

## Studying noise sensitivity on the brain level

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### ABSTRACT

Noise sensitivity (NS) is a common trait predicting noise annoyance. We are still lacking a model for the mechanisms of NS. Many studies have suggested psychological origins but some recent data suggests a neural source for NS. Our study investigating neural auditory processing in relation to NS with combined electroencephalography (EEG) and magnetoencephalography (MEG) found that noise sensitive individuals have compromised pre-attentive encoding and discrimination of sound features as compared to noise-resistant individuals. Subjects were presented with a fast musical multifeature paradigm that included six types of sound feature deviations. Noise sensitive subjects had smaller P1 amplitudes than less sensitive subjects, suggesting that they may have difficulties with sound feature encoding. They also demonstrated diminished mismatch negativity (MMN) responses to feature deviations, especially to the one with increased noisiness. Thus, NS is specifically related to neural mechanisms for processing of noise, but not to other features of sound. Shepherd et al. (2016) found that noise-sensitive individuals exhibit less sensory gating than noise-resistant individuals. These results indicate that NS has its origins in primary auditory functions of the central nervous system.

### INTRODUCTION

Noise sensitivity (NS) is a stable and common trait. It refers to physiological and psychological internal states of any individual, which increase the degree of reactivity to noise in general [1]. It is a predictor of noise annoyance [2, 3]. No strong evidence for an auditory component at threshold levels in NS has been found, since it has not been related to auditory acuity [2, 4, 5, 6]. However, NS has been associated with self-reported hearing disability [5]. NS is not a synonym of hyperacusis, which can be defined as loudness related hypersensitivity involving a perception of discomfort experienced at sound levels lower than the average loudness discomfort level.

Noise sensitive individuals are more prone to noise effects like impaired cognitive performance [7], sleep disturbance [8], cardiovascular disease [9], obesity [10]. Kishikawa et al. (2009) found a significant correlation between self-reported health and noise exposure in the noise sensitive group, whereas no significant correlation was observed in the non-sensitive group [11]. Noise may prevent individuals with high NS from achieving the same work outcome compared to less sensitive individuals leading to psychosomatic, neurotic and other difficulties while individuals with lower NS may be expected to better adapt to noise during mental performance [7]. NS may also be a potential risk factor for disability retirement as evidenced by a Finnish study in which the total disability retirement among noise sensitive individuals was 41 % higher compared with non-sensitive individuals. NS was associated with disability retirement independently of familial background and genetic factors [12].

Yet, we are still lacking a model for the mechanisms of NS. Even if some studies have suggested psychological origins also, biological factors may be related with NS. Along this line, a Finnish study has shown that NS aggregates in families meaning a higher frequency of NS in the first-degree relatives compared to the general population. The estimate of heritability of NS is 36 % [13].

There are some recent studies investigating the neural mechanisms underlying NS. We are reviewing here the latest studies on noise sensitivity on the brain level.

### **Studies on the neural mechanism of noise sensitivity**

Electroencephalography (EEG) measures spontaneous electrical brain activity whereas the Event-Related Potential (ERP) is the portion of the EEG that is time-locked to specific sensory, motor or cognitive stimuli or events. The early ERP waves peaking roughly within the first 100 ms after stimulus are termed sensory or exogenous as they reflect basic encoding of the physical parameters of the stimulus. Later ERPs reflect subsequent operations on the stimulus and are termed cognitive or endogenous. The mismatch negativity (MMN) is a negative component of the auditory event-related potential (ERP), which is usually peaking at 100–250 ms from stimulus onset. The MMN is a marker of sound-discrimination accuracy [14, 15].

In an EEG study of Lee et al. (2012) a tendency to be aroused by noise easily, regardless of the magnitude of annoyance, was evident in noise sensitive participants while non-noise sensitive were aroused only at the presence of the most annoying sounds. Among non-noise sensitive participants a protective effect against noise was found [16].

In another EEG study on the effects of fMRI scanner noise on noise sensitive women, Pripfl et al. (2006) found that noise led to more pronounced N1 and P2 waves but attenuated N2, which most likely reflects different attentional requirements as early ERP components are influenced by attention. They also found that during task processing the slow cortical negative shift was significantly attenuated with annoyed subjects compared to not annoyed subjects. Thus emotion-related subcortical structures may be responsible for the observed difference [17].

Shepherd et al. (2014 and 2016) reported using ERPs with passive listening, auditory attention and visual attention tasks that for the passive listening task, greater sensory gating was evident for the noise resistant group, however, the differences were not statistically significant. For the auditory attention task, sensory gating was reduced in both groups and the difference between them was significant. For the visual attention task the differences between the two sensitivity groups were non-significant. For the paired-click paradigm, two different types of click (standard and deviant) were created in LabView. Participants identified the occurrence of a deviant click by pressing a key within 2000 ms of click presentation. For the

first click a general trend of pronounced early ERP components (<300 ms) were noted for the noise-sensitive group along with evidence of prolonged peak latencies. For the N100 the mean difference scores between the first and second click was significant across the two sensitivity groups for the auditory attention condition. Unlike the noise resistant group, the magnitude of the N100 for the noise sensitive group was relatively invariant across the two clicks, suggesting that greater attentional resources were required to sustain vigilance. For the P200 the mean difference scores between the first and second clicks were greater for the noise-sensitive group in the passive listening task, with P200 strongly attenuated following the second click [18, 19]. Shepherd et al. (2014 and 2016) concluded that noise-sensitive individuals exhibit less sensory gating than noise-resistant individuals.

Our recent study (Kliuchko et al. 2016) addressed whether noise sensitivity is manifested in the way the brain processes sound features. We investigated neuronal sound processing in relation to NS with combined EEG and magnetoencephalography (MEG). Subjects were presented with a musical multifeature Mismatch Negativity (MMN) paradigm that included six types of sound feature deviations. The data set consisted of 71 MEG recordings (34 men, 37 women, age range 19–51, M = 28.48) and 66 EEG recordings (32 men, 34 women, age range 19–51, M = 28.67). We found that NS is related to compromised sound feature encoding. That was manifested in smaller P1 amplitudes in noise sensitive individuals than in non-sensitive ones. The auditory system of noise sensitive individuals was also less responsive to new sound features introduced among repetitive sounds, especially if the novel sound is noisier than the rest. That was evidenced from generally diminished MMN and to the noise deviant in particular. Hence, NS was specifically related to automatic neural processing of noise, but not to sound features like pitch, location or intensity. It may be harder for noise-sensitive people to build a prediction about changes in a varying soundscape, and their auditory system might “tune down” its responsiveness to sounds in order to protect itself from overreacting to noise. These results indicate that NS has its origins in primary auditory functions in the brain [20]. However, we need further studies to investigate whether we have discovered something that is the reason why people are noise sensitive or is this a result of the brain’s contractions against excessive noise.

## **CONCLUSIONS**

We are still lacking a comprehensive model for the mechanisms of NS. Some earlier studies have suggested psychological origins for NS. However, there are recent studies addressing the neural mechanisms of NS. Noise sensitive individuals demonstrate compromised sound feature encoding as compared to noise-resistant individuals. NS is specifically related to processing of noise, but not to features of sound like pitch, location or intensity. Thus, NS has its origins in primary auditory functions in the brain. To understand better the neural mechanisms, we need also MRI studies investigating the association of noise sensitivity with the structural anatomy of auditory and limbic brain areas.

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