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Research Paper

Brain's Frequency Following Responses to Low-Frequency and Infrasound

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Complaints and awareness about environmental low-frequency (LF) noise and infrasound (IS) have increased in recent years, but knowledge about perceptual mechanisms is limited. To evaluate the use of the brain's frequency-following response (FFR) as an objective correlate of individual sensitivity to IS and LF, we recorded the FFR to monaurally presented IS (11 Hz) and LF (38 Hz) tones over a 30-phon range for 11 subjects. It was found that 11-Hz FFRs were often significant already at ~0 phon, steeply grew to 20 phon, and saturated above. In contrast, the 38-Hz FFR growth was relatively shallow and continued to 60 phon. Furthermore, at the same loudness level (30 phon), the 11-Hz FFR strength was significantly larger (4.5 dB) than for 38 Hz, possibly reflecting a higher phase synchronization across the auditory pathway. Overall, unexpected inter-individual variability as well as qualitative differences between the measured FFR growth functions and typical loudness growth make interpretation of the FFR as objective correlate of IS and LF sensitivity difficult.

Keywords: low-frequency hearing, frequency-following response, infrasound, auditory brain.

1. Introduction

There is increasing awareness of environmental noise in the infrasound (IS, < 20 Hz) and low-frequency (LF, 20–200 Hz) ranges (Pedersen *et al.*, 2008; Alves *et al.*, 2015; Yamada *et al.*, 2016). Although absolute hearing sensitivity decreases with lowering frequency as a result of peripheral mechanisms (Cheatham, Dallos, 2001; Jurado, Marquardt, 2016), the annoyance and decrease in life quality resulting from prolonged exposure to IS and LF, particularly those associated with emissions from wind turbines, has recently received much attention (e.g. Leventhall, 2009; Schmidt, Klokker, 2014; Baliatsas *et al.*, 2016). However, there is only limited knowledge of the mechanisms behind human perception of sounds of such low frequencies, particularly IS, and especially the brain's response to LF and IS sounds has received relatively little study. We expected that studying a relationship between the perception of and the brain's response to IS and LF sounds might allow in the future a better understanding of individual complaint cases attributed to these sounds.

Kasprzak (2012) examined the effect of airborne IS stimulation (7 Hz at 120 dB SPL; well above threshold according to (Møller, Pedersen, 2004)) on electroencephalography (EEG) patterns and found a significant reduction in the alpha (8–12 Hz) rhythm power when subjects were stimulated by IS. A similar result was found by Kasprzak (2013) using very narrowband (4–8 Hz) IS noise. Dommes *et al.* (2009) used functional magnetic resonance imaging (fMRI) to determine activation of the auditory cortex when stimulated by short LF and IS tone bursts. They found a significant brain activation for 12 Hz presented at 110 dB SPL, which is well above audibility, while no significant cortical activation was found for 90 dB SPL, which is near threshold (for threshold data, see review by Møller and Pedersen (2004)).

While collectively these studies indicate airborne IS can excite, or influence brain activity, it is not clear how their measures correlate with IS audibility, or loudness. In this work, we used EEG, a readily available technique, to obtain the brain's response to IS and LF tones. The main motivation behind these measurements was to evaluate the use of this technique as an

objective correlate of individual sensitivity to IS and LF sounds. Due to the long periodicity of IS tones, it is impossible to obtain transient responses to brief tone bursts, as widely used for EEG experiments and typically utilised in audiology. We decided therefore to adopt the frequency-following response (FFR) for our purpose, an auditory steady state response (ASSR) that captures spectro-temporal properties of ongoing sounds (Hoormann *et al.*, 1992).

Significant correspondence between ASSR and loudness growths has been found for normal-hearing (Ménard *et al.*, 2008; Zenker Castro *et al.*, 2008; Eeckhoutte *et al.*, 2016) and hearing-impaired subjects (Eeckhoutte *et al.*, 2018). For low-modulation rates (near 40 Hz and below), contributions from the auditory cortex to the ASSR are thought to be prominent (Picton *et al.*, 2003; Alaerts *et al.*, 2009). As cortical activity has been found to be closely related to loudness also in fMRI studies (Uppenkamp, Röhl, 2014), it was expected that, similarly to ASSRs to IS/LF modulated sound, also the FFRs to IS and LF pure-tones might correlate with loudness perception.

There is a general trend of the FFR amplitude to decrease with frequency (Batra *et al.*, 1986; Hoormann *et al.*, 1992; Picton, 2010; Bidelman, Powers, 2018) which likely reflects the facts that the ability of neurons to phase-lock to the stimulus periodicity decreases, and that the phase-locked responses generated along the various stages of the auditory pathway become out of sync due to the gradually increasing response latencies along the pathway. Thus, we suspected that IS may be an effective elicitor of the FFR, because its long periodicity may allow phase-synchronized neural activation from cochlea to cortex.

2. Methods

2.1. Sound generation and calibration

Continuous 11-Hz and 38-Hz tones were presented using a custom-made sound source that did not produce harmonics above the sensation threshold. Stimuli were delivered via an 8 m long polyethylene tube (i.d. 14 mm), that connected to a softer silicon tube (length: 0.38 m, i.d.: 6 mm) to which a foam ear plug (ER1-14A, Etymotic, Elk Grove Village, USA) was attached that formed an air-tight seal to the ear canal. Details of this setup are described in (Kühler *et al.*, 2015). The long tube enabled the sound source to be outside the soundproof EEG-recording room in order to avoid electromagnetic crosstalk. To ensure participant's safety, a 1st order passive RC-lowpass filter was in-line between the audio-interface (RME Fireface UC) and the power amplifier (BEAK BAA 120) so that stimuli would not exceed ~95 phon at maximum hardware output. Sound levels were calibrated in a 1-ccm cavity using a B&K 4165 1/2 inch microphone (Brüel & Kjær A/S, Denmark).

The continuous pure-tones were presented to the subjects' left ear. Guided by pilot results obtained with one subject, levels were set for 11 Hz to 0, 10, 20, and 30 phon (95.3, 98.9, 102.5, and 105.3 dB SPL), according to the proposed IS equal-loudness level contours (ELCs) and absolute threshold by Møller and Pedersen (2004). For 38 Hz, levels were set to 30, 40, 50, and 60 phon (77.8, 83.9, 89.7, and 95.3 dB SPL), according to standardized ELCs (ISO 226, 2003); for the first 3 subjects (S1, S2, S3) levels were unintentionally set to 30, 40, 60, and 70 phon (to include these cases in the across-subject mean calculation, their response level to 50-phon was estimated by linear interpolation of the 40- and 60-phon responses). The use of loudness-level steps was motivated by the drastically diminishing dynamic range of the auditory system as the tone frequency reaches the IS range (see the review of equal-loudness-level contours in (Møller, Pedersen, 2004)). I.e., an equal change in SPL will produce far larger changes in loudness for the 11-Hz than for the 38-Hz tone. Measurement durations were 20 minutes for the two lower levels and 10 minutes for the two higher levels.

2.2. EEG recordings

A BioSemi ActiveTwo System (Biosemi B.V., Amsterdam, Netherlands) was used for recording EEG signals at a sampling rate of 16384 Hz. A vertical montage was used, with an active electrode placed at the vertex (Cz) and two active electrodes placed at the mastoids (ipsilateral: M_1; contralateral: M_2), which were used as reference. The Biosemi-specific two ground electrodes (CMS and DRL) were placed on the forehead. This is a traditional configuration recommended for its high SNR (van der Reijden *et al.*, 2004). The running average of the differential voltage between the CMS ground electrode and each electrode was used to monitor electrode impedance, which was kept within ±40 mV as recommended by the manufacturer (Biosemi, 2012). A stimulus-synchronized trigger signal was sent every second via a soundcard audio channel to the trigger input of the Biosemi system. The real-time monitoring of the EEG recording was done using the ActiView software provided by Biosemi.

Thirteen adult subjects (8 female and 5 male; age range 20–34), participated in the experiments. All ear canals were assessed by otoscopy and normal middle-ear function was checked by tympanometry. Subjects underwent a fast pure-tone audiometric test with 11, 38, and 125 Hz (British Society of Audiology, 2011). Two subjects (1 female and 1 male) were discarded, as they presented practically no significant FFRs at either frequency. To avoid alpha-waves during the EEG recordings, subjects stayed awake while watching a silent subtitled movie. All procedures were approved by the UCL Research Ethics Committee.

2.3. Data analysis

All analyses were done in MATLAB (The MathWorks, Inc., Natick, Massachusetts, 2018) using the toolbox Fieldtrip (Oostenveld *et al.*, 2010). Both, ipsilateral and contralateral responses (M_1 and M_2, respectively) were analysed separately, using each as reference (subtracting) electrode against Cz. Recordings were high-pass filtered (f_c = 2 Hz, 1st order) and down-sampled to 4096 Hz, before stimulus-synchronised epochs of 1-s length were extracted from the continuous recording using the recorded triggers. The phase of the spectral component corresponding to the stimulus frequency was then extracted from the FFT of each single epoch, for later phase coherence analysis. The complex spectrum of each epoch was weighted with the inverse of its overall power, to improve the SNR of the grand spectral average, which was obtained by normalizing the sum of all power-weighted spectra by the sum of all weighting factors (Hoke *et al.*, 1984). The spectral component corresponding to the stimulus frequency was taken as the FFR amplitude of the recording. In order to assess whether responses were significantly phase-locked to the stimulus, the phase coherence across epochs was evaluated using the Rayleigh test (Mardia, Jupp, 2000).

3. Results and discussion

Magnitude and growth of the FFR varied substantially across subjects (Fig. 1). Note that the noise floor was ~35 dB (re. 1 nV) near the 11-Hz spectral component and ~25 dB near the 38-Hz component (Fig. 2),

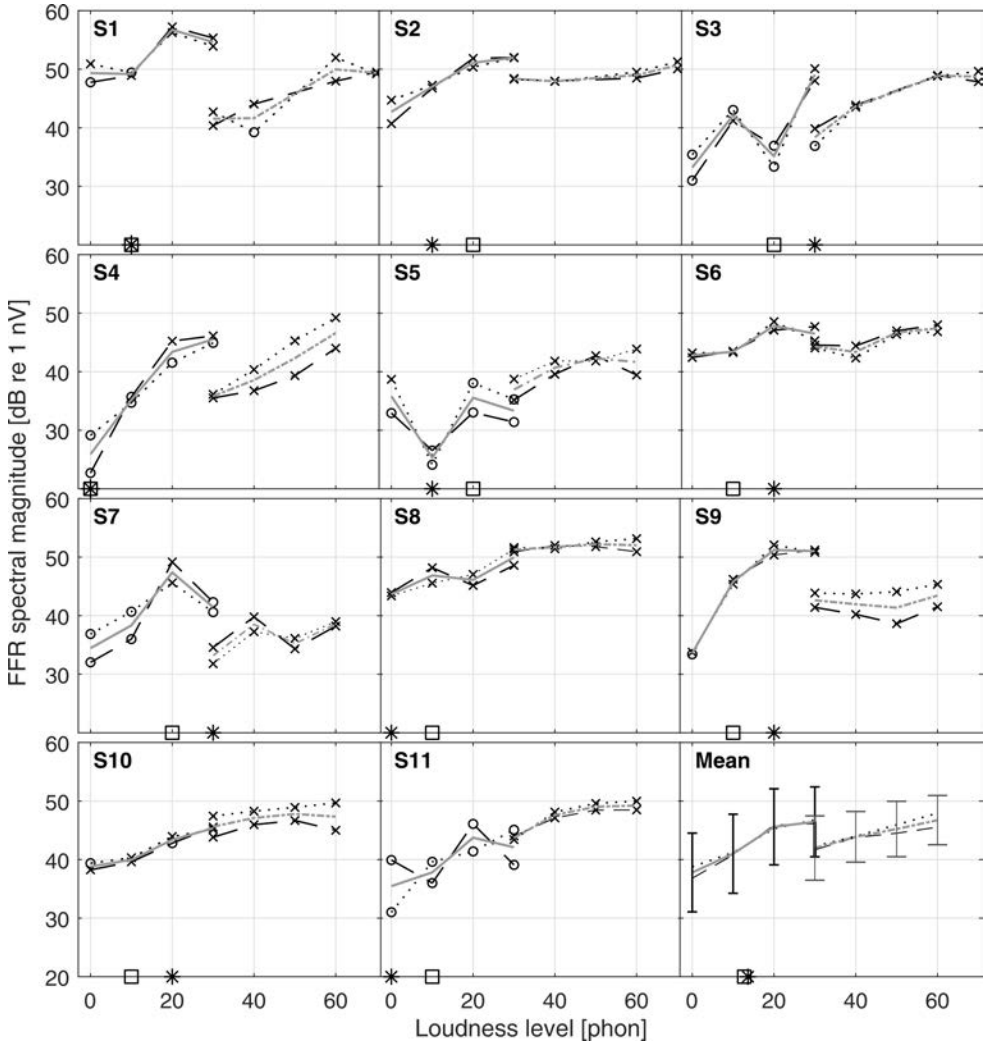

Fig. 1. Spectral magnitudes of individual FFRs for each stimulation frequency as function of loudness level (11 Hz: 0–30 phon; 38 Hz: 30–60/70 phon; ipsilateral: thin dotted lines; contralateral: thin dashed lines). Significant ($p < 0.05$) and non-significant responses are shown with crosses and circles, respectively. The dB-averages of the ipsilateral and contralateral responses are shown as grey lines (solid: 11 Hz, dashed-dotted: 38 Hz). Minimum audible phon levels reported by subjects are given at the bottom of each individual graph (asterisks: 11 Hz, squares: 38 Hz). Group-averages (in dB, with ±1 standard deviations) across all 11 subjects are shown in the right-bottom panel, with the same line styles.

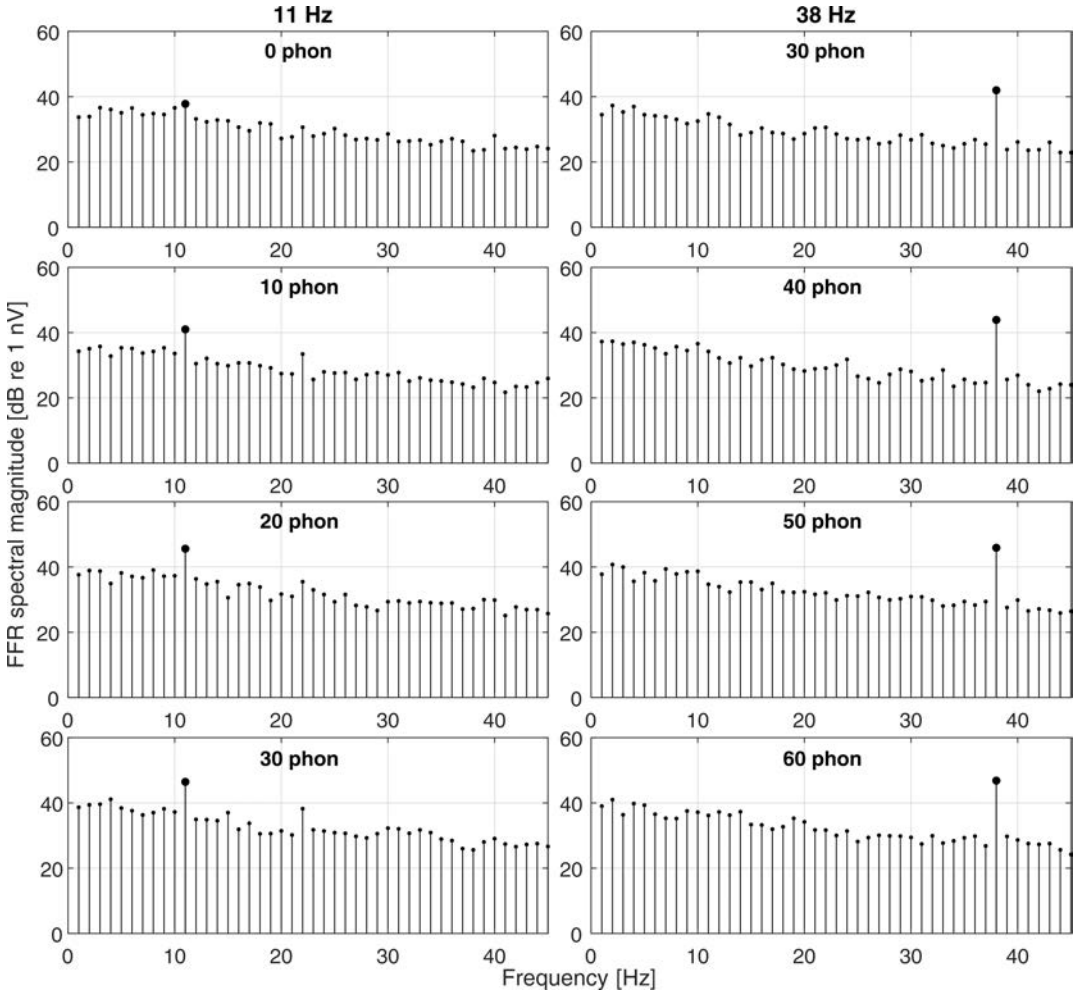

Fig. 2. FFR-magnitude spectra averages across all 11 subjects obtained for each stimulus condition.

so that many weak 11-Hz responses are close to the noise floor (non-significant responses with $p > 0.05$ are marked by circles). Nevertheless, in 7 out of 11 subjects, significant 11-Hz responses were obtained down to 0 phon, and in six of these cases, this stimulus level was apparently below the subject's sensation threshold. This observation, however, should be taken with reservation: The sensation thresholds were estimated rather quickly with a 1-interval Yes-No procedure, as used in clinical audiology (British Society of Audiology, 2011), and not with a more accurate 2-interval AFC staircase method. Nevertheless, many subjects reported after the EEG recording that the 0-phon stimulation was barely audible, or inaudible. In spite of this, a distinct FFR peak is seen in the average magnitude spectrum already for the 0-phon stimulus (Fig. 2). It is worth mentioning here that control recordings were done without placing the earplug into the ear canal, and no electromagnetic crosstalk was detectable even at the highest stimulation levels. Note also that the noise floor in the spectra shown in Fig. 2 remained roughly constant across all stimulus conditions. The spectral component magnitudes at the respective stimulus frequency are equivalent to the values used for the average growth function across the 11 subjects shown in the lower-right panel of Fig. 1.

In addition to the significant synchrony with the stimulus for many of the 0-phon responses, an indication that this spectral peak, so close to the noise floor, reflects the true FFR strength, is the steep increase in the 11-Hz FFR as the stimulus level is increased from 0 phon to 20 phon (0.4 dB/phon). Its slope is twice as steep as that of the 38-Hz growth function (0.2 dB/phon between 30 and 60 phon). This difference is surprising because the stimulating tones were increased in equal loudness-level steps. Although there are marked individual differences, it can be seen that 11-Hz FFRs often saturate already above ~20 phon. In contrast, the 38-Hz FFRs did not clearly saturate up to 60 phon (albeit two of the three growth functions, measured up to 70 phon, indicate that also the 38-Hz FFR might saturate above 60 phon.) These individual trends were reflected in the average growth functions (Fig. 1, lower-right panel). There were no

systematic changes in FFR phase as the stimulation level increased.

Unfortunately, the chosen loudness-level range for the 38-Hz tone (based on pilot recordings that did not show a strong 38-Hz FFR) did not allow us to determine a threshold for the 38-Hz FFR. But nevertheless, we can report that for the same loudness level (30 phon), the average 11-Hz FFR was significantly larger (4.5 dB) than the average 38-Hz FFR ($T = 3.5$, $p < 0.01$; the t-test considered all individual channel data where the FFR to both frequencies was significant). This might be explained by the longer periodicity of an 11-Hz tone, that allows responses from the different generator sites along the auditory pathway to have a higher phase-coherence and therefore to superpose more effectively than those to the shorter 38-Hz tone periodicity, where FFR phases are more dispersed due to the increasing response latency towards higher brain centres. Also, the single periods of the 11-Hz tone are perceptually still resolved as separate events, whereas 38 Hz is already perceived as a single smooth tone. It would therefore not be surprising that the single periods of the 11-Hz tone evoke each a more pronounced "onset" response, with a longer in-between period of recovery from neural adaptation than do the single periods of a 38-Hz tone, for which the neural responses are likely less punctuated in time.

In some cases, the ipsilateral FFR was evidently larger than the contralateral one, as has been observed previously for LF stimulation (Kaf, Danesh, 2008; Tichko, Skoe, 2017). But generally, their strengths were similar, and according to a t-test overall level differences were significant only for 38 Hz (T: 3.2, $p < 0.01$). The fact that the mastoid electrode at the stimulated ear was not clearly stronger activated, lets us conclude that the brain, and not the stimulated cochlea, auditory nerve, or cochlear nucleus, is the main generator of the recorded FFRs. Further, the number of significant FFRs dropped drastically (from 78% to 50%) when analysing data using a horizontal montage (M_1–M_2) vs the vertical montage, indicating FFR contributions are probably larger from brain centres higher than the brainstem, as also their responses are able to phase-lock to the long periodicity of our stimuli (King *et al.*, 2016).

4. Conclusion

Motivated by various studies indicating that human auditory cortex activation is a closer representation of loudness rather than of physical SPL (see (Uppenkamp, Röhl, 2014), for a review), we set out to establish a practical objective measure of individual sensitivity to IS and LF sound using EEG. While the long periodicity, at least of the 11-Hz tone, makes it possible that the auditory cortex is a main contributor to the recorded FFR, preliminary results presented here are rather discouraging, as they do not clearly reflect the perceived loudness of the 11-Hz and 38-Hz tones:

1) Individual response strength varied considerably more than the expected variance in loudness.
2) There was an obvious discrepancy between individually reported sensation thresholds and FFR strength.
3) The FFR growth was often non-monotonic, while the increasing stimulus intensities were clearly perceived as growing in loudness.
4) In addition, the difference in slope of the average growth functions for 11-Hz and 38-Hz FFR is contradictory with the equal loudness-level steps used for both stimuli.
5) The saturation of the FFR growth contradicts the continuous loudness growth perceived with increasing stimulus levels. This all makes an interpretation of the FFR strength in terms of perceived loudness questionable.

While loudness is commonly thought to be associated with the gross-activity of the auditory nerve, the FFR rather captures the superposition of several generator sites along the auditory pathway, which phase lock to the stimulus. Tichko and Skoe (2017) developed such multiple generator model to explain the strong periodic pattern of peaks and troughs seen in their data, which were especially pronounced below 100 Hz down to their lowest stimulation frequency of 16.35 Hz. Although our stimulus frequencies were chosen to roughly coincide with the peaks of their observed patterns, the difference between the FFR strengths to the 11-Hz and 38-Hz tones at the same 30-phon loudness might have been influenced by the exact position within this pattern, rather than only reflecting the commonly reported negative correlation of FFR strength with stimulation frequency (e.g. Hoormann *et al.*, 1992; Picton, 2010).

Nevertheless, previously reported FFR thresholds between 125 and 1000 Hz are in the order of ~30–40 dB SL (e.g. Davis, Hirsh, 1976; Batra *et al.*, 1986; Picton, 2010; Bidelman, Powers, 2018). We were therefore surprised to observe a significant phase-synchrony of the FFR to the 11-Hz tone stimulus when presented very close to its sensation threshold. But also, an increase in cortical connectivity found by Weichenberger *et al.* (2017) for near-threshold IS stimulation, provides supporting evidence from fMRI that a brain activation due to low-level IS indeed occurs. Note also that slowly modulated higher-frequency stimuli evoke an ASSR, which is measurable down to low sensation levels and has been found to increase in strength towards IS modulation rates (Alaerts *et al.*, 2009). Thus, the brain responses to IS pure tones and IS-modulated tones probably both reflect the same

underlying mechanism of synchronized neural phase-locking along the auditory pathway. The slow periodicity of AM sound modulated at IS rates is known to elicit contributions from the auditory cortex to the ASSR (e.g. Weisz, Lithari, 2017), and similarly, FFRs to IS pure tones probably also reflect stimulus-locked activity in the auditory cortex (although the term FFR is often associated with brainstem activity when stimuli with higher frequency content are applied). We conclude that the FFR to IS stimuli can be useful as a positive confirmation of a brain response to barely audible IS stimuli. However, the absence of such response must not lead to the conclusion that a person does not perceive such stimulus.

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