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REVIEW PAPER

# OUTLOOK ON HYPERACUSIS AND MISOPHONIA IN AUTISM SPECTRUM DISORDERS (ASD)

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## Abstract

Individuals with autism spectrum disorder (ASD) frequently experience decreased sound tolerance (DST), which reduces their social interaction and engagement. The purpose of this perspective piece is to look at the gap in the literature concerning the possible genetic and functional bases for the comorbidity of ASD and two distinct types of DST, hyperacusis and misophonia.

**Keywords:** autism spectrum disorders • hyperacusis • misophonia • decreased sound tolerance

## NADWRAŻLIWOŚĆ SŁUCHOWA I MIZOFONIA W ZABURZENIACH ZE SPEKTRUM AUTYZMU (ASD)

## Streszczenie

Osoby z zaburzeniami ze spektrum autyzmu (ASD) często doświadczają obniżonej tolerancji na dźwięki (DST), co ogranicza ich interakcje społeczne i zaangażowanie. Celem niniejszego artykułu jest przyjrzenie się lukom w literaturze na temat możliwych genetycznych i funkcjonalnych podstaw współwystępowania ASD i dwóch odrębnych typów DST – nadwrażliwości słuchowej i mizofonii.

**Słowa kluczowe:** zaburzenia ze spektrum autyzmu • nadwrażliwość słuchowa • mizofonia • obniżona tolerancja na dźwięki

| Key to abbreviations | |
|---|---|
| ABR | auditory brainstem response |
| ASD | autism spectrum disorders |
| CBT | cognitive behavioral therapy |
| DBS | dysregulated behavioral symptoms |
| DPOAEs | distortion product otoacoustic emissions |
| DST | decreased sound tolerance |
| GAD | generalized anxiety disorder |
| MDD | major depression disorder |
| PTSD | post-traumatic stress disorder |
| SNP | single nucleotide polymorphisms |
| SPD | sensory processing disorders |

Autism spectrum disorders (ASD) is a neurodevelopmental disorder affecting communication and social skills and is often characterized by repetitive and restrictive behaviors [1]. Individuals with ASD frequently experience sensory processing disorders (SPD), with decreased sound tolerance (DST) being the most common [1–3]. Everyday sounds and noises considered tolerable to the general population, such as crowds, construction, and yelling, are reported as significantly heightened in individuals with autism spectrum disorder [2]. When exposed to such sounds, individuals with the condition may exhibit adverse reactions such as distress, irritation, and anger [4]. Two distinct types of DST include hyperacusis and misophonia. Both conditions are present in individuals with normal hearing thresholds [5], suggesting that peripheral hearing loss may not be a potential cause [2].

Misophonia is represented by an over-responsiveness to specific "triggering" sounds often produced by humans, such as sniffing, chewing, and coughing [6], or repetitive sounds like pen clicking or tapping [7]. On the other hand, individuals with hyperacusis have a reduced threshold for loudness discomfort, which increases their sensitivity to general everyday sounds and results in a decreased tolerance of them [8]. The sounds are often described as painfully loud and uncomfortable by those with hyperacusis, although they do not cause problems for most neurotypical individuals [4]. Individuals with hyperacusis may also have a higher prevalence of tinnitus, suggesting a possible relationship between the two conditions [9].

It is essential to understand the types of DST, considering they are highly prevalent in the ASD population. Scheerer et al. [2] found that DST leads to fewer opportunities for

**Table 1.** Prevalence of hyperacusis in the ASD population based on five studies

| Study | Participants | Age [years] | ASD category | Prevalence of hyperacusis [%] |
|---|---|---|---|---|
| Williams et al. (2021) [8] | metanalysis | NA | autism | 41–60 |
| Demopoulos & Lewine (2016) [10] | 60 (48 males, 12 females) | 5–18 | high–low functioning | 37 |
| Danesh et al. (2015) [9] | 55 (46 males, 9 females) | 4–42 | high-functioning (Asperger) | 69 |
| Rosenhall et al. (1999) [11] | 199 (153 males, 46 females) | children adolescents | autism | 18 |
| Rimland & Edelson (1995) [12] | 17 (11 males, 6 females) | 4–21 | unspecified | mild – 53<br>moderate – 24<br>strong – 18 |

autistic children and young adults to engage at home, at school, and in the community. In a meta-analysis, Williams et al. [8] reported the current and lifetime prevalence of hyperacusis in ASD to be 41–60%. **Table 1** summarizes a few studies of the prevalence of hyperacusis in the ASD population.

Despite the high prevalence, the etiology of both hyperacusis and misophonia is unclear. Various theories have been proposed to understand the reasoning behind the relationship of DST in this population. The theories extend to anatomical and physiological disruption, efferent pathways of the auditory system, genetic factors, and pharmacological causes [4].

Danesh et al. [4] described a variety of potential correlates of hyperacusis in the ASD population, such as anatomical differences in the inner ear. They indicated that 29% of autistic individuals had superior semicircular canal dehiscence (as reported in [13]). Superior semicircular canal dehiscence involves a window between the cranial cavity and the inner ear, causing those with the condition to perceive sounds extremely loudly. They cited another study [14] which examined the correlation between the stapedial acoustic reflex and loudness tolerance which showed significantly lower stapedial reflex thresholds and significantly delayed responses in young participants with autism. The contralateral suppression of distortion product otoacoustic emissions (DPOAEs) has also been studied in individuals with hyperacusis. A study by Ohmura et al. [15] analyzed stapedial reflex threshold with contralateral suppression of DPOAEs and found that the stapedial reflex was decreased in the ASD population. In addition, Kaf and Danesh [16] noted that sound hypersensitivity in high-functioning autistic children could result from abnormal neural connections at proximal structures to the medial olivary complex, such as the temporal lobe, limbic system, and autonomic nervous system. In a related study of autism [17], the same researchers studied 14 children with ASD and 28 age-matched controls, and noticed that the DPOAEs had smaller amplitude and insufficient contralateral suppression in the ASD group, a finding which tends to support the involvement of the efferent auditory pathways in hyperacusis.

Genetics may also play a significant role in the development of hyperacusis in the ASD population [4]. Mertcati et al. [18] reported that extra copies of the contactin five gene (*CNTN 5*) and deletions and mutations of the contactin six gene (*CNTN 6*) have been found in autistic individuals with hyperacusis. The expression of these gene variants may result in changes in their auditory brainstem response (ABR) waveforms within the auditory pathway, presenting as sound hypersensitivity [4].

Misophonia has been observed to be comorbid with a variety of psychiatric and developmental disorders but cannot be applied exclusively to one specific disorder [8,19]. Triggered by specific sounds, misophonia elicits a negative emotional response (e.g., anger, rage, and irritation) and a fight or flight reaction [1]. As this disorder typically develops during childhood or adolescence, it is important to further investigate misophonia within specific populations to gain a greater understanding of its development and progression, as well as effective therapeutic interventions for it [6]. The current literature considers misophonia as an unclassified disorder, and its nature remains unclear [7]. Siepsiak et al. [6] found that over 50% of their misophonia cases had at least one family member with the condition. In a genome-wide association study, Smit et al. [20] further investigated the genetic etiology of misophonia by focusing on the most reported related symptom, rage. They reported specific single nucleotide polymorphisms (SNP) linked to misophonia, including *TENM2*, *TMEM256*, *NEGR1*, *TFB1M*, and GABA-related genes. They considered that misophonia was not just a sensory disorder as it shares genetic etiology with conditions such as major depression disorder (MDD), post-traumatic stress disorder (PTSD), and generalized anxiety disorder (GAD) [20]. Rinaldi and al. [21] tested 142 children and 379 adults for traits associated with autism and found that autistic traits were more prevalent in those with misophonia compared to controls.

Despite the growing relevance of decreased sound tolerance disorders such as hyperacusis and tinnitus, there is a lack of knowledge and evidence of effective treatment for this disorder [1]. However, as knowledge of the causes, diagnostic criteria, and management of hyperacusis and misophonia improves, potential treatment options have increased. These include cognitive behavioral therapy (CBT), habituation training [4], exposure therapy, mindfulness, and drugs [1]. Pan et al. [1] presented a case of a 32-year-old male with ASD, multiple psychiatric comorbidities, and misophonia. They found that an increase in his risperidone dosage resulted in an unexpected improvement in his misophonia. Naguy et al. [7] reported similar success using a low dose of risperidone in a 4-year-old child with ASD. Despite the positive findings, further research is needed to confirm the efficacy of such pharmacological agents.

In an effort to describe the role of auditory training in the management of hyperacusis in the ASD population, Danesh et al. [4] examined the impact of habituation training as a treatment method for ASD individuals. Habituation training utilizes retraining to desensitize the emotional and non-classical auditory pathways to reduce the fear response to sound. The authors indicated that this specific intervention was effective in reducing the adverse reaction to sounds in ASD individuals.

In a systematic review, Stephanelli et al. [22] looked at various questionnaires used by professionals and highlighted the lack of standardized tools used to determine dysregulated behavioral symptoms (DBS) due to the heterogeneity of ASD manifestations. Although the researchers did not find any evidence-based tools for treatment regarding hyperacusis, they did suggest that the auditory behavior tests showed involvement of the afferent and efferent auditory pathways.

## Conclusions

In this brief piece, we have looked specifically at DST in the ASD population, focusing on hyperacusis and misophonia. There appears to be a gap in the literature regarding hyperacusis and misophonia in individuals with autism. At present information is still lacking about etiology, diagnosis, and effective management. Future research should address therapeutic strategies such as auditory habituation, user-friendly AI-based CBT, and explore low-level electrical stimulation (as currently used for managing tinnitus). Effective treatments will decrease social isolation and increase the quality of life of affected individuals.

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