Four electrophysiological studies into noise sensitivity

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ABSTRACT

Noise sensitivity is present in many clinical populations, describes approximately 20% of the general population, though little is known about its underlying mechanisms. We present findings from four electrophysiological studies designed to expose possible differences in electrophysiological measures between noise sensitive and noise resistant individuals. Noise sensitivity was estimated using self-report measures, while electrophysiological indices included both cardiac (heart rate, heart rate variability) and electroencephalographic (event-related potentials, alpha persistence) measures. All four studies were designed with reference to pre-existing theoretical frameworks. While the findings from all four studies were not definite enough to decide a likely mechanism, they do suggest that electrophysiological investigation of noise sensitivity is viable and in need of further investigation.

Keywords: Noise Sensitivity, Electrophysiology, Biological Mechanisms
I-INCE Classification of Subjects Number: 62.2

1. INTRODUCTION

Noise sensitivity is generally considered a stable trait, describing individuals who are more likely to attend to sound, evaluate sound negatively, experience enhanced emotional reactions to sound, and subsequently have greater difficulty habituating to sound (1). While epidemiological research into noise sensitivity has largely focused on the psychological origins of noise sensitivity, across relevant disciplines few studies have been reported focusing on physiological factors.

Noise sensitivity has a genetic component (2), suggesting biological factors may be significant predictors. While there has been calls to clarify the physiological underpinnings of noise sensitivity (e.g., 3), few studies have been reported, and most focus on sensitivity to noise during sleep, which have progressed sufficiently to identify underlying biological processes (4). Studies on road traffic noise suggest that differences in cardiovascular response exist between noise sensitive and noise resistant individuals for loud (85 dBA) samples of traffic noise, but are negligible for normal exposure levels (5). Stansfeld (1992), studying a sample of female psychiatric patients, noted higher tonic skin conductance and heart rate in those reporting high levels of noise sensitivity, as-well-as slower habituation of heart rate to threatening sound (1).

The electrophysiological correlates of noise-induced annoyance have been well considered (e.g., 6), and given the strong covariance between noise annoyance and noise sensitivity, it can be conjectured that noise sensitivity will likewise covary with electrophysiological measures capturing annoyance reactions. We report four studies undertaken within four distinct theoretical frameworks in order to further understand noise sensitivity. Though of clinical relevance, the studies utilised participants from nonclinical populations as high noise sensitivity is prevalent across the general population, with conservative estimates of approximately one-in-five being reported (2). Specifically, using a non-clinical population may afford greater variability and representation of noise sensitivity in participants, allowing the covariance between noise sensitivity and electrophysiological indices to be more sharply scrutinised.

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2. STUDY 1: Heart Rate

2.1 Introduction

Bradley and Laing (2000), referencing the valance-arousal model of emotion, report that highly arousing sounds rated as unpleasant stimulate a greater deceleration in heart rate (HR) compared to pleasantly rated sounds (7). One of the primary characteristics of noise sensitive individuals is a tendency to respond negatively to sounds in general (1). Because noise sensitivity amplifies the emotional response to sound, it would be expected that these intensified emotional reactions (i.e., arousal) could be manifested physiologically as changes in cardiac activity. Specifically, those sounds considered typically pleasant may be judged negatively by noise sensitive individuals, and so a greater deceleration of HR would be expected relative to non-noise sensitive individuals.

2.2 Method

The participants were 59 university staff members or postgraduate students, all who volunteered for the study. There were 27 males (M_{age} = 41.04, SD = 12.07) and 32 females (M_{age} = 43.37, SD = 12.31). Each participant's sensitivity to noise was measured using the 35-item Noise Sensitivity Questionnaire (NoiSEQ). Each item consists of noise-related statement and a five point Likert-type scale, and after the reverse-coding any negatively-worded items, the average of the 35 items provides a measure of global noise sensitivity. Note that higher scores indicate greater sensitivity to noise.

The study was conducted in a laboratory setting, and all data acquisition processes were undertaken by a trained technician. On arrival, participants were provided with an overview of the experiment, and informed consent was obtained. A pure tone audiometric assessment was performed at the beginning of the study prior to the attachment of electrodes. Throughout the session, participants were comfortably seated in an upright position while their physiological responses were recorded. During the session sounds were presented at one minute intervals in a random order. Twelve, six-second, digitized sound samples were selected from the International Affective Digitized Sounds (IADS) library (7). In total there were six pleasant (birds singing (IADS 151); a bubbling brook (IADS 172); church choir (IADS 812); harp (IADS 809); classical music (IADS 810:811)) and six unpleasant (alarm signals (IADS 712:713:715); a man vomiting (IADS 255); a woman screaming (IADS 275), a jack hammer (IADS 380)) sound samples.

All ECGs were recorded using a 16-bit Nexus-10 analogue-to-digital unit (Mindmedia) set to a sampling rate of 2048 Hz. The electrode sites were degreased with isopropyl alcohol skin cleansing swabs, and subsequently abraded using a defoliant to reduce impedance prior to electrode placement. All ECG was recorded using three disposable pregelled Ag/AgCl electrodes positioned in a three-lead unipolar modified chest configuration. The two active electrodes were placed slightly below the right collar bones and the lowest rib on the left side of the chest, while the ground electrode was positioned on the left collarbone. Each ECG provided estimates of HR (beats per minute: BMP) using proprietary algorithms available in the acquisition software (Biotrace).

In line with previous studies (7), the ECG recordings were partitioned into pre- and post-stimulus epochs (3 seconds before sound onset, and 6 seconds following sound onset). The post-stimulus epochs were divided into 500-ms bins, and then average HR calculated for each. Mean HR was calculated for the three second pre-stimulus interval and then subtracted from the post-stimulus onset bins in order to calculate the mean HR deviations (1), be they an acceleration or deceleration. Three groups were created from the noise sensitivity data using tertiles: low, medium and high noise sensitivity. To gauge the relationship between noise sensitivity and HR change, a 2 (pleasant vs. unpleasant sounds) x 3 (low vs. medium vs. high noise sensitivity) group design was used.

2.3 Results and Discussion

Figure 1 plots mean HR change as a function of time, for both pleasant and unpleasant valences. Scrutinizing the functions suggest no difference between the three noise sensitivity tertiles for unpleasant sounds, and qualitatively these functions follow those reported by Bradley and Laing (7: their Figure 5, p. 212) very closely. For the pleasant stimuli, however, inspection of the functions suggests a difference across the three tertiles, specifically the three seconds following the onset of sound samples. A mixed between-within subject analysis of variance was conducted to assess the impact of valance and noise sensitivity on mean HR deviation, while controlling for age. A repeated-measures ANCOVA revealed no main effect of valence ($F(1,59)=0.037$, $p=.848$) or noise sensitivity ($F(2,59)=0.526$, $p=.594$), but did return an interaction effect between the two

($F(2,59)=0.3486$, $p=.038$). Simple effects tests revealed significant differences between the low and high noise sensitivity groups for pleasant sounds ($p=.011$), with no other paired-comparisons attaining significance. For completeness, Figure 1 shows significance differences ($p<.05$) across the 500-ms bins.

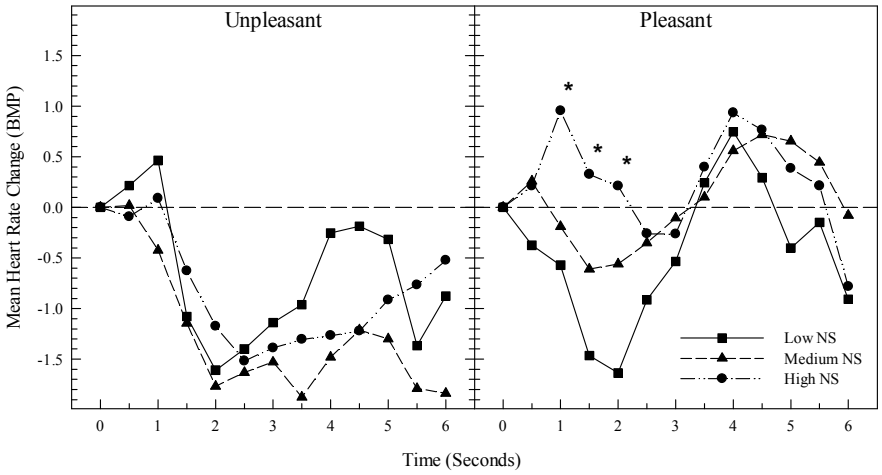

Figure 1 – Mean heart rate change as a function of time for unpleasant (left) and pleasant (right) sounds. Symbols pertain to group tertiles representing level of noise sensitivity, and an asterisk indicates a significant difference in mean HR between low and high tertiles.

The main finding from Study One is that mean HR deviations were significantly influenced by a combined effect of valence and noise sensitivity. For unpleasant stimuli, the differences between the three noise sensitivity tertiles were minimal, suggesting that both psychological and physiological responses were equivalent across the three groups. For the low sensitivity group, the effect of valance was minimal, mirroring the findings of Bradley and Laing (1), and suggesting that arousal to the 12 samples was similarly intense. The medium noise sensitivity group produced a function for the pleasant stimuli that fell between the low and high tertiles, indicating internal validity. For the high noise sensitivity group, an effect of valance was noted, with greater mean HR decelerations for unpleasant as opposed to pleasant stimuli, the latter of which is associated with initial HR acceleration. This finding does not concur with Bradley and Laing's (1) model unless one assumes that the low noise sensitivity group was less aroused than the high noise sensitivity group when presented sounds from the pleasant stimulus set. Regardless, the evidence suggests that an association may exist between noise sensitivity and autonomic activity, a thread continued in Study Two below.

3. STUDY TWO: Heart Rate Variability

3.1 Introduction

Thayer and colleagues (e.g., 8) explain maladaptive responses to environmental stimuli using a neural-visceral integration model. In their scheme, stressors induce maladaptive psychological responses that impact autonomic function, notably decreased parasympathetic dominance and increased sympathetic activity. Parasympathetic dominance is associated with greater cardiac flexibility due to its faster signalling speed (8) and slower cardiac rhythm. Heart Rate Variability (HRV), the temporal variation in the latency between consecutive heartbeats, is thus taken as an indirect measure of autonomic influences on the heart. A plethora of HRV indices exist, all derived from the analysis of inter-beat intervals extracted from the cardiac time series. As the parasympathetic system is responsible for most of the variation in the inter-beat interval, greater HRV is thought to reflect greater parasympathetic dominance. This is important, as cardiovascular function must be variable because environmental demands are in a constant flux. HRV thus reflects the adaptability of the individual to their environment, with greater HRV reflecting improved ability to self-regulate in a constantly changing environment (8). As a maladaptive response to the acoustic environment, noise sensitivity should be associated with lower resting HRV through a decrease in parasympathetic dominance.

3.2 Method

A total of 103 university students and members of staff volunteered: 37 males (M_{age}=36.2 years, SD=14.28) and 66 females (M_{age}=33.34 years, SD=12.83). Noise sensitivity was measured using the same instrument and procedure described in Study One. Participants were put into a supine position, electrodes attached and recording equipment primed, and a resting ECG obtained for a period of 10 to 15 minutes. Cardiac signals were measured at rest, with all recordings undertaken between 11:00 AM and 2:00 PM. The equipment and processes employed to obtain the ECG were identical to those described in Study One. No corrections for respiratory factors were implemented as baseline ECGs collected at rest are not vulnerable to respiratory artifacts.

Pre-processing of the ECG and the determination of inter-beat intervals was performed using ECGLab, which identified QRS complexes and removed ectopic beats. Kubios (v.2) HRV analysis software calculated all HRV indices across a ten minute period of the ECG. Time domain measures of HRV included estimates of overall HRV such as SD-NN, the standard deviation of the normalised inter-beat interval (NN), and rMSSD, the root mean square of successive differences in NN. In the frequency domain, power spectral analysis was performed using a fast Fourier transform algorithm. The normalised power (NP) spectral densities for two frequency bands, 0.04 to 0.15 Hz (low frequency: NP-LF), and 0.15 to 0.40 Hz (High frequency: NP-HF) were calculated by spectral integration. The NP-HF bandwidth is thought to be representative of parasympathetic modulation, while the NP-LF bandwidth represents sympathetic function. The frequency domain parameters (NP-LF, NP-HF) were subjected to a Briggsian logarithmic transformation to correct for positive skew. The ratio of the two, NP-LF/NP-HF is thought to provide a meaningful index of overall autonomic balance, that is, the balance between the sympathetic and parasympathetic nervous systems which reflects physiological arousal. Finally, two nonlinear indices calculated with reference to the line of identity in a Poincare plot, SD1 (reflecting parasympathetic modulation of HR) and SD2 (reflecting sympathetic modulation of HR), were calculated. .

3.3 Results and Discussion

Correlation analysis was used to assess the relationship between HRV and global noise sensitivity, with a number of significant coefficients noted (Table 1). Note the effect of removing the sleep-items from the NoiSeQ, evident in Table 1 (Row 2: 'Wake NS'), with a slight increase in the magnitude of the correlation coefficients. Evident for both measures of NS are small but significant correlations across both the time and frequency domain, and nonlinear, HRV indices. The third row in Table 1 contains the seven sleep-related items from the NoiSeQ, with no significant correlations noted. Exploratory analyses also revealed strong and significant correlations between noise sensitivity measures and age, and HRV indices and age. Subsequently, controlling for age removed all of the significant relationships reported in Table 1. Figure 2 plots mean standardised scores (*z*-scores) as a function of HRV indices for three levels of noise sensitivity. Greater HRV, as reflected by the time domain measures, is associated with lower noise sensitivity, with two-of-three measures significantly different between the low and high sensitivity groups ($p<.05$). No statistical significance was found when the frequency domain measures were tested, though the trends were as expected, with parasympathetic dominance (NP-HF) associated with the low noise sensitivity group, and higher sympathetic activity and autonomic balance associated with high noise sensitivity. Statistical significance was, however, found with the two nonlinear measures SD1 and SD2, with both measures differing between the low and high noise sensitivity tertiles.

Table 1: Partial correlation coefficients (controlling for mean heart rate) for measures of noise sensitivity (rows) and HRV indices (columns 2 to 9).

	SD-NN	**RMSSD**	**NN50**	**NP-LF**	**NP-HF**	**NP-LF/HF**	**SD1**	**SD2**
Global	-0.267**	-0.256**	-0.226*	0.219*	-0.42	0.047	-0.257**	0.261**
Wake NS	-0.298**	-0.302**	-0.316**	0.178*	-0.177*	0.284**	-0.296**	0.288**
Sleep NS	-0.114	-0.087	0.019	-0.073	0.073	-0.047	-0.087	0.118

* $p<.05$, ** $p<.001$

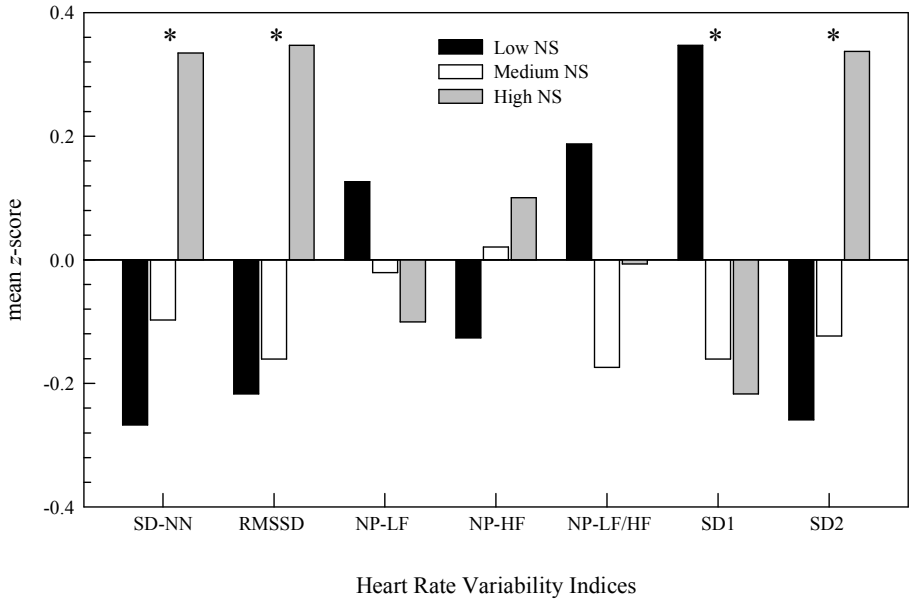

Figure 2 – Mean standardised scores for HRV variables across NS-Global tertiles. Asterisks represent significant differences ($p<.05$) between low and high noise sensitivity group means. Note that no significant differences were noted when the medium noise sensitivity group was compared to either the low or high groups.

The main finding in Study Two is a relationship between noise sensitivity and HRV indices. Pertinently, as HRV (e.g., SD-NN and rMSSD) and parasympathetic dominance (e.g., NP-HF and SD1) decreases, and sympathetic activity (e.g., NP-LF/NP-HF and SD2) increases, so too do self-report levels of global noise sensitivity. Furthermore, these findings concur with Thayer and Lane's (8) model. Noise sensitivity may be explained by a hypoactive parasympathetic response, and a hyperactive and sustained sympathetic response, due to an uncoupling of the autonomic nervous system and the amygdala-prefrontal circuits that interpret stressful stimuli and enact the appropriate stress response. The result is that the sympathoexcitatory circuits get caught in a positive feedback loop leading to hyper-vigilance and misattribution that then produce maladaptive cognitions (e.g., noise annoyance).

4. STUDY THREE: ALPHA PERSISTENCE

4.1 Introduction

Ando (6) proposes that subjective responses to environmental noise are correlated with the persistence of brain activity. Electroencephalograms (EEG) allow the brain's cortical activity to be inferred by recording minute electrical signals from the scalp. Alpha activity within the EEG coincides with a particular state of the brain, notably a relaxed mental state, and is associated with feelings of pleasantness and comfort. The persistence of alpha activity following the presentation of a sound can be documented using autocorrelation functions. If noise sensitive individuals have a predisposition to attend to sounds and judge them more negatively relative to non-sensitive individuals (1), then noise sensitivity should be associated with a lower persistence of alpha waves following sound presentation. Study Three sought evidence to support this proposition.

4.2 Method

Study Three involved 30 participants (16 female), aged between 18 and 44 years old (M_{age} = 24.77 years, SD = 5.47). Noise sensitivity was measured using the same instrument and procedure described in Study One. All EEG recordings were conducted in an electrically shielded room (Belling Lee – Model L3000) using128-channel Ag/AgCl electrode nets (Electrical Geodesics Inc.). Electrode nets were set according to the 10/20 international system of electrode placement, and different net sizes

accommodated differences in head size across participants. The EEG signals were recorded continuously at a sampling rate of 250 Hz (0.1 –100 Hz analogue bandpass) using Electrical Geodesics Inc. amplifiers (200 MΩ input impedance). During recording electrode impedances were kept below 40kΩ. All EEG signals were acquired using a common vertex (Cz) reference and were obtained using Net Station (v. 4.2) on an 8-core Apple Mac Pro workstation.

After placement of the electrode net, participants were seated 57 cm in front of a computer monitor and instructed to remain as still as possible and to passively listen to sounds. A total of 15 digitized sounds, each three seconds in duration, were prepared using LabView (v.8.5). These stimuli were modified from three sound types: a C major seventh piano chord (hereon "chord"), a 100 Hz low-pass filtered burst of white noise (hereon "noise"), and a 50 Hz low frequency sinusoid (hereon "tone"). To create sounds likely to differ in annoyance, the modulation frequency of each of the three sounds was set to one of five modulation depths: 0, 0.25, 0.50, 0.75 and 1 Hz, giving a total of 15 sounds (3 sounds by 5 modulation depths).

All EEG raw files were segmented, according to event markers, into 3000-ms epochs, including a 1000-ms pre-stimulus baseline. These EEG files were referenced to the average reference before filtering with a Butterworth band-pass filter (alpha band: 8 Hz to 12 Hz). The EEG data for each participant were then grouped according to the 15 sounds, and rereferenced to the average reference for each sound across all participants. The electrodes over the frontal-central area were selected for the averaging process. The voltages for these electrodes were averaged according to the sound types judged least and most annoying for the noise resistant and the noise sensitive participants. All grand averages were computed and the averaged autocorrelation from time zero were calculated according to 600 ms post-stimulus onset.

4.3 Results and Discussion

Autocorrelation functions were computed to examine the persistence of alpha activity across the least and most annoying sound types for both noise resistant and the noise sensitive groups. Figure 3 shows the amount of alpha persistence after the presentation of sound stimuli, where the two valances (i.e., least and most annoying) were averaged for each group. In terms of alpha persistence, the noise resistant group produced two functions that mostly overlap, while for the noise sensitivity group alpha persistence appears to differ between least and most annoying sounds. Between the two sensitivity groups, alpha activity is sustained more in the noise resistant than the noise sensitive group, with functions diverging from the 50-ms delay onwards.

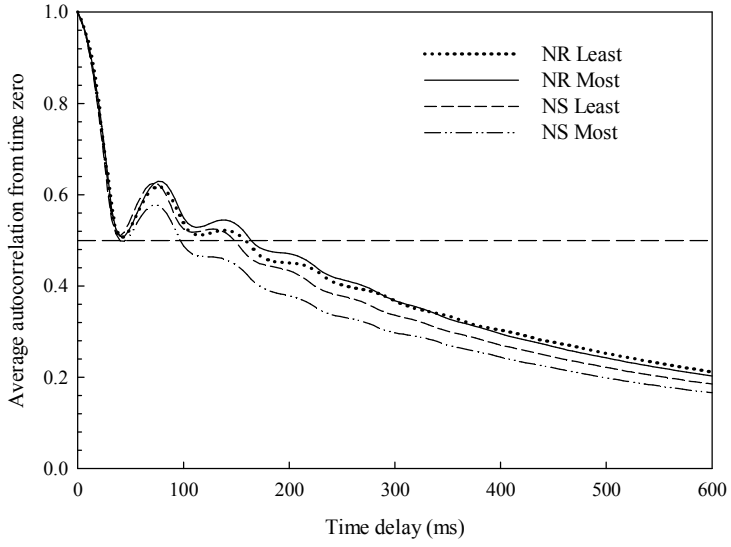

Figure 3 – Alpha persistence for the least and most annoying sound samples for noise resistant and noise sensitive participants.

Alpha rhythms are associated with states of mental and physical relaxation, and are inversely correlated with arousal level. Additionally, weak alpha activity has been correlated with increased cognitive load, and decreases in alpha persistence indicates the activation of neural networks involved

with cognitive processing. In the context of the present study, it would be expected that diminishing alpha persistence reflects increases in subjective annoyance. Because the magnitude of annoyance to sound should be greater in noise sensitive individuals, a relationship between noise sensitivity and alpha persistence is expected. The data in Figure 3 largely conform to this expectation, with the greatest reductions in alpha persistence occurring in the noise sensitivity group, and furthermore, for the sound judged most annoying. This reduction in alpha persistence could reflect either an increase in cognitive arousal driven my negative evaluations of the sound, undesired directed attention which also a defining characteristic of noise sensitivity, or both.

5. Study 4: Sensory Gating

5.1 Introduction

Hypersensitivity to environmental stimuli, including sound, may be due to early information processing deficits in the brain (9). Clinical evidence suggests that patients with schizophrenia or brain injury are unable to filter out (or 'gate') irrelevant sensory information from the immediate environment, and experience sensory overload as a result. Sensory gating refers to an internal filtering mechanism regulating incoming sensory events from the environment (9), a process largely occurring in the thalamus. Sensory gating is commonly measured using the paired-click paradigm (9), by which pairs of identical auditory clicks are presented, and the brain's response to each measured and compared. The P50, a positive deflection in the electroencephalogram (EEG) occurring circa-50 ms following the termination of a click, is commonly used in the estimation of sensory gating. The P50 represents a protective mechanism preventing the flooding of higher cortical areas with unnecessary sensory information (10), enabling an individual to process updated sensory information.

It is feasible that noise sensitivity could reflect impaired sensory gating. Therefore, greater perceived annoyance reported by noise sensitive individuals could be connected to difficulties in inhibiting unnecessary sensory inputs. Study Four employed an orthodox paired-click paradigm to test the directional hypothesis that average sensory gating will be greater for lower than higher noise sensitivity groups. Additionally, while sensory gating is largely considered pre-attentive, P50 gating could be modified by top-down attentional processes, and because variability across noise sensitivity may be explained by attentional differences, the role of attention was also examined (10).

5.2 Method

The same 30 participants who participated in Study Three also volunteered for Study Four. Noise sensitivity was measured using the same instrument and procedure described in Study One, as were all EEG recordings. For the paired-click paradigm, two different types of click (standard and deviant) were created in LabView. The standard click was a 1000 Hz tone, and the deviant click a 2500 Hz tone. The duration of each standard click was 4 ms with 1-ms rise and fall times (cos2), while the deviant click was 8 ms with a 2-ms rise and fall time. A standard pair of clicks consists of two identical standard clicks, and a deviant pair of clicks consists of one standard click and one deviant click. For the deviant pair, the standard click is always presented first, followed by the deviant click. For both standard and deviant click-pairs, the inter-click interval was 500 ms, while the inter-pair interval was drawn from a rectangular distribution between 3000 ms to 5000 ms. The clicks were scaled to 70 dB SPL, and were delivered using the same process and equipment as described in Study Three.

There were three conditions: passive listening, auditory attention, and visual attention. During the passive listening condition, participants fixated on a cross presented on the computer monitor while listening to a series of 80 pairs of standard clicks. In the auditory attention condition, 80 pairs of standard clicks and 20 pairs of deviant clicks were presented to the listener in random order. Participants identified the occurrence of a deviant click by pressing a key within 2000 ms of click presentation. Response accuracy was recorded, and participants received no feedback. The visual attention condition was similar to the auditory attention condition but with a silent movie playing on the computer monitor. Participants were asked to concentrate on the movie and to answer questions about the content of the movie at the end of the experiment. Response accuracy was again recorded.

To assess differences in attention across low and high sensitivity groups, response accuracy in either the auditory or visual attention conditions was compared using independent samples *t* tests. The EEG data were subjected to event-related potential (ERP) analyses, for both the first and the second click, within all conditions. For each click, all EEG recordings were segmented into 700-ms epochs (including a 100-ms pre-stimulus baseline). Event related potentials for the first and the second

standard clicks were re-referenced to the average reference within each condition, for each participant relative to their group membership (i.e., low or high noise sensitivity). Thus ERPs from individual participants in the same sensitivity group were combined to produce grand-averaged ERPs for each condition. Magnitude of the P50 for each click was calculated by averaging the amplitudes 4 ms before and after the largest peak within the time window of interest. The P50 was scored as the largest positivity between 60 to 96 ms after the presentation of the click. Electrodes surrounding the fronto-central scalp distribution were selected for analyses. For any one participant, sensory gating was calculated by subtracting the averaged amplitude of the second click from that of the first click, with difference expressed in microvolts. The more positive the amplitude difference, the more efficient the sensory gate.

5.3 Results and Discussion

Figure 4 plots estimates of sensory gating across the three experimental conditions for both noise resistant and noise sensitive groups. For the passive listening task, greater sensory gating is evident in the noise resistant group though the differences are not statistically significant (p<.05). For the auditory attention task, sensory gating is reduced in both groups and the difference between them significant (p=.032). For the visual attention task the differences in sensory gating between the two sensitivity groups is non-significant (p>.05), with the non-sensitive group trending towards greater levels of gating.

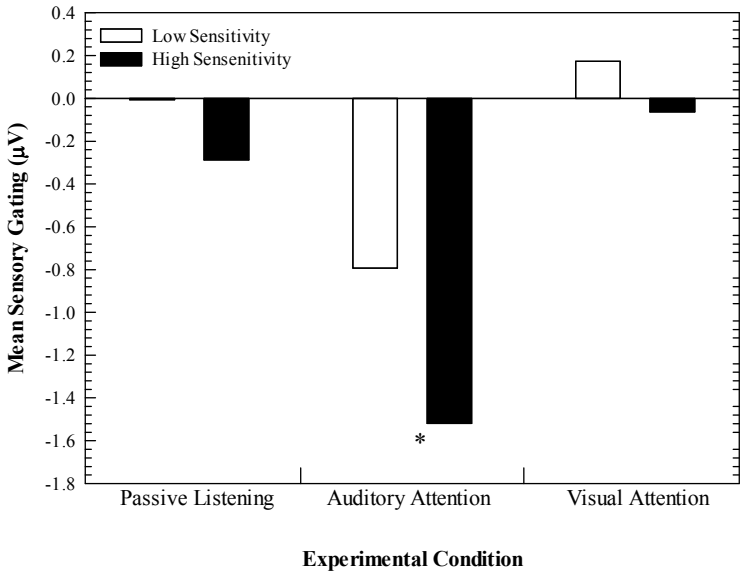

Figure 4 – Degree of sensory gating as a function of noise sensitivity for three experimental conditions. The asterisk represents a significant difference between low and high noise sensitivity groups for the auditory attention condition.

Averaged evoked potentials for the noise sensitive and resistant groups are displayed in Figure 5, for the first (top row) and second (bottom row) clicks. For the first click a general trend of pronounced early ERP components (< 300 ms) for the noise sensitivity group are noted, along with evidence of prolonged peak latencies. Early ERP components can be modified by attentional processes, and augmented early components in the presence of distracting noise likely reflect increased cognitive load (12). Statistical testing revealed that for the N100, whose amplitude depends upon features of the stimulus as-well-as selective attentional processes, the mean difference scores between the first and second click was significant across the two sensitivity groups for the auditory attention condition (p=.043). Unlike the noise resistant group, the magnitude of the N100 for the noise sensitive group was relatively invariant across the two clicks, suggesting greater attentional resources were required to sustain vigilance. Interestingly, for the P200, also modulated by attentional processes, the mean difference scores between the first and second clicks were greater for the noise sensitivity group (p=.018) in the passive listening task, with the P200 strongly attenuated following the second click.

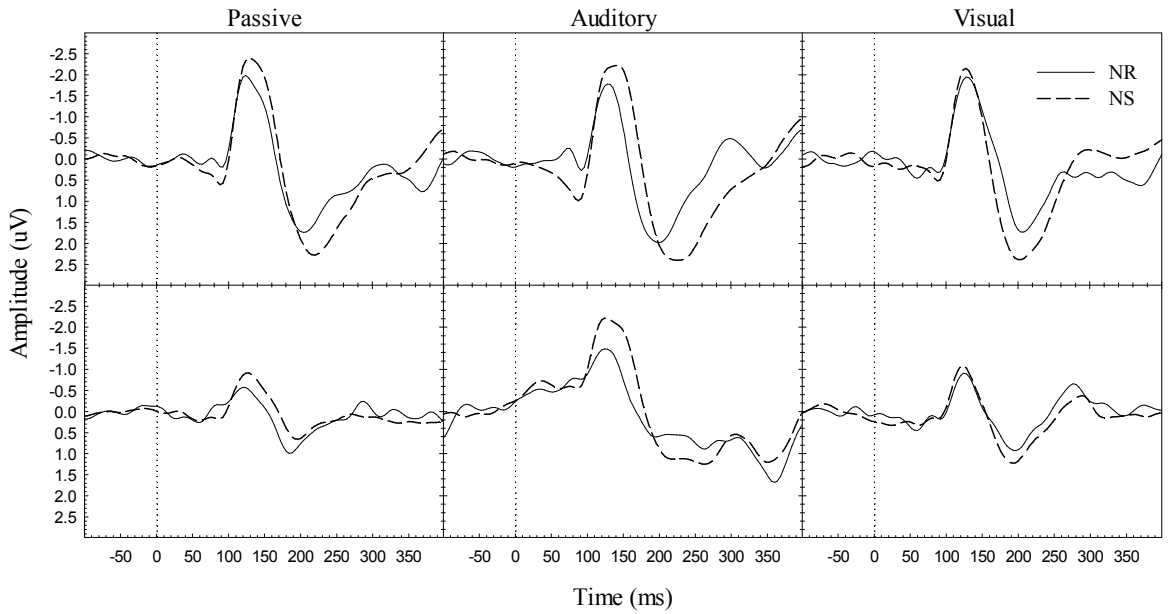

Figure 5 – Grand averages of ERP amplitudes for the first (top row) and the second (bottom row) clicks across three attention conditions. Solid and dashed curves are the noise resistant and noise sensitive group respectively. The dotted vertical line divides the pres-stimulus and post-stimulus intervals. The P50 (between 60 to 96 ms), N100 (between 100 to 160 ms), and P200 (between 164 and 264 ms) components can be inspected by eye.

6. DISCUSSION

The Across four studies we adapt a number of psychophysiological approaches that hitherto have not been applied to the noise sensitivity context. The analysis of heart rate data in Study One and Two indicated that autonomic response may covary with noise sensitivity. An interesting finding emerging from Study 2 is a difference between waking and sleep-related noise sensitivity, supporting physiological findings (4) indicating that the two types of 'noise sensitivities' may be independent. Study Three, employing EEG, demonstrated the utility of the alpha persistence approach, while the data collected in Study 4 suggested that differences in sensory gating may exist between noise sensitivity extremities. Overall, the data suggests that further investigation into the electrophysiological correlates of noise sensitivity is warranted, not only in terms of etiology, but also as potential measures in diagnostic and treatment outcome indicators.

These findings present an omnibus approach to investigating the biological substrates of noise sensitivity, filling a gap in the clinical literature that consistently suggests the potential importance of biological mechanisms (e.g., 11), and yet supports such assertions with scant empirical data (but see 1). As such it is difficult to compare these findings with previous studies as the few electrophysiological investigations into maladaptive responses to auditory stimuli do not sufficiently operationalise or even measure noise sensitivity, with the exception of sleep-related studies. For example, Pripfl et al., (12), utilising EEG indices to study the relationship between noise sensitivity and fMRI scanner noise, operationalised noise sensitivity on the basis of those who reported they an annoyance response and those who did not. Such an approach fails to disentangle those with trait noise sensitivity to those merely exhibiting a state of annoyance to an unpleasant noise. Issues of measurement aside, Pripfl et al., (12) reported differences in early EEG components (N1, N2 and P2) between their sensitive and non-sensitive groups, concurring with the findings of Study 4.

Biologically, and as a generalisation, noise sensitivity may manifest the sensitisation of neural networks involved in the allocation of attentional resources and the modulation of arousal. Subcortical (i.e., limbic) plasticity has been implicated in both sensitisation and its opposite, habituation, thus offering potential explanations as to why individuals differ in their sensitivity to environmental stimuli (13). Of the four theoretical approaches described in the current study, the neural-visceral integration approach (re: Study 2) offers a holistic approach that accounts for both cortical (i.e., attentional) and subcortical (i.e., arousal) processes. The findings from all four studies are consistent with the predictions of this model, which offers a promising approach in future study. Furthermore, given its

multi-component and network focus, the model can also account for variability across clinical populations in terms of mechanism. For example, there is evidence that those with autism have deficits in auditory scene analysis, a process localised to the auditory cortex, though the subsequent experience of sensory overload may be indistinguishable from that induced by the impaired sensory gating characteristic of schizophrenia.

There are a number of caveats concerning the validity of the findings. Firstly, while noise sensitivity is a common symptom across a number of clinical syndromes, the sample utilised in the current study could not be considered from a clinical population. Consequently, the true differences between noise resistant and noise sensitive individuals on the electrophysiological measures reported herein may be attenuated. That said, there is no argument as to why noise sensitivity cannot exist in non-clinical populations, as by definition clinical syndromes are made up of clusters of symptoms, and when present in isolation these symptoms present as traits. Secondly, and typical of exploratory studies of this type, the sample size was modest and the findings will require further independent corroboration. Thirdly, existing knowledge would also benefit from further investigation of covariates including age, gender, and trait anxiety, the latter which may sustain arousal and induce a general reactivity to sensory events.

To conclude, a number of viable approaches to investigating the electrophysiological correlates of noise sensitivity exist, with our findings suggesting that there a number of measures sensitive enough to discriminate across individuals and groups differing in their sensitivity to noise. Future research should focus on refining these measures to inform the development of clinical noise sensitivity measures able to aid diagnosis and treatment not only of noise sensitivity in isolation, but also as part of a symptom cluster characterising a clinical syndrome.

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