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HYPOTHESIS PAPER

THE NEUROPHYSIOLOGICAL MODEL FOR HYPERACUSIS AND MISOPHONIA

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Abstract

Tinnitus patients frequently complain about Decreased Sound Tolerance (DST) as well. Based on the observations of several hundred patients with Decreased Sound Tolerance the existence of unrecognized earlier auditory disorder with distinctive and different from hyperacusis characteristics has been identified. Patients with this disorder exhibited negative emotional and autonomic reactions evoked by a specific for a given patient patterns of sound. Consequently, the name *misophonia* for this previously not described disorder and its definition based on data gathered from our patients was proposed in 2001. It is possible to have tinnitus, hyperacusis, and misophonia in combination or as isolated conditions.

On the basis of analysis of characteristic features of patients with DST, and general neuroscience, the neurophysiological model for both misophonia and hyperacusis has been proposed. The key characteristic of misophonia is the formation of inappropriate, pattern-specific, subconscious connections, between the auditory system and other systems of the brain, governed by the principles of conditioned reflexes. Notably, the brain systems and connections involved in misophonia are the same as in the case of tinnitus.

The mechanism of hyperacusis is based on abnormally increased gain within the subconscious part of the auditory pathways. This yields a high level of neuronal activity, equivalent to activity evoked by a much stronger sound in normal subjects. The activation of the other systems in the brain is a consequence of spreading this abnormally enhanced sound-evoked activity by normally functioning neuronal connections from the auditory to other systems in the brain. In misophonia, sound-evoked signals within the auditory pathways are normal, but development of incorrect pattern-specific connections yield abnormally strong activations of various systems in the brain. In hyperacusis and misophonia reactions evoked by bothersome sound are very similar, even identical and cannot be used to differentiate these two disorders. Importantly, both clinical observations and predictions of the model point out that misophonia requires a different approach for diagnosis and treatment than hyperacusis.

Keywords: The Neurophysiological Model of Tinnitus and Decreased Sound Tolerance • DST • hyperacusis • misophonia • tinnitus

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NEUROFIZJOLOGICZNY MODEL SZUMÓW USZNYCH I OBNIŻONEJ TOLERANCJI NA DŹWIĘKI

Streszczenie

Pacjenci cierpiący na szumy uszne często skarżą się również na obniżoną tolerancję na dźwięki (DST). Na podstawie obserwacji kilkuset pacjentów z DST zostało zidentyfikowane nieznane dotąd zaburzenie słuchu, wyraźnie różniące się od nadwrażliwości słuchowej (hyperakuzji). Pacjenci cierpiący na to zaburzenie wykazują negatywne reakcje emocjonalne i odpowiedzi z autonomicznego układu nerwowego wywołane przez specyficzne dla danego pacjenta wzorce dźwiękowe. W 2001 roku została zaproponowana nazwa *mizofonia* dla tego nieznanego dotąd zaburzenia, a jego definicję oparliśmy na danych zebranych od naszych pacjentów. Szumy uszne, nadwrażliwość słuchowa i mizofonia mogą występować łącznie lub jako oddzielne schorzenia.

Na podstawie charakterystycznych cech pacjentów z DST zaproponowano neurofizjologiczny model zarówno hyperakuzji, jak i mizofonii. Niniejszy artykuł rozszerza aspekty modelu przedstawionego podczas 7th International Conference on Hyperacusis and Misophonia (ICHM), która odbyła się w dniach 15–17 września 2024 r. w Warszawie. Kluczową cechą mizofonii jest rozwój – regulowany przez odruchy warunkowe – nieodpowiednich, specyficznych dla danego wzorca, podświadomych połączeń między układem słuchowym a innymi częściami mózgu. Co istotne, zarówno w przypadku mizofonii, jak i szumów usznych aktywne są te same układy i połączenia mózgowe.

Mechanizm nadwrażliwości słuchowej opiera się na nieprawidłowo zwiększonej czułości w podświadomej części ścieżek słuchowych. Powoduje to wysoki poziom aktywności neuronalnej odpowiadający aktywności wywołanej przez znacznie silniejszy dźwięk u osoby zdrowej. Aktywacja tych innych układów nerwowych wynika z rozprzestrzeniania się nadmiernie wzmocnionej aktywności wywołanej dźwiękiem z układu słuchowego do innych układów w mózgu poprzez prawidłowe połączenia neuronowe. W przypadku mizofonii sygnały wywołane dźwiękiem w ścieżkach słuchowych są prawidłowe, ale rozwój nieprawidłowych połączeń specyficznych dla danego wzorca powoduje nieadekwatnie silną aktywację różnych innych układów w mózgu. W przypadku nadwrażliwości słuchowej i mizofonii reakcje wywołane przez uciążliwy dźwięk są bardzo podobne, a nawet identyczne, i nie można ich wykorzystać do rozróżnienia tych dwóch zaburzeń. Zarówno obserwacje kliniczne, jak i prognozy modelowe sugerują, że mizofonia wymaga innego podejścia do diagnozy i leczenia niż nadwrażliwość słuchowa.

Słowa kluczowe: neurofizjologiczny model powstawania szumów usznych i obniżonej tolerancji na dźwięki • DST • nadwrażliwość słuchowa • mizofonia • szumy uszne

Key to abbreviations	
DST	Decreased Sound Tolerance
GAD	Generalized Anxiety Disorder
GAD-7	General Anxiety Disorder-7 (questionnaire)
ICD-10-CM	International Classification of Diseases, 10th Revision, Clinical Modification
ICD-11	International Classification of Diseases, 11th Revision
PHQ-9	Patient Health Questionnaire-9
TRT	Tinnitus Retraining Therapy

The aim of this paper is to expand description of the model described recently in *Frontiers in Neuroscience* publication [1], which we presented during the 7th International Conference on Hyperacusis and Misophonia, 15–17 September 2024, Warsaw, Poland. As such, we do not attempt to describe the model in detail, but only to delineate it, stressing specific issues which were not sufficiently described in previous publications [1,2].

Most of our tinnitus patients treated since 1990 reported having problems when exposed to external sound. Based on the literature, interactions with professionals from the field of auditory disorders, and our accumulated knowledge from clinical practice, we proposed to define Decreased Sound Tolerance (DST) as present when a person exhibits negative reactions following exposure to sound that would not evoke the same response in an average listener. DST is listed as a syndrome in many medical conditions (e.g., acoustic trauma, migraines, William's syndrome, Lyme Disease, and some mental health conditions, e.g., autism). For years, two terms, hyperacusis and phonophobia, have been used in literature to describe abnormal reactions to sound. These two conditions were not carefully defined and still are frequently used interchangeably, resulting in confusion. Clinical observations revealed that a certain category of DST cannot be classified as either hyperacusis or phonophobia. Therefore, in 2001 we proposed a new term – misophonia – for this type of DST [3].

First, based on clinical observation, behavioral definitions for hyperacusis and misophonia have been proposed, which are independent of etiology or potential theoretical mechanisms [1,2].

- *Hyperacusis* is defined as a condition when negative reactions to a sound depend only on its physical characteristics (i.e., spectrum, intensity). Time course (coded in the phase of spectrum) and meaning of the sound are irrelevant.

- *Misophonia* defined as condition when negative reactions occur to sounds with specific patterns and/or meaning to a given patient; physical characteristics are secondary (for detailed definitions see [1,2,4]).

Considering these behavioral definitions and general knowledge of neuroscience, the following mechanisms-based definition have been proposed [1,2].

- *Hyperacusis* – reflects abnormally strong reactivity of the auditory pathways to sound, which only in turn yields activation of the limbic and autonomic nervous systems (**Figure 1**).

- *Misophonia* – reflects abnormally strong reactions of the autonomic and limbic systems resulting from enhanced functional connections between the auditory, limbic, and autonomic systems for specific for a given patient pattern of sound (**Figure 2**).

There is a discussion regarding definitions of DST and furthermore misophonia. Proposed definitions are based on neurophysiology and should not be considered to be audiological, or psychological. Fundamental postulate of the neurophysiological model of tinnitus and DST is that in these disorders the auditory system plays a secondary role and other systems in the brain are dominant. We have never postulated that misophonia is a purely auditory condition! It is clearly stated in our definition of misophonia: Misophonia – reflects abnormally strong reactions of the autonomic and limbic systems resulting from enhanced functional connections between the auditory, limbic and autonomic systems for specific for a given patient pattern of sound (**Figure 2**) [1,2]. The basic postulate of the neurophysiological model of tinnitus and DST is that the auditory system plays a secondary role in clinically-significant (i.e., problem impacting subjects to such extent that they search for help and frequently become patients) tinnitus, hyperacusis and misophonia.

In 2022 Swedo at al. [4] published paper presenting attempt to create a consensus definition of misophonia, using Delphi method. However, the definition presented in Swedo's paper should not be treated as final one, but as first step in creating misophonia definition. This is clearly stated in Swedo's paper: "This definition represents an important first step for researchers and clinicians to progressively build-upon and revise as the body of knowledge in the published scientific literature grows over time" [4]. This point is presented in Brout's [5] paper as well: "The consensus definition is fluid and will change over time, and we should read it with that in mind".

The Delphi method defines consensus as 80% of committee members agreeing on an issue, acknowledging that some disagreement is expected. This highlights that the "consensus definition" within the Delphi framework is different from unanimity, as certain points will always have dissenting opinions among committee members. While a definition may be widely accepted, individuals are free to propose their own interpretations, and it is up to the readers to decide whether to accept them.

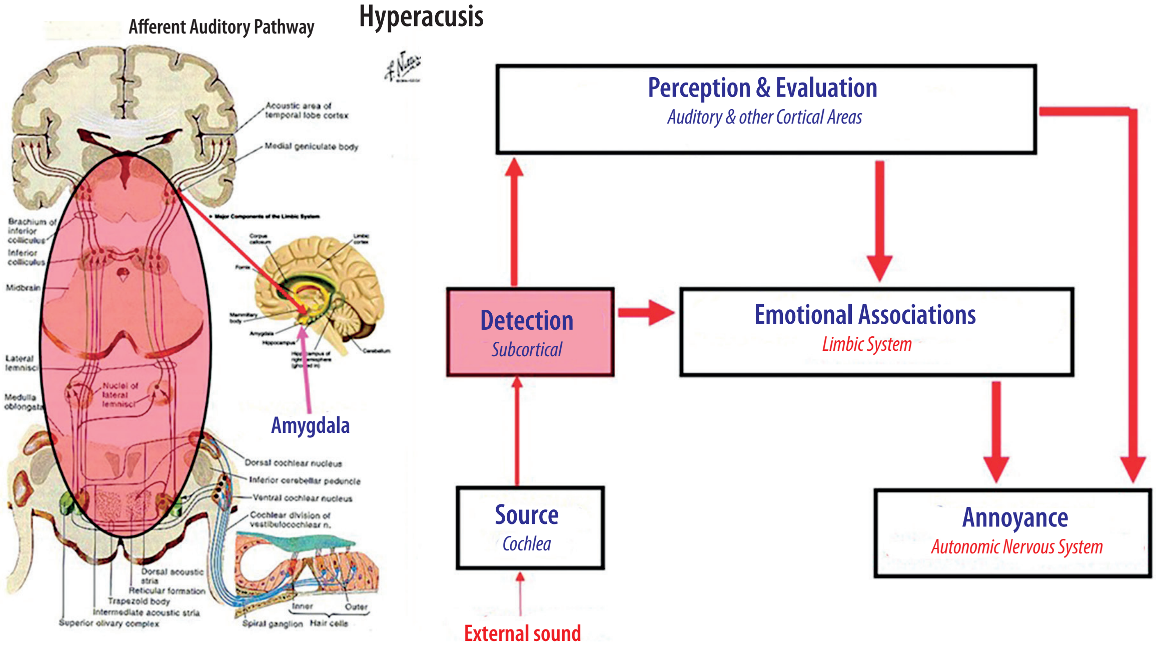

Figure 1. Proposed neurophysiological mechanism of hyperacusis. Hyperacusis reflects abnormally strong reactivity (abnormally high gain) of the auditory pathways to sound, which only in turn yields activation of the limbic and autonomic nervous systems

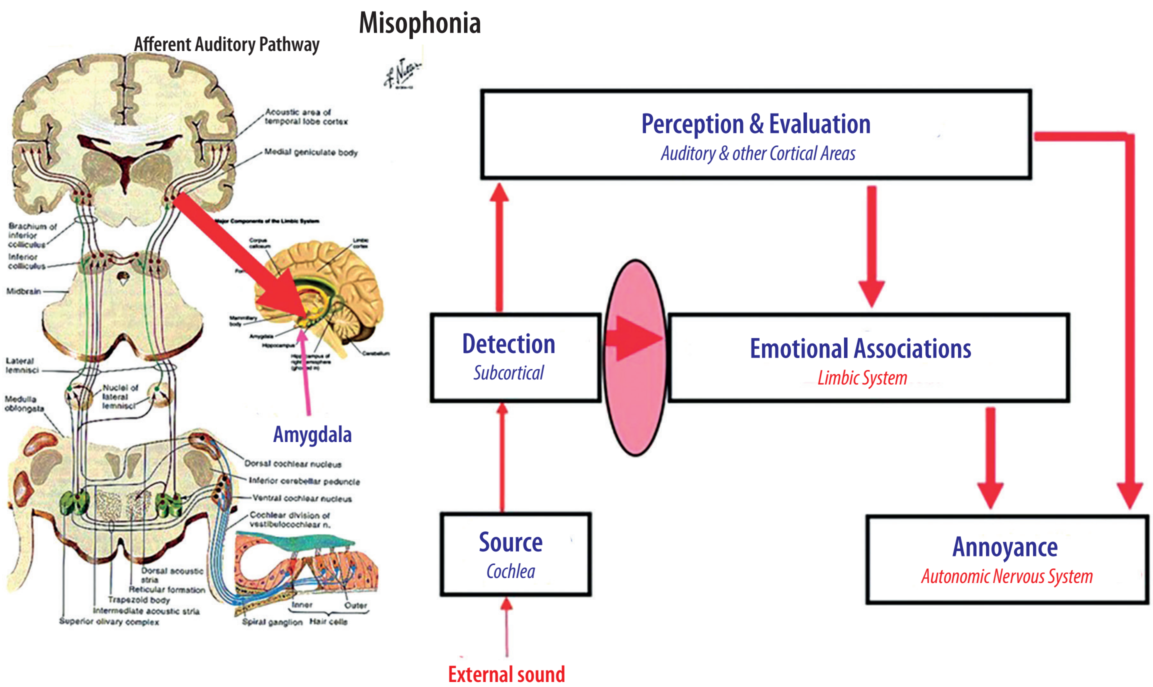

Figure 2. Proposed neurophysiological mechanism of misophonia. Misophonia reflects abnormally strong reactions of the autonomic and limbic systems resulting from enhanced functional connections between the auditory, limbic, and autonomic systems specific to a given patient and patterns of sound

In our opinion misophonia is a specific case of a category of disorders manifested by negative reactions to a sensory stimulus from variety of modalities, which develops when a sensory stimulus is paired with negative reinforcement, e.g., *misoángigma* (touch), *misoórasi* (vision), *misokinisi* (movement), *misoosmi* (smell), *misogefsi* (taste), etc. In our population of patients, we have cases where negative reaction occurs only to olfactory, or only visual stimuli without any involvement of sound. This points out the possibility of the existence of other disorders in which negative

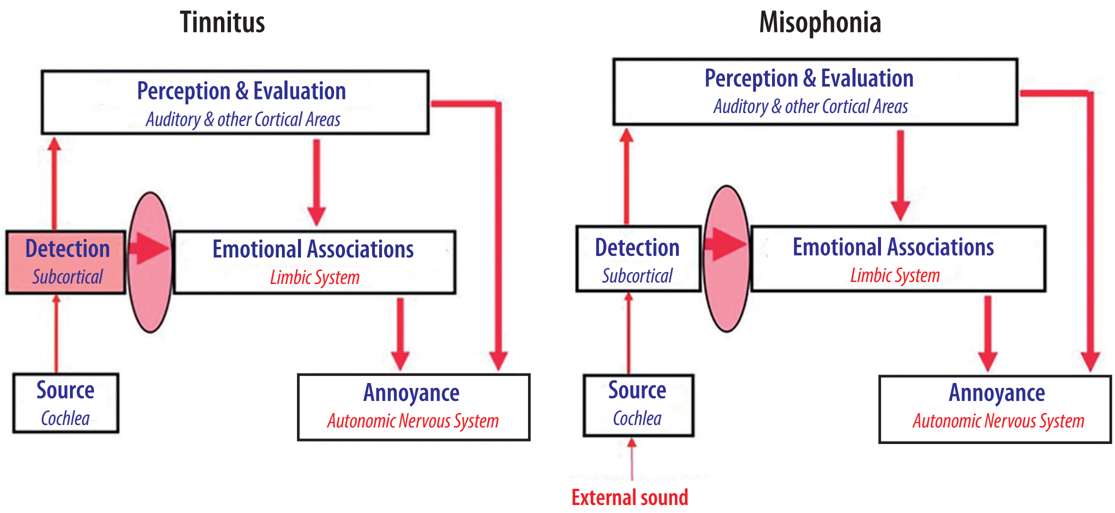

Figure 3. Similarity of mechanisms of tinnitus and misophonia. Tinnitus and misophonia differ by the origin of the sound – internal for tinnitus (phantom auditory perception) and external for misophonia, but otherwise the same systems and connections in the brain are involved

reactions are evoked by the stimuli of various modalities without sound being involved. Of course, reactions may be evoked by complex conditioned stimuli consisting of stimuli of different modalities and stimuli generalization may occur as well, which seems to be predominant situation in misophonia, e.g., presence of trigger sound while its source is visible (e.g., lawnmower, somebody eating) causes association of this visual stimulus with discomfort and consequently create secondary conditioned reflex causing that this specific visual stimulus alone evokes negative reaction. Again, similarly with smell, possibly with taste, as well as with repetitive movements.

There are some similarities, and crucial differences between mechanisms of tinnitus, hyperacusis and misophonia. All these phenomena reflect abnormal (i.e., different that for general population) reactions to sound. In all of them subconscious conditioned reflexes, connecting the auditory system with other systems in the brain are crucial, with conscious part playing secondary role, particularly in the acute phase of these disorders. None of them require dysfunction of other systems in the brain.

Psychological profile of a patient as well as potentially coexisting mental disorders may facilitate development of tinnitus, hyperacusis, and misophonia and they may influence the extent of reactions; note however, that mental disorders are not necessary for existence of tinnitus, hyperacusis or misophonia. Both tinnitus and hyperacusis (but not misophonia) involve some dysfunction within the auditory system.

Notably, reactions to external sound observed in hyperacusis and misophonia are remarkably similar, frequently identical, and cannot be used to differentiate these two phenomena. Tinnitus and misophonia differ by the origin of the neuronal activity perceived as sound – internal for tinnitus (phantom auditory perception) and external (sound-evoked) for misophonia, but otherwise the same systems and connections in the brain are involved (**Figure 3**). In misophonia the important postulate is that it may exist with all the systems of the brain functioning in the normal manner - problems arise from creating subconscious, functional connections between the auditory system and other systems in the brain governed by principles of conditioned reflexes. In hyperacusis, the underlying problem is an abnormal increase of the gain within the subconscious part of the auditory system, consequently resulting in activation of other systems in the brain, as it would occur in the person without DST, stimulated with high level of sound.

The crucial point which we are postulating is that tinnitus and misophonia do not require abnormal activity of any system in the brain (except abnormal neuronal activity in the auditory system perceived as tinnitus), but their mechanisms are based at misjudgment of the significance of perceived sound, treating these sounds as something linked to unpleasant or dangerous situation, threatening. Consequently, the brain forms automatic reactions at a subconscious level which are governed by the principles of conditioning reflexes. These reactions link the perception of tinnitus or misophonic triggers to activation of the limbic system (which deals with emotions, memory, and motivation) and the autonomic nervous system (which controls involuntary bodily functions like heart rate, breathing, and digestion). As a result, exposure to these triggers can automatically lead to emotional distress, anxiety, or physical symptoms. Reactions are correct, but to incorrect stimuli. This happens because misophonic triggers or tinnitus are misclassified (subconsciously, and sometimes consciously) as having a negative meaning [1].

Dysfunctions of some brain systems may facilitate emergence of tinnitus or misophonia and then may enhance negative reactions evoked by these disorders. However, they are not necessary for the emergence of tinnitus or misophonia! This issue may explain why imaging studies performed for many years in the field of tinnitus [6], and recently in misophonia failed to provide coherent results [7].

Kumar's [8] and Neasciu's [7] data support our model but push attention into direction which is in our opinion secondary. We hypothesize that all brain systems and their connections in misophonia patients may function entirely normally. Any observed alterations in the functionality of various brain centers are likely a consequence of already developed misophonia, rather than the primary cause. Furthermore, it is not even necessary for these functional modifications to be present in all misophonic individuals. While such modifications might contribute to the development of misophonia or intensify the reactions to triggers, they are not considered fundamental or essential for the condition to exist.

Neasciu's [7] observation of lack of consistency in imaging data further supports our postulate. The situation seems to be the same as in the case of tinnitus, where after many years and extensive researcher efforts, existing data are inconsistent, and the only common observation is involvement of limbic system. In 2023 Husain and Khan [6] published a review paper on neurophysiological basis of tinnitus, concluding "Currently, the most consistent findings of neural patterns associated with tinnitus have been observed when using the tool of resting-state fMRI. Results in structural imaging literature appear to be inconsistent". While research on misophonia is much shorter the conclusion is similar "The rapid advancements in understanding the neuroscience behind misophonia are promising. One drawback of the current literature is the little overlap in findings, despite similar paradigms" [7].

Described neurophysiological model provided guidance in creating a treatment approach, known as Tinnitus Retraining Therapy (TRT). Note that since a very beginning when TRT was introduced in 1990, hyperacusis and its treatment were a crucial element and part of classification of patients with tinnitus and DST into 5 categories. Just for brevity it was not included in the name of TRT. Once we noticed existence of phenomenon which we labeled "misophonia" the model has been expanded to include it and specific treatment has been developed, with protocol level (1) published in 2002 [9]. Treatment of misophonia is different from treatment for hyperacusis and applying hyperacusis treatment for misophonia is not effective. Description of misophonic treatment has been provided already [1,2,10], with published success rate of 83%. Interestingly, in many cases it was possible to achieve a cure, i.e., total disappearance of negative reaction to previously highly bothersome misophonic triggers [1,10,11].

Conclusions and recommendations

Misophonia is not a disease but a disorder resulting from development of conditioned reflexes linking the auditory system with other systems in the brain, particularly limbic and autonomic nervous systems, created for specific for a given patient patterns of sound. Misophonia can develop in anyone, triggered by any sound, when that sound is paired with a strong negative emotional or physiological experience. This pairing is sufficient and necessary conditions to create conditioned reflex, where the previously neutral sound becomes associated with the unpleasant state, ultimately triggering similar negative reactions on its own.

As such it should not be categorized as a mental disorder as is frequently postulated [7]. The confusion started in 2013 when Schroeder on the basis of results obtained in population of patients form psychiatric clinic proposed different definitions of misophonia and claimed that it is a mental disorder [12]. We strongly disagree with his definition and presented detail critique in our paper [1]. As with all medical disorders there is a comorbidity of misophonia with mental disorders. Recent literature suggests that it is not as common as initially thought resulting in a postulate that is a separate unique disorder [7]. Moreover, it was reported "that up to 50% of those who describe misophonic distress do not have any other mental health disorders" [13]. On the basis of our experience and literature we postulate that misophonia is not a mental disorder.

The issue of categorizing misophonia as a mental disorder is exacerbated by an insufficient distinction, in research, publications, and proposed treatments, between depression and anxiety that arise from specific situations and those that have a primarily physiological basis. The ICD-10-CM (the International Classification of Diseases, Tenth Revision, Clinical Modification, used to code and classify medical diagnoses) which differentiate between the Situational-Evoked (Reactive) Depression, ICD-10 codes F43.21 (Adjustment Disorder with Depressed Mood) or F43.23 (Adjustment Disorder with Mixed Anxiety and Depressed Mood) are versus Chronic Depression, codes F32.0 (Major Depressive Disorder, Single Episode, Mild), F32.1 (Moderate), F32.2 (Severe without psychotic features), or F32.3 (Severe with psychotic features), Generalized Anxiety Disorder (GAD) (F41.1): this code is intended to be used for anxiety that is ongoing and pervasive, not merely a reaction to specific circumstances. It should only be applied to situation-specific anxiety if the individual's symptoms meet the established diagnostic criteria for Generalized Anxiety Disorder [14,15]. Corresponding codes in ICD-11 are 6B41 (Adjustment disorder with depressed mood), 6B43.Z (Adjustment disorder, with mixed emotional and conduct symptoms), 6A70.3 represents (Single episode depressive disorder, severe, without psychotic symptoms) and 6A71.3 (Recurrent depressive disorder, current episode severe, without psychotic symptoms) and 6B00 (Generalized Anxiety Disorder) [16].

Commonly used questionnaires for depression (PHQ-9) and for anxiety (GAD-7) do not differentiate between situation-evoked problems versus chronic, medical problems. In published results on misophonia there is not information whether this distinction was taken into account and presence of depression or anxiety is treated as a proof that patient have "mental disorder". Applying this reasoning, e.g., cancer or other medical problems evoking depression or anxiety should be classified as a mental disorders. While psychologists and psychiatrists acknowledge the difference between situation-induced and physiologically-based anxiety, there is a lack of clear evidence that this distinction is consistently applied in their misophonia research, clinical treatments, and published works. In our experience practically all patients with significant misophonia exhibit depression and anxiety, but detailed interviews reveal that the vast majority of them got these problems after the start of misophonia and that depression and anxiety are situation-evoked.

Importantly, the differences in postulated mechanisms between hyperacusis and misophonia necessitate different treatments. Specifically, treatment as for hyperacusis is not effective for misophonia while treatment as for misophonia has limited effectiveness for hyperacusis. According to proposed model and our clinical observations focusing on the psychoacoustical parameters of misophonic triggers as well as on psychological or psychiatric categorization of patients are counterproductive. Analysis of social and environmental factors is important for understanding, treatment, and prevention of misophonia to occur and may play a significant role in the development of misophonia.

Noteworthy, patients' accommodation should be kept to a bare minimum as it enhances the severity of misophonia and hinders its treatment. Our observation for over 30 years suggested that the extent of accommodation correlates with misophonia severity and that gradually decreasing accommodation improves severity while increasing accommodation yields worsening of the problem. While some accommodation is needed, particularly at the beginning of the treatment, it should be kept to a minimum and for limited time only.

Recent publication support concerns regarding accommodation pointing out that "Excessive and maladaptive accommodation may be one potential candidate to target in interventions when considered within a broader treatment plan. Importantly, adaptive accommodations should also be considered in day-to-day management if they improve functioning and quality of life" [17].

Misophonia can be induced by social norms imposed in society, e.g., Prof. Norena's postulate "misophonia can be caused by a failure in the organization of the perceived world" and "In this context, the role of social rules acquired throughout life is considerable. Table manners, for example, are a set of deeply regulated and controlled behaviors (it's considered impolite to eat with the mouth open and to make noise while eating), which contribute to shape the way the perceived world is organized. So, it's not surprising to find sounds from the mouth (chewing etc.) among the most common misophonic sound triggers" [18].

Discussed ideas are presented in more detail in Jastreboff PJ and Jastreboff MM (2023) "The neurophysiological approach to misophonia: theory and treatment" [1].

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