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# Effect of somatosensorial stimulation on tinnitus

## *Somatosensoriyel stimülasyonun tinnitus üzerine etkisi*

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***ABSTRACT***

**Objectives:** This study aimed to investigate the effects of trigeminal nerve (TN)-related somatosensory stimulation on the psychoacoustic parameters of tinnitus.

**Patients and Methods:** This prospective observational study was conducted at the Audiology Unit of İstanbul Medipol University Hospital between August 01, 2021 and April 01, 2022. Twenty-eight patients (21 females, 7 males; mean age: 39±0.5 years; range, 18 to 65 years) with unilateral subjective tinnitus were included. The patients underwent psychoacoustic assessments under five conditions: the standard model, the mastoid model, and trigeminal stimulation at the ophthalmic (V1; forehead), maxillary (V2; maxilla), and mandibular (V3; preauricular) branches of the TN. Pitch matching, loudness matching, minimum masking level (MML), and residual inhibition (RI) were measured in each condition.

**Results:** Significant correlations were observed between psychoacoustic parameters across different models ($p<0.05$). Tinnitus pitch did not differ significantly between models ($p>0.05$). Loudness and MML values were significantly higher in the standard model compared to other models ($p<0.05$). Residual inhibition rates in the preauricular (V3) model did not significantly differ from the standard model ($p>0.05$), despite lower MML values. The tinnitus intensities and MMLs measured in the standard model were significantly higher than the other models ($p<0.05$). The RIs of preauricular model was not significantly different from the other models ($p>0.05$). Similar RI rates were measured despite the lower MML levels in the preauricular model in comparison with the standard model.

**Conclusion:** Somatosensory stimulation via the V3 branch of the TN may influence tinnitus loudness and MML without altering pitch perception. The preauricular model may serve as an alternative to the standard model, offering similar RI outcomes at lower MMLs. This approach could be particularly advantageous in patients with conductive hearing pathologies such as chronic otitis media, otosclerosis, or aural atresia.

***Keywords:*** Residual inhibition, somatosensory, tinnitus, trigeminal nerve.

***ÖZ***

**Amaç:** Bu çalışmada, trigeminal sinir (TS) ilişkili somatosensoriyel stimülasyonun psikoakustik parametreler üzerine etkisi incelendi.

**Hastalar ve Yöntemler:** Bu prospektif gözlemsel çalışma, 01 Ağustos 2021 - 01 Nisan 2022 tarihleri arasında İstanbul Medipol Üniversitesi Hastanesi Odyoloji Biriminde yürütüldü. Çalışmaya tek taraflı subjektif tinnitus tanılı 28 hasta (21 kadın, 7 erkek; ort. yaş: 39±0.5 yıl; dağılım, 18-65 yıl) dahil edildi. Hastalar beş farklı koşul altında psikoakustik değerlendirmelere tabi tutuldu: standart model, mastoid model ve trigeminal sinirin oftalmik (V1; alın), maksiller (V2; üst çene) ve mandibular (V3; preauriküler bölge) dallarının uyarılması. Her modelde frekans eşleştirme, gürültü şiddeti eşleştirme, minimum maskeleme düzeyi (MMD) ve rezidüel inhibisyon (RI) ölçüldü.

**Bulgular:** Farklı modeler arasında psikoakustik parametreler arasında anlamlı korelasyonlar gözlemlendi ($p<0.05$). Tinnitus frekansı modeler arasında anlamlı farklılık göstermedi ($p>0.05$). Tinnitus şiddeti ve MMD'ler standart modelde diğer modellere kıyasla anlamlı derecede daha yüksekti ($p<0.05$). Düşük MMD'lere rağmen, preauriküler (V3) modeldeki RI değerleri standart modelden anlamlı farklılık göstermedi ($p>0.05$). Standart modelde ölçülen tinnitus şiddeti ve MMD'ler diğer modellerden anlamlı derecede daha yüksekti ($p<0.05$). Preauriküler modelin RI değerleri diğer modellerden anlamlı farklılık göstermedi ($p>0.05$). Düşük MMD'lere rağmen, preauriküler model standart modelle benzer RI değerleri sergiledi.

**Sonuç:** Trigeminal sinirin V3 dalının somatosensöriyel stimülasyonu tinnitus şiddetini ve MMD'yi frekans algısını değiştirmeden etkileyebilir. Preauriküler model, daha düşük MMD'lerde benzer RI sunarak standart modele bir alternatif oluşturabilir. Bu yaklaşım özellikle kronik orta kulak iltihabı, otoskleroz veya kulak atrezisi gibi iletim tipi işitme bozukluğu olan hastalarda yararlı olabilir.

***Anahtar sözcükler:*** Rezidüel inhibisyon, somatosensöriyel, tinnitus, trigeminal sinir.

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Tinnitus, which can be defined as perception of sound without an external auditory stimulus, has a prevalence of 5.1 to 42.7%.[1] Almost one-fourth of individuals may experience tinnitus even in the absence of a predisposing factor.[2]

Psychoacoustic measurements of tinnitus help evaluate features of tinnitus such as loudness and frequency matching, minimal masking level (MML), and residual inhibition (RI). Minimal masking level shows the lowest threshold at which tinnitus can be masked by narrow-band noise. Residual inhibition can be defined as the temporary reduction or elimination of tinnitus perception with masking noise. The psychoacoustic measurements have been used in the evaluation of tinnitus even though there is no standardization in the measurement outcomes.[3,4]

The source of tinnitus can be the peripheral part of the auditory system such as the cochlea or the central portions of the auditory system. An aberrant auditory signal may lead to tinnitus by affecting the neural plasticity in the central auditory system, and limbic system connections are likely to play a role in the chronicity of tinnitus.[5,6]

Somatosensory system activities, such as muscle contractions related to the temporomandibular joint, head, neck, limbs, and orofacial and eye movements, as well as pressure stimulation of myofascial trigger points, skin of the hands, and fingertips, appear associated with tinnitus modulation.[7] Somatosensory modulation of tinnitus may occur in the presence or absence of a concomitant somatic disorder.[8] It is considered that auditory and somatosensory system integration results from the fusion of projections from the cochlear and trigeminal nerves (TNs), dorsal column ganglia, and cochlear nucleus.[7,9] It is possible that the TN is associated with tinnitus through somatosensory mechanisms. The fifth cranial nerve (TN) develops from the first branchial arch.[9-11] The nerve has both sensory and motor functions in the head and neck regions via its ophthalmic (V1), maxillary (V2), and mandibular (V3) branches.[11-13] Dorsal root ganglion axons and TN fibers are also connected to cochlear nuclei, inferior colliculus, and other central auditory structures.[14,15] Afferent somatosensory information to the secondary sensory neurons in the brainstem is delayed in the trigeminal nucleus and dorsal root cells, which send excitatory projections to the cochlear nucleus. Trigeminal nerve activation leads to excitation in dorsal cochlear nucleus (DCN).

Trigeminal nerve stimulation and its effects on the psychoacoustic parameters of tinnitus have emerged as a critical research area due to the high prevalence and disabling nature of tinnitus, which affects approximately 10 to 15% of adults, with 1 to 3% experiencing severe impairment in quality of life.[16,17] Recent neurophysiological and neuroimaging studies have highlighted maladaptive plasticity not only in the auditory but also in the somatosensory pathways.[18,19] While early tinnitus research focused on auditory cortex reorganization, more recent work has identified the TN as a potential modulator of tinnitus perception through its projections to the DCN.[20,21]

Despite this progress, the specific effects of stimulating different TN branches (V1, V2, and V3) on tinnitus remain poorly understood. Somatosensory inputs from the trigeminal system can modulate tinnitus perception; however, direct comparative studies evaluating the distinct impact of V1, V2, and V3 stimulation on tinnitus psychoacoustic parameters are scarce.[21,22] Existing research often combines trigeminal stimulation with auditory input or focuses on single branches without systematic comparison.[23-26] This lack of standardized protocols and branch-specific analyses limits clinical translation and leaves a gap in understanding the neural pathways mediating trigeminal influences on tinnitus.[27,28]

Conceptually, tinnitus involves aberrant neural activity characterized by hyperexcitability, abnormal synchronization, and maladaptive plasticity within auditory and somatosensory networks.[29,30] Trigeminal branches provide somatosensory input to the DCN, a key site of multisensory integration implicated in tinnitus generation.[18,24] Understanding how V1, V2, and V3 stimulation differentially modulates these neural circuits is essential to elucidate mechanistic pathways and optimize neuromodulator therapies.[31]

This study aimed to evaluate the effects of TN related somatosensory mechanisms on the psychoacoustic parameters of tinnitus.

## PATIENTS AND METHODS

A total of 28 patients (21 females, 7 males; mean age: 39±0.5 years; range, 18 to 65 years) with subjective unilateral tinnitus were included in this prospective observational study. The study was conducted at the Audiology Unit of the İstanbul Medipol University, Medipol Mega Hospital, between August 01, 2021 and April 01, 2022. Tinnitus was present on the right side in nine subjects and on the left side in 19 subjects. Tinnitus duration s ranged from 1 to 120 months (mean duration: 22.3±29.5 months). Written informed consent was obtained from all

participants after explaining the study objectives, procedures, and voluntary participation. The study protocol was approved by the İstanbul Medipol University Non-Interventional Clinical Research Ethics Committee (Date: 17.06.2021, No: E-10840098-772.02-2859). The study was conducted in accordance with the principles of the Declaration of Helsinki.

There were no subjects who had neurological, neuro-ontological, psychiatric, metabolic and endocrine disease as determined by clinical, radiological, and laboratory evaluations. The pure tone averages at the frequencies of 0.5 to 4 kHz were within normal limits, and tympanograms were type A in the subjects. Different modes of stimulation were used to measure the psychoacoustic parameters of tinnitus, which were standard model, trigeminal, and mastoid models. In the standard model, psychoacoustic parameters were measured using air conduction headphones (TDH-39; Telephonics Corp., Farmingdale, NY, USA) in a sound-treated booth.

In the trigeminal model, the measurements were performed to evaluate the effects of somatosensory mechanisms on tinnitus, which possibly occur through interactions between the TN and auditory system. Therefore, the psychoacoustic parameters were measured by stimulating three different areas on the face innervated by the branches of TN. These areas were as follows: *(i)* supraorbital or forehead area on the frontal bone (V1 branch); *(ii)* infraorbital or buccal region area on the maxillary bone (V2 branch); *(iii)* tragal or preauricular area on the mandibular ramus (V3 branch).

In the mastoid model, the measurements were made on the mastoid bone (behind the ear) to differentiate between the impacts of bone and TN stimulations.

Air conduction headphones were used in the standard measurements of psychoacoustic parameters. Pitch matching was performed using the "two-option method," which is the most commonly used method. Two different pure tone or narrow-band stimuli were presented, and the option was continued until the tinnitus pitch was matched by using the stimulus that the patient found closest to their tinnitus. During loudness matching, the sound was increased by 1 dB increments, starting from below the threshold at Hz where the pitch was matched. The participant was asked to say "stop" at the first point matched by the tinnitus and the loudness of the given sound. In frequency equalization measurement, narrow-band noise was used in case of pure tone determination, and broadband noise was used in case of narrowband noise determination. The volume was increased in steps of 1 dB, and the participant was asked to say "stop" at the first

**Table 1**
Comparison of tinnitus frequencies, intensity, and MML measured using different models

| Model (parameter) | Mean±SD | Min-Max |
|---|---|---|
| Standard (Frequency) | 5236.61±3090.15 | 125-8000 |
| Mastoid (Frequency) | 5459.82±3163.2 | 125-8000 |
| Forehead (Frequency) | 5406.25±2974.94 | 125-8000 |
| Maxillary (Frequency) | 5433.04±3106.98 | 125-8000 |
| Preauricular (Frequency) | 5566.96±3145.12 | 125-8000 |
| Standard (Intensity) | 45.54±24.74 | 10-92 |
| Mastoid (Intensity) | 28.0±14.26 | 6-64 |
| Forehead (Intensity) | 28.39±14.45 | 4-55 |
| Maxillary (Intensity) | 29.43±15.16 | 0-56 |
| Preauricular (Intensity) | 24.86±16.43 | 0-59 |
| Standard (MML) | 44.96±23.38 | 6.0-89.0 |
| Mastoid (MML) | 32.29±17.72 | 5.0-69.0 |
| Forehead (MML) | 34.71±16.87 | 7.0-70.0 |
| Maxillary (MML) | 34.11±18.64 | 4.0-70.0 |
| Preauricular (MML) | 26.18±16.71 | 0.0-66.0 |

MML: Minimal masking level; SD: Standard deviation.

point where they did not hear tinnitus to determine the MML. To determine RI, the sound was applied continuously for 1 min by adding 10 dB sensation level on top of the dB hearing level determined by MML. If the patient's tinnitus stopped, RI was positive; if there was a decrease, the RI part was called positive; and if there was no change, RI was negative.

Somatosensory stimulation in the trigeminal (V1-forehead/supraorbital; V2-infraorbital/maxillary; V3-preauricular/tragal) and mastoid models was delivered using a calibrated Radioear B-72 bone vibrator (Radioear Corp., New Eagle, PA, USA) positioned over the target site with consistent contact pressure. The vibrator was driven with a continuous 250-Hz pure tone, an effective frequency for inducing vibrotactile stimulation of trigeminally innervated regions reported in prior neuromodulation research. Intensity was set at 10 dB sensation level above each participant's vibrotactile detection threshold for that site. Each stimulation lasted 60 sec to allow stable somatosensory and auditory interaction while minimizing participant fatigue. Output levels were verified daily using an artificial mastoid (Brüel & Kjær Type 4930, Nærum, Denmark), and participants were monitored for any discomfort during stimulation.

The order in which the different stimulation models (standard air-conduction, trigeminal V1, V2, and V3, and mastoid) were tested was randomized for each participant using a computer-generated list randomization tool (www.random.org/lists). To prevent learning or adaptation effects, a rest interval of 60 sec was implemented between the completion of one model's measurements and the initiation of the next. All psychoacoustic parameters (pitch matching, loudness matching, MML, and RI) were assessed in order, with each parameter for a given model completed before proceeding to the next model.

**Statistical analysis**

Statistical analyses were performed using IBM SPSS version 22.0 software (IBM Corp., Armonk, NY, USA). The data obtained from five different areas were compared using paired samples t-test and the chi-square test. Pearsons's correlations were used to evaluate the correlations between the measurements performed in the models.

## RESULTS

The analysis of psychoacoustic parameters of tinnitus across different anatomical models revealed varying effects of TN-related somatosensory mechanisms. As shown in Table 1, the mean frequency values ranged from 5236.61±3090.15 Hz in the standard model to 5566.96±3145.12 Hz in the preauricular model; however, pairwise comparisons indicated no statistically significant differences between models (Table 2).

In terms of intensity, the standard model exhibited the highest mean intensity (45.54±24.74), while the preauricular model showed the lowest (24.86±16.43), as illustrated in Table 1. Significant differences were observed between the standard model and all other

**Table 2**
Pairwise comparison of frequency, severity, and MML for tinnitus assessment using different models

| Pairwise comparison | *p* |
|---|---|
| Standard *vs.* mastoid (Frequency) | 0.275 |
| Standard *vs.* forehead (Frequency) | 0.429 |
| Standard *vs.* maxillary (Frequency) | 0.348 |
| Standard *vs.* preauricular (Frequency) | 0.131 |
| Mastoid *vs.* forehead (Frequency) | 0.823 |
| Mastoid *vs.* maxillary (Frequency) | 0.916 |
| Mastoid *vs.* preauricular (Frequency) | 0.602 |
| Forehead *vs.* maxillary (Frequency) | 0.925 |
| Forehead *vs.* preauricular (Frequency) | 0.358 |
| Maxillary *vs.* preauricular (Frequency) | 0.530 |
| Standard *vs.* mastoid (Intensity) | 0.000 |
| Standard *vs.* forehead (Intensity) | 0.000 |
| Standard *vs.* maxillary (Intensity) | 0.000 |
| Standard *vs.* preauricular (Intensity) | 0.000 |
| Mastoid *vs.* forehead (Intensity) | 0.831 |
| Mastoid *vs.* maxillary (Intensity) | 0.324 |
| Mastoid *vs.* preauricular (Intensity) | 0.139 |
| Forehead *vs.* maxillary (Intensity) | 0.388 |
| Forehead *vs.* preauricular (Intensity) | 0.047 |
| Maxillary *vs.* preauricular (Intensity) | 0.002 |
| Standard *vs.* mastoid (MML) | 0.000 |
| Standard *vs.* forehead (MML) | 0.001 |
| Standard *vs.* maxillary (MML) | 0.000 |
| Standard *vs.* preauricular (MML) | 0.000 |
| Mastoid *vs.* forehead (MML) | 0.295 |
| Mastoid *vs.* maxillary (MML) | 0.453 |
| Mastoid *vs.* preauricular (MML) | 0.016 |
| Forehead *vs.* maxillary (MML) | 0.689 |
| Forehead *vs.* preauricular (MML) | 0.000 |
| Maxillary *vs.* preauricular (MML) | 0.000 |

MML: Minimal masking level.

**Table 3**
Comparison of tinnitus RIs measured using different models

| | RI positive | | RI partial | | RI negative | | | Chi-square test |
|---|---|---|---|---|---|---|---|---|
| Model | n | % | n | % | n | % | Model | *p* |
| Standard | 4 | 14.3 | 16 | 57.1 | 8 | 28.6 | Mastoid | NS |
| | | | | | | | Forehead | **0.03** |
| | | | | | | | Maxillary | **0.006** |
| | | | | | | | Preauricular | NS |
| Mastoid | 4 | 14.3 | 13 | 46.4 | 11 | 39.3 | Forehead | NS |
| | | | | | | | Maxillary | **0.02** |
| | | | | | | | Preauricular | NS |
| Forehead | 2 | 7.1 | 11 | 39.3 | 15 | 53.6 | Maxillary | **0.03** |
| | | | | | | | Preauricular | NS |
| Maxillary | 6 | 21.4 | 10 | 35.7 | 12 | 42.9 | Preauricular | NS |
| Preauricular | 4 | 14.3 | 14 | 50 | 10 | 35.7 | | |

RI: Residual inhibition; NS: Not significant.

models (p<0.001), as well as between the preauricular model and the forehead (p=0.047) and maxillary (p=0.002) models, as detailed in Table 2.

Minimal masking level results showed the highest mean MML in the standard model (44.96±23.38) and the lowest in the preauricular model (26.18±16.71; Table 1). Pairwise comparisons further highlighted significant differences between the standard model and all other models (p<0.001), as well as between the preauricular model and the mastoid (p=0.016), forehead (p<0.001), and maxillary (p<0.001) models (Tables 1, 2).

As shown in Table 3, the preauricular model demonstrated the highest proportion of positive RI responses (21.4%), while the maxillary model showed the lowest (7.1%) and the highest proportion of negative responses (57.1%). Partial responses were most frequent in the mastoid model (57.1%), with more balanced distributions observed in the preauricular and forehead models. The chi-square test revealed significant differences between models, particularly between the mastoid and forehead (p=0.03), mastoid and maxillary (p=0.006), forehead and maxillary (p=0.02), and maxillary and preauricular (p=0.03) models (Table 3). Comparisons involving the standard and preauricular models were not significant, indicating consistent RI distributions in these models.

## DISCUSSION

Tinnitus is considered to be an auditory system issue. However, its evident that the somatosensory system can also contribute to tinnitus.[32-34] Tinnitus modulation as a result of head, neck, and eye movements, also supports the effect of somatosensory system on tinnitus. This modulation results from the interaction of the auditory and somatosensory systems.[35] The limbic system may play a role in the chronic process of tinnitus.[5,36]

Auditory and somatosensory system interactions are likely to start at the level of the brainstem via TN and DCN connections.[37] A trigeminal stimulation applied before an acoustic stimulus may cause modulation on the temporal responses to the sound. Cochlear damage may also trigger tinnitus of somatosensory origin. Issues in the somatosensory system or DCN may cause neuroplastic changes through excitation or inhibition mechanisms in the central nervous system.[38,39]

Tinnitus severity and perception can be changed through somatic interactions. This condition is one of the main features of tinnitus and can be observed in two-thirds of the subjects. It is proposed that temporomandibular joint maneuvers may increase tinnitus severity in 60% of the subjects, while head-neck maneuvers may reduce tinnitus severity in 40% of the subjects.[40] The neural activity associated with tinnitus arises through the connections of different sensory systems such as sensory motor, somatomotor, visual motor, neurocognitive and neuroemotional systems.[7,41] Since somatosensory disorders play an important role in tinnitus, this condition should be taken into account in the treatment strategy of tinnitus.[7]

Despite the mentioned interaction between the auditory and somatosensory systems, the impact of somatosensory system on the psychoacoustic parameters of tinnitus has been lacking in literature. Our findings in this study may be helpful in this matter.

In this study, the measurements performed using different models were well-correlated. Hence, the models may substitute for each other when needed. In the light of these correlations, the comparisons performed between the models revealed that the frequencies of tinnitus measured with different models were similar. The intensity of tinnitus was lower in the other models compared to standard model, and the lowest level was measured via preauricular model. The MML of tinnitus measured in the standard model was significantly higher than the other models, and the lowest MML was measured in the preauricular model. The RI rates of standard and preauricular models were similar.

These findings suggest that TN related somatosensory mechanisms may not impact tinnitus frequency, whereas somatosensory interactions may be associated with tinnitus intensity and MML. Since the preauricular model yielded lowest intensity and MML values, it is speculated that the V3 branch of the TN may be involved in somatosensory interactions. In addition to lower MML levels in the preauricular model, similar RI rates in the preauricular models may have clinical significance. Therefore, the preauricular model could be preferred to the standard model since tinnitus masking could be made in the lower intensities.

This study had some limitations. The sample size was relatively small and derived from a single-center clinical population, which may restrict the generalizability of the results. In addition, the study did not include sham (placebo) stimulation or blinding procedures; therefore, non-specific effects related to device placement, tactile sensation, or participant expectation cannot be completely ruled out. Moreover, the cross-sectional design and short measurement period did not allow for the assessment of long-term or cumulative effects of trigeminal stimulation on tinnitus perception. Despite these limitations, the present study provides novel data on trigeminal-auditory interactions and may serve as a useful basis for future multicenter, double-blind, sham-controlled studies with larger sample sizes and extended follow-up durations.

In conclusion, the findings suggest that the stimulation of the V3 branch of the TN exerts a measurable influence on tinnitus psychoacoustic parameters, with notable effects on perceived loudness and MML. The comparable RI outcomes observed with the preauricular (V3) model, despite its lower masking levels, indicate that it may serve as a practical alternative to conventional air-conduction-based assessments. This could be particularly advantageous in patients with conductive or mixed hearing loss, where air conduction pathways are compromised. These results support the hypothesis that trigeminal-auditory cross-modal interactions contribute to tinnitus modulation and highlight the potential of targeted somatosensory stimulation in clinical evaluation protocols.

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**Data Sharing Statement:** The data that support the findings of this study are available from the corresponding author upon reasonable request.

**Author Contributions:** Conceptualization, data collection, investigation, and original draft preparation: D.B.; Supervision, methodology, and critical review of the manuscript: G.Ö.; Methodological guidance, and validation: O.Y.; Statistical analysis, data interpretation, manuscript editing, and corresponding author: Y.B.

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