Title

Registered Report: Are anticipatory predictions enhanced in tinnitus and independent of hearing loss?

Authors

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Abstract

Phantom perceptions occur without any identifiable environmental or bodily source. The mechanisms and key drivers behind phantom perceptions like tinnitus are not well understood. The dominant view suggests that tinnitus results from hyperactivity in the auditory pathway following hearing damage. This “altered-gain“ framework, however, has explanatory shortcomings, motivating the pursuit of alternative perspectives. For example, researchers have tried to explain tinnitus within a predictive-coding framework. Supporting this view and commensurate with strong perceptual priors, a magnetoencephalography (MEG) study recently reported that individuals with tinnitus engage more strongly in anticipatory sensory predictions compared to controls without tinnitus. While this pattern did not correlate with hearing loss within the tinnitus group, any correlation among individuals without tinnitus is unknown because they were not given audiometric testing. This registered report aims to close this gap. We will use an established passive-listening paradigm, in which the regularity (i.e. predictability) of pure-tone sequences **is either random or ordered**. Analyses will encompass data from participants with tinnitus and control subjects without tinnitus, matched not only for age and gender, but importantly also in terms of hearing loss. Data from 40 participants with tinnitus and 40 control subjects is already available, and data have not yet been processed. We will utilize previously established decoding-based measures to quantify the extent to which individuals engage in anticipatory auditory prediction. Our hypothesis is that we will replicate our previous main finding: tinnitus and control individuals differ in the extent to which carrier-frequency-specific neural activity patterns become pre-activated, supporting the hypothesis that chronic tinnitus is associated with dysregulated predictive neural processing. This would lay the foundation for any later works that need to disentangle whether dysregulated predictive processes **are a side product of tinnitus or rather pose a risk factor for developing tinnitus.**

Keywords: *auditory perceptions, tinnitus, predictive coding, decoding, hearing loss, MEG*

Introduction

Subjective perceptual awareness is based on huge amounts of environmental inputs (sensations), which are transduced by sensory receptors. Phantom perceptions are peculiar in that they cannot be explained by sensory input. In the case of tinnitus, individuals consciously perceive one or more pure tones or narrowband noises that lack any identifiable source in the environment or the body (Baguley et al., 2013).

Approximately 10-15% of the young to middle-aged adult population experience tinnitus as a common auditory phantom perception, with greater prevalence of 24% in older adults (Henry et al., 2020; Jarach et al., 2022). For a smaller portion of the population, the sensation of bothersome tinnitus poses a significant detriment to quality of life, due to reduced sleep quality, substantially increased distress, and anxiety (Dobie, 2003) – all largely independent of the intensity or duration of the phantom perception (Kandeepan et al., 2019; Meyer et al., 2014).

What neural mechanisms contribute to the generation of tinnitus remains unresolved. Hearing loss has been identified as **a main risk factor** for tinnitus **(Kim et al., 2015)**. Indeed, for 75-80% of people with tinnitus, objective audiometric testing indicates hearing loss (Wallhäusser-Franke et al., 2017). Previous findings support the idea that some form of auditory damage – even without clear audiometric changes – facilitates tinnitus development (Roberts et al., 2006; Schaette and McAlpine, 2011; Schaette et al., 2012; Weisz et al., 2006) and provokes maladaptive changes.

Based on the observation of enhanced neural activity following hearing loss in animal models (Eggermont and Roberts, 2004; Roberts et al., 2010), a still-influential “altered-gain“ view holds that reduced auditory input following hearing damage leads neurons in the auditory pathway to increase their responsivity, thereby restoring their

activity level; in this framework, the perception of phantom sounds is a “downside“ to this homeostatic process, as spontaneous activity can engage downstream auditory regions (Schaette and Kempter, 2006; Sedley, 2019). This model of phantom sound perceptions is supported by research in both animals and computational models (Roberts and Salvi, 2019; Schaette, 2014; Schaette and Kempter, 2012). In humans, resting-state M/EEG studies reported divergent patterns, especially in the delta, alpha and gamma frequency band ranges within and beyond auditory regions (de Ridder et al., 2011; van der Loo et al., 2009; Weisz et al., 2005). **In this regard, the thalamocortical dysrhythmia hypothesis proposes as well that tinnitus development is a consequence of altered neural thalamo-cortical coherence. Findings concerning this hypothesis state that tinnitus is both related to enhanced theta, delta and gamma-band activity in the auditory cortex as well as decreased connectivity between the thalamic medial geniculate body and auditory regions (Brinkmann et al., 2021; De Ridder et al., 2015; Llinas et al., 1999).**

Other potential explanations for tinnitus perception are for instance noise cancellation models (Rauschecker et al., 2010). The noise cancellation model states that due to cochlear lesions and resulting neuroplastic reorganization, hyperactivity in auditory pathways generates or enables acute tinnitus. Normally, noise cancellation mechanisms in the limbic system start identifying and inhibiting the wrong sound signal but in cases of dysfunctions in the limbic system and especially in the anterior cingulate cortex, noise signals persist consciously as tinnitus in the auditory system. Permanent dysfunctions lead to cortical reorganizations which result in chronic phantom sound perceptions (Rauschecker et al., 2010; Song et al., 2015). More recent work also states alterations in a more general cognitive network including

prefrontal, limbic, and subcortical structures which lead to the chronicity of tinnitus (Lan et al., 2022).

Apart from a significant shortage of data bridging animal and human research in these different frameworks, empirical support in humans is weak, difficult to replicate, and marked by strong interindividual variability (Eggermont and Roberts, 2015; Elgohyen et al., 2015)**. Beyond the lack of solid evidence, the models face further explanatory challenges** (Sedley, 2019): 1) People with hearing loss do not necessarily experience tinnitus (Wallhäusser-Franke et al., 2017). 2) The onsets of tinnitus and hearing loss often do not occur at the same time. 3) Not all cases of acute tinnitus transform into chronic tinnitus (Mühlmeier et al., 2016; Vielsmeier et al., 2020). On the whole, this situation calls for the pursuit of alternative or complementary models that place less emphasis on the hearing status of the individual.

One attempt along these lines has been the development of a Bayesian inference framework for tinnitus perception (Sedley et al., 2016). This framework emphasizes the constructive nature of perception being guided by internal models (von Helmholtz, 1867). Therein, sensory input is dynamically compared to predictions or so-called priors. The framework holds that spontaneous activity in the auditory pathway acts as a precursor of tinnitus. In the healthy auditory system, spontaneous activity is “ignored,“ due to the default prior of silence. However, certain circumstances can shift this prior, such that a sound is expected (Hullfish et al., 2019; Sedley et al., 2016). This conceptual model bridges several explanatory gaps: **for example, the inconsistent findings in humans regarding the “altered gain” view which states enhanced neural activity in the auditory pathway. The Bayesian inference framework could, therefore**, explain the experience of tinnitus in lieu of any increase in neural activity in the auditory system.

However, convincing empirical support is still sparse, due to the difficulty of deriving robust measures for tinnitus-supporting priors from ongoing brain activity. Few studies have provided support for altered prediction processes in tinnitus, which is in line with the predictive coding framework using either EEG evoked responses (Mohan et al., 2022; Sedley et al., 2019) or computational modeling (Hu et al., 2021). Furthermore, the question of why only some individuals would shift priors, thus developing tinnitus, remains unclear.

“In a previous work (Partyka et al., 2019), we proposed that, given the tendency to predict auditory events, individuals with stronger prediction tendencies are more vulnerable to developing tinnitus (this is similar to the strong prior hypothesis developed by Corlett et al., 2019). **However, using a cross-sectional design alternative explanations cannot be excluded with certainty, such as tinnitus being the cause of altered prediction tendencies or that there is a third variable being responsible for predictions and tinnitus development. Adjudicating research** would require longitudinal studies **in humans or animals**. As such research is **challenging to implement, especially in humans**, we first focussed on finding group differences between individuals with and without tinnitus.

We utilized a powerful, recently established experimental approach (Demarchi et al., 2019) showing anticipatory activations of tonotopically specific auditory templates for regular tone sequences. The results were highly supportive of increased anticipatory engagement of predictive processes in tinnitus individuals: That is, with increasing regularities of sound sequences, people with tinnitus exhibited stronger anticipatory representations of upcoming stimuli. While these patterns were not correlated with hearing loss *within* the tinnitus group, we lacked audiometric data for individuals without tinnitus. Thus, conclusions that our identified patterns are due to tinnitus rather than hearing loss could not be drawn with certainty.

For this registered report, we recruited a large, new sample in which individuals with and without tinnitus are matched for hearing loss. Using a highly similar experimental design – one more targeted to the core hypothesis – as well as identical analysis methods, we aim to replicate our previous findings, thus strengthening the previous claims.

Hypotheses

We specify the following hypotheses, based on the findings of our previous work (Partyka et al., 2019), in general terms here. The underlying experimental procedure and methods are described in the next section.

H1: Regularity-dependent anticipatory auditory predictions are enhanced in tinnitus.

Our study design allows us to analyze group differences between people with and without tinnitus who are individually matched for age, gender, and hearing loss. We therefore expect group differences in the analyses to be driven by aspects of tinnitus. In the experiment, participants listened passively to tone sequences of four unique carrier frequencies with one of two regularity levels (i.e. random or ordered). As previous results (Demarchi et al., 2019) suggest, we assume anticipatory activations of auditory templates during regular tone sequences but not during unpredictable sequences. Additionally, anticipatory activations seem to be enhanced in patients with tinnitus (Partyka et al., 2019). Using the same analysis steps, we can draw conclusions about 1) how neural information is affected by regularity of carrier-frequency sound sequences and 2) how this is affected by tinnitus while taking into account hearing loss as a potential confound. We hypothesize that we will find differences in regularity-driven carrier-frequency-specific neural pre-activations between the tinnitus group and the matched control group (Figure 1b, middle).

With the second and third hypotheses, we aim to strengthen the findings regarding H1 by analyzing potential influences of the results.

H2: Individuals with tinnitus show normal processing of tone-carrier frequencies.

With our paradigm, we plan to analyze both the group effects of regularity-dependent neural activity from sound sequences and the influences of hearing loss and tinnitus characteristics on these effects. We aim to ensure that the actual effects of interest – i.e. the patterns of anticipatory predictions established by the regularity of tone sequences in people with tinnitus versus those in control subjects – are not due to altered encoding of tone-carrier frequencies in general for tinnitus individuals. Therefore, it is important that the decoding accuracy for carrier frequencies is similar for both groups in the random sound sequence (Figure 1b, left).

H3: Enhanced regularity-dependent anticipatory predictions in tinnitus are not related to subjective tinnitus distress.

People with tinnitus vary in levels of subjectively perceived tinnitus distress. These individual differences within the tinnitus sample were previously addressed in our work and no influence on the main effect was found. In order to strengthen the results, we plan to address these differences in a statistical manner as well to draw conclusions about potential influences on auditory predictions. In the case that enhanced anticipatory auditory prediction is more a general feature for individuals developing tinnitus, we hypothesize that our main effects will not be correlated to tinnitus distress. In line with the direction of the hypothesis, we will be able to support the assumption that temporally more stable features of each individual will draw the effects, instead of current tinnitus characteristics (Figure 1b, right).

Methods

For this study, participants have already been recruited and measured using magnetoencephalography (MEG), but the data have not yet been processed. We propose Level 3 for the registered report since MEG data is accessible to the corresponding author via a group intern database. However, we justify that no part of the data has been observed yet. Due to the procedure to create anonymous participant codes, it is not apparent to the authors which data files correspond to individuals with or without tinnitus. Further analyses of the participant characteristics are necessary to link information regarding tinnitus to the participant codes and corresponding files. These characteristics are with a colleague and have not been accessed by the authors yet. **In order to further blind the researchers during the analyses, the subject files will be assigned to two groups (tinnitus vs. control) without the involved researchers knowing which group represents which condition. The information will not be passed to the involved researchers until the analyses are completed.**

We obtained approval for the experimental procedure from the ethics committee of the University of Salzburg (EK-GZ: 22/2016 with Addenda). The study design consisted of pure-tone audiometry, followed by the MEG experiment (see below).

Sampling Plan

We planned to reach a sample size of 80 individuals - i.e. 40 participants with tinnitus and 40 age-, gender- and hearing-matched controls without tinnitus. The following arguments strengthen this decision. Most MEG studies targeting tinnitus include smaller samples up to 25 participants per group (see for example Lorenz et al., 2009; Okamoto et al., 2009; Schlee et al., 2009). One recent work based their analyses on an outstanding „larger MEG data set“ (Paraskevopoulos et al., 2019),

including 40 tinnitus patients and 40 control subjects. However, individuals were not matched for audiometric hearing loss as it is the case in our proposed work. Based on previous research in this field, our sample is therefore even more unique and outstanding.

Next, we target a clinical topic for which recruiting participants is more challenging since we are looking for specific characteristics in volunteers. Additionally, finding suitable controls for our strict matching procedure is time consuming as well since lab capacities are restricted. With respect to clinical relevance, solely strong effects are worth pursuing because of the difficult circumstances in data collection. **Therefore, our power analysis was based on a theoretical estimate of a medium to large effect size (*d*=0.75). Using G*Power (Faul et al. 2009), we calculated an estimated sample size for a one-sided t-test, expecting a true effect of *d*=0.75. We used a power of 0.95 and a one-sided α-error probability of 0.05, which resulted in a required total sample size of at least 80 participants.** This is also in line with our previous arguments warranting a sample of 40 individuals with tinnitus and 40 controls.

At the date of the Stage 1 submission, data collection of 80 participants was already completed. **With our analyses, we therefore aim for a smallest estimated effect size of interest of *d*=0.75 to be found with a power of 95 % and a conventional alpha at 5 %.**

Participants

40 individuals with tinnitus (16 females, age 24-74 years, *mean*=57.73, *sd*=14.12), as well as 40 hearing-, age- and gender-matched control subjects (16 females, age 24-76 years, *mean*=57.43, *sd*=13.94) have completed the experiment. For the tinnitus group, inclusion criteria was a tinnitus duration of more than six months. No participants with psychiatric or neurological diseases were included in

the sample. Participants were recruited via two procedures. First, we used an online study by our group on hearing epidemiology in the county of Salzburg (Austria) as a recruiting database. The online study included demographic information as well as questionnaires covering tinnitus (**German short version of Tinnitus Questionnaire, Mini-TQ** (Goebel and Hiller, 1992)) and hearing characteristics (German version of the Speech, Spatial and Qualities of Hearing Scale, SSQ (Kiessling et al., 2012)), along with an online hearing test (Shoebox, Ottawa, Canada). **The Mini-TQ includes subscales targeting emotional distress, cognitive distress and sleep disturbances which we will use to draw conclusions about the impact of tinnitus distress (Hiller & Goebel, 2004).** We included a question asking whether participants would be interested in further investigations in the laboratory, and we contacted them depending on their consent and their hearing profile from the online hearing test. Second, our paradigm was part of a broader epidemiological study (Frey et al., 2022), and participants from this cohort were invited to the MEG lab for further measurements, including the experimental paradigm described in this study.

We performed standardized pure-tone audiometry for frequencies from .125 to 8kHz in all participants using an Interacoustic AS608 audiometer to characterize hearing status. **Hearing loss was defined by a hearing threshold above 30 dB in at least one frequency. Four individuals with tinnitus did not show any audiometric peculiarity; four of the participants showed unilateral hearing impairments; 26 volunteers had high-frequency hearing loss; and six individuals were hearing impaired over most frequencies.** The control group was recruited afterward in order to match the distribution of the tinnitus group by age, gender and hearing status. **Accordingly, we aimed to find the best possible match that our data allowed for between individuals with tinnitus and control subjects regarding the results of the audiometry. Using independent t-tests,**

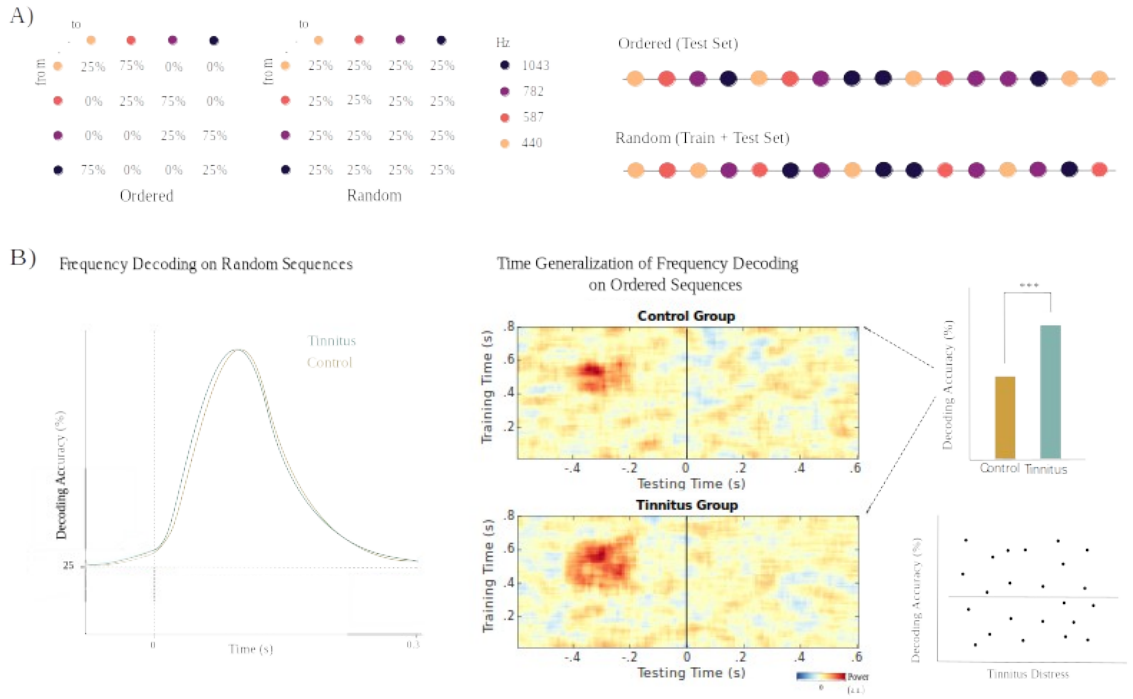
we found no differences in hearing status between groups for the left (*t*=-1.32, *p*=.192) and right ear (*t*=-1.27, *p*=.212). Control subjects were age-matched to each tinnitus participant by a +/-2-year criterion, choosing the closest match when more than one subject was suitable. All participants provided written informed consent before participating.

Stimuli and experimental procedure

Prior to entering the shielded MEG room, we applied five head position indicator (HPI) coils to the scalp of each participant. We used a Polhemus FASTRAK (Polhemus, Colchester, Vermont, U.S.A) digitizer to register head shape and position for each individual by marking nasion and left/right pre-auricular points, location of the HPI coils and approximately 300 additional points over the scalp. After this preparation, we performed a 5-minute resting-state recording and a 20-minute audiobook block (neither used in the analyses of this work). Next, participants passively listened to sound sequences without further instruction, while watching a silent nature documentary. The movie was displayed using a projector (PROPIXX, VPixx technologies, Canada) and a periscope onto a screen inside the shielded room. Auditory stimulation was presented to both ears via MEG-compatible pneumatic in-ear headphones (SOUNDPixx, ibid).

We presented four different pure (sinusoidal) tones, with carrier frequencies logarithmically spaced between approximately 400 and 1000Hz (i.e. 440 Hz, 587 Hz, 782 Hz, 1043 Hz; Figure 1a). This frequency range differs from our original paradigm (Demarchi et al., 2019) of frequencies between 200 and 2000Hz. We reduced the carrier frequencies to a maximum of 1000Hz to further ensure that the sounds provided were within **a region of normal audiometric thresholds**. Specifically, we aimed to avoid potential effects of high-frequency hearing loss on the highest-frequency tones. Each tone lasted 100 ms, tapered at both ends with 5 ms linearly

ascending/descending periods, and we presented the sounds at a constant 3Hz stimulation rate. **Sound intensity was individually determined by presenting a short audio sequence to the participants and adjusting the loudness according to an individual pleasant volume.** We combined the sound sequences into two continuous blocks, each lasting approximately 8 minutes. In contrast to our previous work (Partyka et al., 2019), we did not include omissions of single tones in the sequences. We balanced the number of stimuli across blocks, and each block contained 1500 particular tone frequencies. Within each block, groups of 500 consecutive stimuli followed the same regularity (entropy) level, which was parametrically modulated using various transition matrices (Nastase et al., 2014). We used two entropy conditions for the design. The random condition had the highest entropy (i.e. the lowest regularity), and the transition probabilities from one sound to another were equal, preventing any possibility of accurately predicting upcoming stimuli. By contrast, the ordered condition had the lowest entropy level (i.e. the highest regularity), and in 75% of trials, one sound was followed by a specific other sound. In 25% of trials, the same sound was repeated (Figure 1a). These groups of 500 stimuli with a particular entropy condition were presented in random order within each of the two blocks. **To balance the number of conditions, one of the two blocks started with a random condition (500 stimuli), followed by an ordered sequence (500 stimuli) and ended with a random condition (500 stimuli). For the other block, sounds started accordingly in an ordered condition, followed by random sounds and a second sequence of ordered sounds. Therefore, data collection comprised 1500 stimuli of each condition.** The experiment was written using the MATLAB-based (version 9.1 The MathWorks, Natick, Massachusetts, U.S.A) Psychophysics Toolbox (Brainard, 1997).



330 ***Fig. 1:*** *Stimulus design and expected results.* ***A)*** *Left panel: We presented sound sequences of four*
331 *different carrier frequencies to participants. Transition probabilities varied between the different*
332 *entropy conditions (ordered vs. random). Right panel: Example sequences for the ordered and*
333 *random conditions.* ***B)*** *Using decoding approaches, the hypotheses focus on both the pre- and post-*
334 *stimulus intervals. Left panel: for the post-stimulus interval, we will perform frequency decoding on*
335 *random sequences, and we expect no differences between the tinnitus and control groups (H2).*
336 *Middle and upper right panel: However, when training on random sound sequences and testing on*
337 *ordered in a time-generalized manner, we expect higher activation during the pre-stimulus interval in*
338 *the tinnitus group (H1). Right lower panel: We will correlate the individual decoding accuracies of the*
339 *pre-stimulus interval with the subjective tinnitus distress of each individual in the tinnitus group, and*
340 *we expect no significant effects (H3).*

341 *MEG data acquisition and preprocessing*

342 We measured magnetic brain activity using a whole-head MEG (Triux, MEGIN
343 Oy, Finland), in which brain signals were captured by 102 magnetometers and 204
344 orthogonally placed planar gradiometers. Participants sat in a dimly lit magnetically
345 shielded room (AK3b, Vacuumschmelze, Germany) and were measured with a
346 sampling rate of 1000 Hz and default hardware filters set by the manufacturer (0.1

Hz high pass - 330 Hz low pass). We plan to use a signal-space separation algorithm (SSS (Taulu and Kajola, 2005)) implemented in the Maxfilter program (version 2.2.15) to reduce external noise from the MEG signal (mainly 16.6Hz, and 50Hz-plus harmonics) and to realign data of different measurement blocks to a common standard-head position ("-trans default" Maxfilter parameter), based on the head position measured at the beginning of each block (Cichy and Pantazis, 2017). **Additionally, the Maxfilter algorithm will detect bad channels, remove and interpolate the data.**

The analyses will be based on magnetometers only, since information between magnetometers and gradiometers is mixed after the Maxfilter step (Garcés et al., 2017) and will be carried out with our own scripts, including the Fieldtrip toolbox (Oostenveld et al., 2011). For preprocessing the data, we will apply a high-pass filter at 0.1 Hz (6th order zero-phase Butterworth filter), as well as a low-pass filter at 30 Hz, to the raw data and use it as an input for an Independent Component Analysis (ICA) algorithm. Next, we will inspect the ICA components visually to detect and remove unwanted artifacts, such as eye blinks and movements, heartbeats and 16 ⅔ Hz artifacts (the level of German/Austrian train power supply). **We will report the number of removed components for each group to highlight whether the number of components differed substantially across groups.** After eliminating these components, we will epoch the continuous data into chunks from 400 ms before to 500 ms after sound onset to enable analysis of both regularity-dependent pre-activations and post-stimulus decoding accuracies. In a final step, we will down-sample the data to 100 Hz to further use it for multivariate pattern analyses (MVPA).

Multivariate Pattern Analysis (MVPA) and decoding weights projection analysis

We aim to use Multivariate Pattern Analysis (MVPA) as implemented in the MVPA-Light toolbox (https://github.com/treder/MVPA-Light), which was modified to

extract classifier weights (https://github.com/gdemarchi/MVPA-Light/tree/devel). For decoding, we will define four target classes in line with the frequencies of the sound presented in each specific trial. In order to avoid potential carryover effects from previous sounds and to focus exclusively on carrier-frequency-related information and the corresponding neural templates, we will train the classifier solely on the random sound sequences.

We plan to train a multiclass linear discriminant analysis (LDA) classifier on each sample point of the random condition and to average the classification accuracy for each subject at a group level for further comparisons. Additionally, we will use a temporal generalization method (King and Dehaene, 2014) to analyze the ability of the classifier to generalize across time points in the training set to time points in the testing set. When testing on the ordered condition, we will not perform any cross-validation, as our approach already consists of cross-decoding. **For testing on the random tones, we will perform a 5-fold cross-validation. It is further important to specify that we will train on the post-stimulus interval and test on the pre-stimulus interval of the random tones.** We will construct two time-generalization matrices: one for each condition.

In the final step, we will extract the training decoder weights of relevant pre-stimulus time frames and project them in the source space in order to localize the informative activity of carrier-frequency processing (Demarchi et al., 2019; Marti and Dehaene, 2017). We will compute single-shell head models (Nolte, 2003) by co-registering the headshapes of the participants with a standard brain template from the Montreal Neurological Institute (MNI, Montreal, Canada). A grid with 1 cm resolution and 2982 voxels will be morphed to fit the individual brain volumes of the participants. As a result, we will be able to perform group-level analyses, since all grid points belong to the same brain regions across subjects.

Statistical analysis

With the decoding approach, we will obtain decoding accuracies over time for each participant. For statistical analyses, we will use cluster-based permutation t-tests (Maris and Oostenveld, 2007), with 1000 permutations and a value of $p < .05$ to threshold the clusters in order to account for multiple comparisons. We will use these cluster-based permutation t-tests to compare the tinnitus and control groups in terms of H1 and H2. Accordingly, we will target the pre-stimulus and post-stimulus intervals separately.

For H1, we will analyze group comparisons of whether regularity-dependent pre-activations of carrier-frequency-specific information differs between individuals with and without tinnitus. For this, we will consider the pre-stimulus interval **(-400 to 0 ms)** to perform cluster-based permutation t-tests. In a time-generalized manner, we will train the classifier on the random sound sequences and test on the ordered sequences to take into account the predictability in the ordered sound sequences. Using both entropy conditions, we will be able to extract potential regularity-dependent pre-activations of carrier-frequency-specific information. Next, we will compute group averages and extract relevant clusters in the pre-stimulus interval as an indicator for regularity-dependent pre-activations. Finally, we will statistically inspect the differences in the clusters between the groups by performing cluster-based permutation t-tests and comparing mean decoding accuracies between tinnitus and control groups.

Then, considering the post-stimulus interval **(0 to 400 ms)** for statistical analysis, we will be able to draw conclusions about H2, regarding normal carrier-frequency processing in the tinnitus and control groups. This will allow us to strengthen the effects of the first analysis by controlling for potential basic differences in carrier-frequency processing between the tinnitus and control groups. For this analysis, we will consider solely the random-sound condition to analyze

frequency decoding per se, without potential predictability effects. We will then be able to compare the resulting decoding accuracies over time between groups by identifying significant clusters and implementing cluster-based permutation t-tests. Since we expect no difference between groups, we will add equivalence testing to strengthen our results.

We will analyze H3 by extracting individual values **of the short version of the Tinnitus Questionnaire (Mini-TQ)** and calculating the mean subjective tinnitus distress for each individual of the tinnitus group. Next, we will correlate the individual tinnitus distress values with the mean decoding accuracy of each individual in the previously analyzed pre-stimulus interval.

Previous results

As described above, our hypotheses and analyses derive from previous work (Partyka et al., 2019). We therefore describe the prior results in this section and connect them to the current hypotheses.

For the analysis targeting regularity-dependent pre-activations of neural information, a classifier was trained on the random-sound sequences and applied to both regularity conditions in a time-generalized manner to capture carrier-frequency-specific dynamics showing predictive processing. We intend to use the same methods for our current hypothesis H1. Decoding accuracy acted as an indicator for the strength of internal representations of the stimulus frequency. In the previous study, linear regressions between decoding accuracy and regularity level were calculated at each time point for each participant in order to quantify how the predictability of the carrier frequency modulates corresponding neural information. In both groups, anticipatory pre-activation of carrier-frequency-specific neural templates was reported for early training-time periods (Figure 2c). Additionally, an independent t-test was applied to compare individual β-coefficients between groups for each point

in the time-generalization matrix (Figure 2d). For individuals with tinnitus, a greater increase of decoding accuracy by regularity level was reported prior to the onset of the to-be-decoded stimulus. The results were therefore interpreted as showing stronger correct anticipation of a stimulus in high-regularity conditions among participants with tinnitus. Additionally, in the post-sound-onset time window, group differences in deactivation of carrier-frequency patterns appeared. Individuals with tinnitus showed quick deactivations in regular sound sequences, while control subjects showed reactivated decoding patterns until the next stimulus was presented. These findings supported the hypothesis that individuals with tinnitus process auditory events in a more anticipatory manner by using internal models.

Considering the post-stimulus interval, the previous results reported a rapid increase in above-chance decoding accuracy immediately after sound onset in both groups (Figure 2a). Additionally, decoding accuracy remained statistically significant for approximately 500-600 ms. Approximately 100 ms after the onset of the following sound (i.e. 450-500 ms after the target sound), accuracy increased as well, but at a smaller magnitude. The current hypothesis H2 is supported by these previous results, in which no differences between the tinnitus and control groups were observed for the decoding of randomly presented carrier frequencies. Importantly, the upper carrier frequency of 2000 Hz was near the audiometric edge of the majority of individuals with tinnitus, whereas participants in the control group did not show matching hearing loss. In the present study, we control for hearing loss by matching the control group and including lower carrier frequencies, between 440 and 1043 Hz, below any potential audiometric edge, to avoid limited interpretability due to the study design.

The last analysis took hearing status and tinnitus characteristics into account. In the current H3, we address solely tinnitus distress, since we have already controlled for hearing status with our hearing-matched control group. In the previous

results, Spearman correlations between the averaged β-regression values were calculated, which corresponded to significant clusters and magnitudes of hearing loss, as well as tinnitus distress. With the previous sample, no significant correlations within the tinnitus group were reported. However, since the sample was not matched for hearing loss between the tinnitus and control groups, interpretability of the results was limited. In the current sample, we aim to overcome these limitations and to re-analyze the correlation between regularity-dependent activations and subjective tinnitus distress.

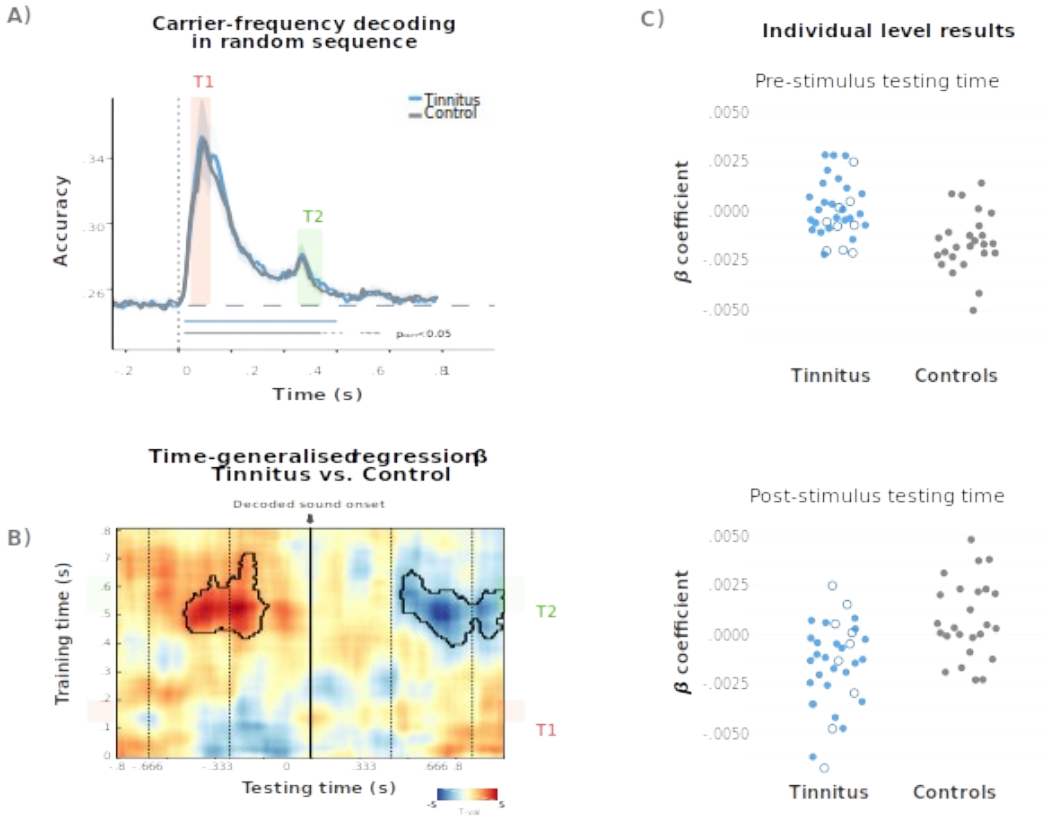


Fig. 2: A) *Temporal decoding of carrier frequencies in the random-sound sequence for tinnitus and control groups, respectively. In both groups, peak accuracy is reached after ~100 ms after sound onset. Above-chance decoding accuracy is observed in a sustained manner up to ~600 ms (p < .05, Bonferoni corrected). No differences were observed between groups.* ***B)*** *Group comparison of β-coefficient values between tinnitus vs. control groups in time-generalized matrix. Colors indicate t-values and solid black borders delimit periods of significant difference (p < 0.05, cluster corrected).* **C)** Individual β-coefficient values within pre- and post-sound clusters.

494 Table 1. Design Table

Question	Hypothesis	Sampling Plan	Analysis Plan	Rationale for deciding the sensitivity of the test for confirming or disconfirming the hypothesis	Interpretation given different outcomes
Do individuals with tinnitus show different regularity-dependent pre-activations of carrier-frequency-specific information compared to a control group without tinnitus?	H0: No. H1: Yes. Referring to our previous results, we expect higher regularity-dependent pre-activations in the tinnitus group.	See section *Sampling Plan*	**Mean** decoding accuracies in the pre-stimulus interval will be compared between groups, using a cluster-based permutation t-test.	We base our decision on the minimum requirement of an effect size of d=0.75 with a certainty of 95% and an alpha-level at 0.05.	H0: Finding no group differences would contradict our previous results (Partyka et al., 2019) and highlight discrepancies between study designs. Either differences in the stimuli or in the sample might be responsible for such results. In the latter case we would not be able to exclude influences of hearing loss on the results. H1: Similarly, to our previous results, we would cautiously interpret stronger regularity-dependent pre-activations as a sign of increased vulnerability to developing tinnitus. This is in line with reports about auditory hallucinations and links to strong priors.
Are tone-carrier frequencies processed normally in individuals with tinnitus?	H0: No. H1: Yes. As previous results suggest, there are no differences in processing of different tone-carrier frequencies between individuals with tinnitus and without.	See section *Sampling Plan*	**Mean** decoding accuracies in the post-stimulus interval will be compared between groups using a cluster-based permutation t-test.	We base our decision on the minimum requirement of an effect size of d=0.75 with a certainty of 95% and an alpha-level at 0.05.	H0: Deviations in normal tone-carrier-frequency processing in individuals with tinnitus contradict our previous results. It is important to extract differences in the study designs and to filter out the variables that might influence results. H1: We interpret normal tone-carrier-frequency processing in individuals with tinnitus as in-line with previous findings, indicating no abnormal tonotopic representations in individuals with tinnitus.
If individuals with tinnitus show different regularity-dependent pre-activations of carrier-frequency-specific information, are these effects not driven by any influence of subjective tinnitus distress?	H0: No. H1: Yes. These effects are explained exclusively by tinnitus and not by confounds like tinnitus distress.	See section *Sampling Plan*	Mean decoding accuracies **in the pre-stimulus interval** will be correlated with a mean value of subjective tinnitus distress.	We will decide based on the significance of the correlation.	H0: Correlations between the effects and subjective tinnitus distress would suggest the importance of the current tinnitus state. We would suggest longitudinal studies to further investigate the influence of tinnitus characteristics on regularity-dependent pre-activations of carrier-frequency-specific information. H1: Similar to our previous results, we interpret independence of tinnitus distress and the effects as a sign of individual predispositions to tinnitus development and resulting regularity-dependent pre-activations of carrier-frequency-specific information, which are not correlated to the current characteristics of tinnitus but more likely temporally stable “trait-like” features.

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