Studying the origins of noise sensitivity – negative affect or biological factors

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Summary
Noise sensitivity (NS) is currently well described but its etiology has not been sufficiently explained. Noise-sensitive individuals are more likely to attend to sound and evaluate it negatively, perceive it as annoying, have stronger emotional reactions to sound and greater difficulty habituating to sounds. NS describes a vulnerability to negative health effects associated with noise exposure. In this paper, we review our most recent studies with respect to the origins of NS. The most common explanation has been that NS reflects negative affectivity, a dispositional tendency to negatively evaluate situations and the self. However, results from the study of Shepherd et al. (2015) failed to support the notion that, by itself, negative affectivity explains sensitivity to noise. There are explanations of NS which are based on cognitive processes by which noise-induced memory and attentional deficits are leading to annoyance or distress. NS has also been attributed to hypervigilance to noise sources due to fear and anxiety, significantly impacting annoyance. More recent studies have shown evidence of a neural mechanism for NS. The study of Kliuchko et al. (2016), using magneto- and electroencephalography (MEG/EEG), suggests biological origins for NS. NS was specifically related to neural mechanisms linked to the processing of noise, but not to other features of sound. Thus NS has its origins in primary auditory functions of the central nervous system. A study using magnetic resonance imaging (MRI) by Kliuchko et al. (2018) found that individual differences in NS seem to be associated with the structural organization of brain areas playing a role in auditory perception,

interoception and, the processing of emotions and salience. Thus, the findings of these recent studies provide evidence of biological origins and a neural mechanism for NS.

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1. Introduction

Noise sensitivity (NS) is well described but not yet sufficiently explained. It refers to the physiological and psychological internal state of any individual, which increases the degree of reactivity to noise in general [1]. It describes a vulnerability to the negative health impacts associated with environmental noise exposure. It is a common and a relatively stable trait [2,3] which has been found to be either equally present in men and women or in some cases, higher in women, on the basis of their NS score. NS is different to hyperacusis which is defined as an abnormally strong reaction to sound occurring within the auditory pathways [4] when normally tolerable sounds are perceived as excessively loud. Patients with hyperacusis may have normal hearing or they may have impaired hearing, whereas NS has not been associated with auditory acuity [5-7]. Hyperacusis is a common symptom in patients with tinnitus, William's syndrome, autism, and other neurologic diseases.

We are still lacking a comprehensive model for the mechanism of NS. By definition, NS manifests negative evaluations of noise and the challenge is to determine the cause of these negative evaluations. At the moment, there are several explanations of the etiology of NS found in the literature (Figure 1.).

The most common explanation found in the epidemiological literature specifically is that NS is explained by negative affect [8,9], a dispositional tendency to negatively evaluate situations and the self. Individuals high in such traits may report a greater sensitivity to other sensory stimuli, such as smell, bright light and pain. However, research investigating the relationship between NS and sensitivity to stimuli associated with other sensory modalities has not always supported the notion of a common underlying trait, such as negative affect, driving them. NS and multiple chemical sensitivity seem to be different entities [10].

The most common explanations for NS in the clinical literature are explanations based on bottom-up cognitive processes by which noise-induced memory and attentional deficits lead to annoyance or distress. These cognitive explanations rely upon information-processing models of auditory distractors [11]. Noise-induced interference of cognitive processes has been well described. NS exerts a negative effect on cognitive functions, such as attention, working memory and episodic recall [12].

NS can also have biological origins. Along this line, a Finnish study has shown that NS probably has a genetic component. It aggregates in families and the estimate of heritability of NS is 36 % [13]. Recent studies have shown evidence of a neural mechanism of NS. The study of Kliuchko et al. (2016), using magneto- and electroencephalography (MEG/EEG), linked NS to the primary functions of the central auditory system. Namely, high NS was found related to the processes of sound encoding and discrimination of sound properties [14]. A further study using magnetic resonance imaging (MRI) by Kliuchko et al. (2017) found that individual differences in NS seem to be associated with the structural organization of brain areas playing a role in auditory perception, interoception (the sense of the physiological condition of the body) and the processing of emotions and salience. In this paper we review our latest studies on the negative affect hypothesis on NS, the neural mechanism of NS and the morphological markers associated with NS [15].

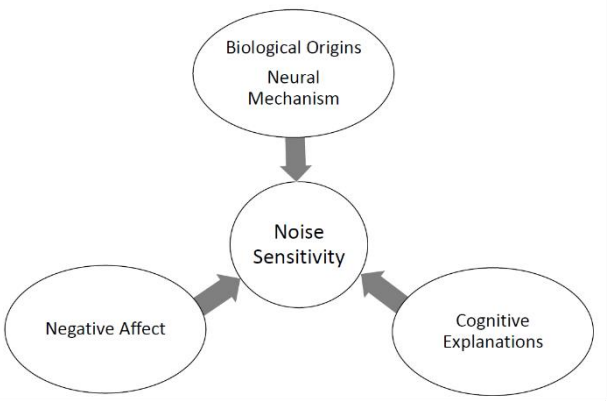

Figure 1. Possible mechanisms of noise sensitivity

2. The negative affect hypothesis of NS

The negative affect hypothesis states that noise annoyance is not especially stimulus-oriented, rather

that sound only has to be audible to be annoying. Thus, sound level or other qualities such as modulation would not always be useful predictors of noise annoyance. The negative affect approach would predict annoyance responses to be uniform across sensations, irrespective of the sensory modality of origin. The negative affect hypothesis, which is usually described at the psychological level, can also have a neurological basis.

The results from a recent study of Shepherd et al. (2015) failed to support the notion that, by itself, negative affectivity explains sensitivity to noise. The findings did not provide support for the negative affect hypothesis, but they might support the so-called "noise vulnerability hypothesis", which asserts that noise has its greatest impact on vulnerable (i.e., noise-sensitive) individuals. Negative affect is unlikely to be a general cause of NS, and other mechanisms are worthy of examination [16].

3. The Neural Mechanism of NS

Kliuchko et al. (2016) addressed whether NS is manifested in the way the brain processes sound features and investigated neuronal sound processing in relation to NS in a combined EEG/MEG study. Two components of auditory event-related potential were evaluated: the P1 component, which reflects early stages of sound feature encoding on the cortical level, and the mismatch negativity (MMN), which reflects preattentive detection of stimulus changes. The MMN is elicited to a deviant sound occurring among standard sounds. In study by Kliuchko et al. (2016), subjects were presented with a musical multifeature MMN paradigm that had six types of sound feature deviations, including a sound with increased noisiness.

The study found a compromised representation of standard sounds in the auditory cortex as was manifested in the form of smaller P1 amplitudes in noise-sensitive compared with non-sensitive individuals. This deficiency in encoding auditory information was followed by attenuated response to new sound features introduced among repetitive sounds, especially if the novel sound was noisier than the rest, as evidenced from the generally diminished MMN and to the noise deviant in particular in participants with high NS as compared to the least sensitive participants [14].

Hence, NS was specifically related to the automatic neural processing of noise, but not to sound features such as 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 is connected to the brain's primary auditory functions [14]. However, we need further studies to investigate whether the reported findings are a discovery of perceptual deficits at the origin of NS or whether they are due to a maladaptive inhibition of response to auditory sensory input that developed in the central nervous system because of high sensitivity to environmental noises.

4. NS is visible in brain structures

The MRI study of Kliuchko et al. (2017) investigated morphological markers associated with NS. For that, grey matter volume, cortical thickness, and other anatomical parameters were measured from anatomical MRI images and correlated with NS. The study focused on the primary and non-primary areas of the auditory cortex involved with sound processing, and other areas such as insular cortex, amygdala and hippocampus, which are playing role in the prediction of aversive stimuli, evaluation of emotional salience and control of autonomic stress reaction in response to auditory stimuli [15].

The volume of each area were proportionally adjusted for the intracranial volume to control for differences in head size and corrected for age effects on the cortical organisation. The results show that NS was associated with the volume of grey matter over the selected brain areas, so that higher NS corresponded to larger volumes. In particular, this appeared in the left Heschl's sulcus, bilateral hippocampi and temporal poles, as well as the right anterior insula. However, the observed associations did not survive a correction for multiple comparisons and are only suggested as candidate areas for an involvement with NS [15].

Greater grey matter volume in auditory sensory areas in people with high NS may suggest that it requires them more neural resources to be involved in dealing with sound. Noise sensitivity may be related to an extent of self-awareness about the sensations that noise induces in noise-sensitive individuals - the anterior part of the right insular cortex, in which this study observed a tendency to increase in NS, is known to be important for matching external sensory information with the internal state of the body and bringing it to one's conscious awareness. The relation of hippocampus

to NS, in turn, could be because of its role in contextual processing and modulation of emotions [15].

Taken together, the study by Kliuchko et al. (2017) suggest that NS is related to the structural organisation of a brain network related to sensory and emotion aspects of sound processing. This work brings new insight into the physiological mechanisms of NS.

5. Discussion

We have been lacking a comprehensive mechanism for NS. In the past, the most common explanation has been that NS reflects negative affectivity. The results from a recent study of Shepherd et al. (2015) failed to support the notion that, by itself, negative affectivity explains sensitivity to noise. A specific future research challenge, both in terms of theory and empirically, is the disentanglement of negative affect, global sensitivities, and other potential mechanisms of NS. Specific subgroups also need to be studied as they may represent distinct etiologies in relation to their sensitivity to noise. However, negative affect is unlikely to be a general cause of NS, and other mechanisms are worthy of examination.

NS may have biological origins and we have recent evidence for a neural mechanism. NS is related to compromised early sound feature processing and the neural discrimination of noisy sounds, but not of sounds with, e.g., deviant intensity or frequency (Kliuchko et al. 2016). Thus, the neuronal mechanisms of sound processing could be the key to understanding the origin of NS and worth a further investigation. However, we do not imply that alternations in the auditory brain functions are causal to NS. Previous studies have shown that subjects without peripheral hearing damage but continuously exposed to noise due to their occupation may exhibit subpathological changes in cortical responses to sounds, especially to noisy sounds [17,18]. It could be the case that the functional changes in the auditory system of noise-sensitive individuals occur because of susceptibility of their central auditory system to noise. We need further studies to uncover whether we have discovered something explaining why people are noise sensitive or whether it is a result of the brain's contractions in response to excessive noise.

An MRI study of Kliuchko et al. (2017) found that higher NS was associated with the greater grey matter volume in in the brain temporal regions, the hippocampus and the anterior part of the right insula. Thus, individual differences in NS seem to be associated with the structural organization of brain areas playing a role in auditory perception, sensory awareness and, in the processing of emotions and salience. This study did not find an association between the amygdala's volume and NS, though the luck of structural changes does not rule out a functional involvement of amygdala in NS, which should be addressed in further studies. The differences in the brain morphology related to NS could also be contributed from genetic factors. It is possible that noise-sensitive individuals are born with a predisposition for larger volumes of the primary auditory cortex, anterior insula, and hippocampus which could make them more prone to evaluating environmental auditory stimuli in an aversive manner [17]. A genetic component to NS has been previously indicated in a study by Heinonen-Guzejev et al. (2005).

Based on the observation of a change in the grey matter volume, the above-mentioned brain structures should be investigated further for their functional role in NS, as well as confirmatory investigations on the reported trends are called upon. Future studies should also address whether anatomical and functional connections between these brain areas are affected in NS.

6. Conclusions

The mechanisms of NS may be multifactorial in nature, with factors influencing the degree of NS acting either independently or interactively. Recent studies have shown strong evidence for a neural mechanism of NS.

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