kugler et al. 2014), regardless of the LF sound frequencies. The Bounce phenomenon can be elicited with all frequencies used in this study (30–480 Hz). The effect strength, however, decreased with increasing SOAE frequency, up to 480 Hz, where the effect strength increased again (Fig. 6f). Significant changes relative to SOAE recordings with preexposure to 30 Hz sounds (paired *t* test, adjusted for multiple comparisons) were seen at 120 and 240 Hz (Fig. 6c, d).

DISCUSSION

Here, we have shown that the Bounce, quantified in terms of SOAE level changes after LF sound offset, can be induced with a 30-Hz, 1.5-min LF sound exposure at levels as low as 93 dB SPL, with a median of 102 dB SPL. For the induction of bouncing SOAEs, very low frequency sound is not a necessary condition: 480-Hz sounds (the highest frequency tested in this study) with a loudness level of 80 phons, corresponding to 81 dB SPL, are sufficient to induce the Bounce. Kemp and Brill (2009) also concluded that for the Bounce, as assessed with transiently evoked OAEs, very low-frequency sound was not a necessary condition, but OAE frequencies needed to be higher than the Bounce-inducing low-frequency sound, at least at the quite intense stimulus level of 105 dB (A) used in that study. It can be assumed that sounds with parameters similar to the present study exist in our environment (e.g. inside vehicles: Berglund et al. 1996) and that, consequently, ears occasionally bounce when exposed to sufficiently loud and low-frequency sounds in our environment. It is unclear whether this reaction is part of the normal cochlear repertoire and hence harmless, or whether it is a very first indication of cochlear impair-

FIG. 6. **a–e** Scatter plot of SOAE level changes (maximum–minimum, *circles*) as a function of SOAE frequency for different LF sound frequencies given in the title of each panel. *Data points* in the scatter plot represent individual SOAE level changes. Histograms show the distribution of SOAE level changes and of bouncing SOAE frequencies. The Bounce can be elicited with all LF frequencies used in this study (30–480 Hz). Significant differences in SOAE level changes can be seen between LF frequencies of 30 Hz and 120 and

240 Hz, respectively (paired, two-sided *t* test, between the rel. SOAE level changes at 30 Hz and 60, 120, 240 and 480 Hz, respectively, *p* values adjusted for multiple comparisons with the Bonferroni correction). **f** Mean of SOAE level changes shown in **a–e** as a function of LF sound frequencies. Note the monotonic decline with increasing LF sound frequency, followed by a slight increase at 480 Hz.

ment (Kugler et al. 2014). It is not within the scope of this paper to resolve this matter. The results reported here are, however, suited to shed more light on the possible biophysical and biochemical mechanisms of the Bounce.

Intuitively, if the Bounce is a localised, and not a pan-cochlear, phenomenon, one would assume that SOAEs are affected the most when the SOAE characteristic frequency site and the site of maximal LF

sound excitation are closest, i.e. when the frequency difference between the SOAE frequency and the LF sound frequency is minimal. This is indeed reflected in the fact that SOAEs in the lower frequency range are most affected and show overall the largest Bounce (Kugler et al. 2014, this study). A similar relation between SOAE suppression or biasing tone frequency on the one hand and SOAE frequency on the other hand was found (Bian 2008; Bian and Watts 2008;

Frick and Matthies 1988; Manley and van Dijk 2016). In these studies, a minimal difference between SOAE suppression or biasing tone frequency and SOAE frequency resulted in the largest suppression or biasing, respectively. Following this line of reasoning, SOAEs in this study, which were always higher than the highest tested LF frequency, should be more affected as the LF frequency increases, and thus the spectral excitation pattern of the LF sound approaches the SOAE characteristic frequency site. Our data show that this is not the case, and the Bounce remained almost constant or even decreased as the LF sound approached the SOAE frequency. This supports the hypothesis that particular properties of OHCs, with very low time constants, only manifest themselves at low stimulation frequencies and play a major role for the Bounce. Patuzzi (2011) suggested that large AC components of OHC receptor potentials (which occur only at relatively low stimulus frequencies due to the low-pass filter properties of the OHC lateral wall membrane) are pivotal to the aftereffects of intense low-frequency sound. Cody and Russell (1987) indeed showed that, at frequencies lower than about 2 kHz, the DC component of the OHC receptor potential is relatively small, compared to the AC component, and, at high intensities, depolarising. A sufficiently large OHC receptor potential can increase the average opening probability of L-type voltage-gated Ca^{2+} channels (with asymmetric activation profiles) in OHCs (Rodríguez-Contreras and Yamoah 2003) and, as a consequence, cause a net influx of Ca^{2+} . Due to Ca^{2+} -induced Ca^{2+} -uptake and release mechanisms with differing time constants, OHC Ca^{2+} levels can become unstable and start to slowly oscillate, which will be translated into slow OHC motility (Patuzzi 2011). Slow OHC motility, by changing stereocilia angle, can control the opening probability of OHC mechanoelectrical transduction channels and thus the cochlear operating point and gain (Patuzzi 2011). A mechanism solely based on OHC AC receptor potentials should result in a decreasing Bounce magnitude as the absolute frequency of the LF sound increases and thus the AC component of the OHC receptor potential decreases. We have not consistently observed this, possibly for the following reasons: first, the highest frequency for the bounce-inducing stimulus we employed was 480 Hz, which is well below the 2-kHz cut-off frequency of the OHC membrane low-pass filter, and it may be that frequencies higher than 2 kHz result in a significantly smaller or even absent Bounce. Second, the loss in Bounce magnitude with increasing LF sound frequency might to some extent be compensated for by the LF sound excitation pattern approaching the SOAE characteristic frequency site. The Bounce magnitude at changing LF sound frequencies might thus be the

result of two competing mechanisms: increasing the LF sound frequency diminishes the “cellular” component, related to the OHC receptor potential, but enhances the spectral component, related to the overlap between the LF sound excitation pattern and the SOAE characteristic frequency site.

SOAEs themselves are a fragile phenomenon and can be easily modified; therefore, alternative explanations for the cause of aftereffects of relatively intense sounds on SOAEs are conceivable (Kugler et al. 2014). A distinguishing feature, however, of the Bounce as it relates to SOAE changes, are level and frequency changes in the *same* direction, i.e. SOAE frequency increases with SOAE level and vice versa (Kugler et al. 2014), whereas other manipulations such as middle ear pressure changes (van Dijk et al. 2011), head position changes (de Kleine et al. 2000) or activation of the medial olivocochlear reflex (Zhao and Dhar 2010) cause SOAE level and frequency changes with *opposite* signs, i.e. SOAE frequency decreases while SOAE level increases and vice versa. There is little doubt that the Bounce reflects changes of inner ear properties, and it has been shown that activation of the medial olivocochlear system during the Bounce-inducing stimulus results in a mild reduction of Bounce magnitude (Kugler et al. 2015), indicating that the Bounce indeed reflects changes of OHC properties.

Taken together, the Bounce appears to be a byproduct of the slow recovery of OHCs from a cochlear insult caused by rather low-frequency sound. Further experiments are now warranted to directly identify the actual mechanisms of the OHC recovery processes. A better understanding of OHC recovery processes might also improve our understanding of inner ear pathologies related to homeostatic dysfunction.

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COMPLIANCE WITH ETHICAL STANDARDS

All experiments were approved by the ethics committee of the University Hospital of the Ludwig-Maximilians-University Munich, Germany, in agreement with the Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans, and all subjects gave their written informed consent.

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