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LITERATURE REVIEW

Tinnitus Mechanisms, Comorbidities and Therapeutic Strategies

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Abstract: Tinnitus is the perception of sound without an external source and affects about one tenth of the population, with a small proportion experiencing severe disability. It arises from cochlear injury, central auditory hyperexcitability, maladaptive neuroplasticity, and involvement of limbic, prefrontal, and somatosensory systems, with additional contributions from inflammation, oxidative stress, and metabolic factors. This narrative review summarizes current concepts of tinnitus mechanisms, frequent psychological, auditory, somatosensory, and systemic comorbidities, and available treatments. The strongest evidence supports psychological and sound based interventions such as cognitive behavioral therapy, counseling, hearing rehabilitation, and tinnitus retraining therapy, which reduce distress and improve daily functioning even when loudness changes little. Pharmacological therapy mainly targets comorbid depression, anxiety, sleep disturbance, inflammation, and oxidative stress. Adequate intake of zinc and iron may help prevent tinnitus. Despite several promising options, clinical evidence remains heterogeneous and no single therapy is universally effective, so individualized multidisciplinary care informed by underlying mechanisms and further supported by advances in basic research, biomarker development, and rigorous clinical trials is required.

Keywords: Tinnitus, Pathophysiology, Comorbidities, Therapy

INTRODUCTION

Tinnitus is the term used to describe the sensation of sound without any discernible external stimulus and is often perceived as buzzing, hissing, or ringing that is heard only by the affected individual.[1] This prevalent audiological condition has far-reaching implications for quality of life, including emotional distress, impaired concentration, and sleep disturbances.[1,2]

The global prevalence of tinnitus ranges from 9% to 15%, with approximately 1–2% of the population experiencing severe, debilitating symptoms.[1,2] Tinnitus may occur as a primary condition or as a symptom of other medical or neurological disorders.[3] Therefore, a deeper understanding of its underlying pathophysiology and associated

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comorbidities is crucial for devising effective treatment strategies.

In this context, an integrated overview of mechanisms, comorbidities, and available treatments is essential to support individualized, mechanism-informed management strategies.[4] This narrative literature review therefore aims to: (1) summarize current knowledge on the pathophysiological mechanisms underlying tinnitus; (2) describe key psychological, auditory, somatosensory, and systemic comorbidities that shape its clinical expression; and (3) critically review contemporary therapeutic approaches, including pharmacological and non-pharmacological modalities, with the goal of informing clinical practice and highlighting priorities for future research.

PATHOPHYSIOLOGY OF TINNITUS

Tinnitus is now understood as the result of a dynamic interaction between peripheral cochlear injury, maladaptive plasticity within the central auditory system, and the recruitment of non-auditory brain networks that shape the conscious perception and emotional impact of the symptom.[3,5,6]

Peripheral auditory triggers

In most patients, tinnitus is initiated by dysfunction or damage within the peripheral auditory apparatus, particularly the cochlea.[3] Noise trauma, age-related hearing loss, ototoxic drugs, and sudden sensorineural hearing loss can injure outer and inner hair cells, spiral ganglion neurons, or synapses between inner hair cells and auditory nerve fibres.[3,6] This cochlear deafferentation reduces afferent activity from specific frequency regions while leaving neighbouring regions relatively intact, leading to an imbalance of excitatory and inhibitory input to central auditory nuclei.[3]

Even when pure-tone thresholds remain within the clinically normal range, animal and human data suggest that synaptopathy of high-threshold auditory nerve fibres (“hidden hearing loss”) can impair suprathreshold sound coding and trigger central compensatory gain mechanisms that favour tinnitus generation.[3,6]

Central auditory maladaptive plasticity

Loss or distortion of peripheral input induces homeostatic plasticity along the central auditory pathway.[3,6] In the dorsal cochlear nucleus (DCN), a key candidate generator neurons develop increased spontaneous firing rates, enhanced bursting activity, and abnormal neural synchrony after cochlear injury.[6] Reduced GABAergic and glycinergic inhibition, together with strengthened glutamatergic excitation, produces a state of hyperexcitability that can encode a persistent phantom sound.[3,6]

Similar changes are observed in higher structures, including the inferior colliculus, medial geniculate body of the thalamus, and auditory cortex.[3,5] Thalamocortical circuits exhibit so-called thalamocortical dysrhythmia, with abnormal low-frequency and high-frequency oscillations that allow aberrant activity to reach awareness.[5] In the auditory cortex, tonotopic map reorganization with over-representation of edge frequencies adjacent to the hearing-loss region and increased gamma-band activity are closely

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associated with the conscious perception and loudness of tinnitus.[5,6]

Non-auditory networks: limbic, cognitive, and somatosensory systems

The clinical impact of tinnitus cannot be explained by auditory pathways alone. Functional and structural neuroimaging studies demonstrate consistent involvement of limbic and prefrontal regions, including the amygdala, hippocampus, anterior cingulate cortex, and dorsolateral prefrontal cortex.[5] These regions participate in emotional evaluation, attention, and cognitive control, forming an "auditory–limbic" network that determines whether the tinnitus signal is interpreted as threatening or neutral.[1,5]

This framework aligns with clinical data showing strong associations between tinnitus severity, anxiety, and depression.[1,7] Network analysis of tinnitus sufferers has identified central anxiety and depressive symptoms such as uncontrollable worry and hopelessness as key nodes that link tinnitus to broader emotional distress.[7] Dysfunction within salience and default-mode networks further contributes to persistent monitoring of the tinnitus percept and difficulty disengaging attention from it.[5]

Somatosensory and motor systems also modulate tinnitus. The DCN receives convergent input from trigeminal and dorsal column nuclei, allowing cervical spine and temporomandibular joint afferents to influence auditory processing.[3,6] In somatic tinnitus, jaw movements, neck muscle tension, or pressure on myofascial trigger points can alter tinnitus loudness or pitch, reflecting cross-modal plasticity in which deprived auditory circuits become increasingly driven by somatosensory input.[3,6]

Inflammation and oxidative stress

Beyond neural plasticity, chronic inflammation and oxidative stress have emerged as important modulators of tinnitus pathophysiology. Systematic review and meta-analytic data indicate that patients with tinnitus more frequently exhibit elevated inflammatory markers and indices of oxidative damage compared with controls, suggesting that immune-mediated processes contribute to cochlear and central neurodegeneration and to altered neurotransmitter balance.[8]

Local inflammation within the cochlea and auditory brainstem can exacerbate hair-cell loss and synaptic dysfunction, while systemic inflammatory states may affect cerebral and cochlear microcirculation.[8] Case-based evidence shows that, in rare instances where tinnitus is driven by distinct inflammatory disease, anti-inflammatory treatment can lead to symptom improvement, underscoring the need to consider inflammatory aetiologies during diagnostic work-up.[9]

Randomized trials and reviews of antioxidant supplementation such as combinations of vitamins C and E, melatonin, and other antioxidant compounds support a modest but clinically relevant reduction in tinnitus severity and improvement in sleep and quality of life in selected populations, particularly older adults.[10,13] These therapeutic observations indirectly reinforce the mechanistic role of oxidative stress in tinnitus maintenance.[8,10,11]

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Taken together, current evidence supports a multidimensional model in which tinnitus arises from peripheral cochlear injury, triggers central gain enhancement and maladaptive plasticity within thalamocortical auditory circuits, and is shaped by interactions with limbic, cognitive, somatosensory, and inflammatory–metabolic systems.[1,3,6,8] This complex pathophysiology explains the marked heterogeneity of clinical presentations and underpins the need for individualized, mechanism-informed management strategies.

TINNITUS COMORBIDITIES

Tinnitus is frequently associated with a wide spectrum of comorbid conditions that strongly influence its clinical presentation, perceived severity, and response to treatment. In clinical samples, patients with bothersome tinnitus rarely present with tinnitus alone; instead, they often exhibit a combination of auditory, psychological, somatosensory, and systemic problems that together determine overall disease burden.[1,2]

Psychological and psychiatric comorbidities

Cross-sectional and epidemiological studies report high rates of depression, generalized anxiety, and stress-related disorders in individuals with chronic tinnitus, particularly in those with severe or long-standing symptoms.[2,1] Patients with greater symptom severity tend to have higher levels of tinnitus-related handicap and reduced quality of life.[1,2]

In a network analysis, Chen et al.[7] demonstrated that symptoms such as uncontrollable worry, nervousness, and feelings of hopelessness occupy central positions in the psychopathological network of tinnitus sufferers, while bridge symptoms like fear of impending doom and poor concentration link anxiety and depressive clusters. These findings support a bidirectional relationship in which tinnitus exacerbates emotional distress and, in turn, negative affect and maladaptive cognitions increase tinnitus-related attention and suffering.[1,7] Neuroimaging studies further show abnormal activation and connectivity within limbic and prefrontal regions, underlining the neurobiological basis for this tight coupling between tinnitus perception and emotional regulation.[5]

Sleep disturbances and cognitive impairment

Sleep disturbance and cognitive complaints are also highly prevalent. Many patients report difficulty initiating or maintaining sleep, non-restorative sleep, and increased fatigue, which can worsen daytime tinnitus distress and lower stress tolerance.[1,2] Sleep problems are often intertwined with anxiety and depression, forming a vicious cycle that maintains tinnitus-related suffering.[7] Concentration difficulties, reduced working memory, and attentional bias toward tinnitus-related cues have been described and are thought to reflect altered activity within prefrontal and attentional-control networks that are concurrently engaged in tinnitus processing.[3,5]

Auditory comorbidities

From an auditory perspective, tinnitus most frequently coexists with

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sensorineural hearing loss. Population-based data show that noise exposure, age-related cochlear degeneration, and ototoxic medication are major shared risk factors for both tinnitus and hearing loss.[2] Damage to cochlear hair cells reduces peripheral input and promotes central gain enhancement, thereby contributing simultaneously to tinnitus and hyperacusis.[3,6] Patients with combined tinnitus, hearing loss, and sound intolerance tend to report higher levels of handicap and may require more complex rehabilitative strategies.[1,4]

Somatosensory and musculoskeletal comorbidities

Somatosensory and musculoskeletal disorders represent another important comorbidity group. Tinnitus can be modulated by jaw movements, neck position, or muscle tension and is frequently associated with cervical spine dysfunction and temporomandibular disorders.[3,6] Abnormal somatosensory input from the cervical and trigeminal systems converges onto neurons in the DCN, where it can alter firing rates and synchrony and thereby modify tinnitus loudness or pitch.[6] Clinical studies on temporomandibular disorder related orofacial pain further support the relevance of musculoskeletal factors in head and neck symptoms that often coexist with tinnitus.[14]

Systemic, vascular, and metabolic comorbidities

A range of systemic and vascular comorbidities has been described. Advancing age, chronic exposure to loud occupational or recreational noise, hypertension, and cardiovascular disease all increase tinnitus risk and may worsen prognosis.[2,1] These conditions are closely linked to low-grade inflammation and endothelial dysfunction, which aligns with evidence implicating inflammatory mechanisms in tinnitus pathophysiology.[8,9] Metabolic factors and nutritional status also appear relevant. Oxidative stress is elevated in many tinnitus patients, and low dietary intake of antioxidants and trace elements such as zinc and iron has been associated with a higher risk of incident tinnitus over long-term follow-up.[12,15]

Taken together, these findings show that tinnitus should be viewed as a multidimensional condition in which psychological distress, sleep disturbance, hearing loss, somatosensory dysfunction, and systemic disease interact within shared neural and biological networks.[3–6] Comprehensive assessment of comorbidities, including targeted screening for depression, anxiety, sleep problems, hearing impairment, musculoskeletal disorders, cardiovascular risk factors, and nutritional deficits is therefore essential to design individualized, multidisciplinary treatment plans and to improve overall outcomes in patients with chronic tinnitus.[1,2,4,7]

THERAPEUTIC APPROACHES FOR TINNITUS

Given the multifactorial pathophysiology of tinnitus and its frequent psychological and somatic comorbidities, management relies on combining several complementary modalities rather than a single curative treatment.[4,1] In the following sections, three major pillars of care are outlined: (1) psychological and sound-based

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strategies such as cognitive behavioral therapy (CBT), counselling, and tinnitus retraining therapy (TRT); (2) pharmacological treatments targeting cochlear pathology, central hyperexcitability, and mood or anxiety disorders; and (3) traditional, complementary, and herbal interventions, including acupuncture, dietary supplements, and plant-based medicines used as adjunctive therapies.

1. Psychological and sound-based management

Non-pharmacological interventions that target the emotional and perceptual components of tinnitus are central to current management strategies. Contemporary models emphasize that tinnitus distress is driven not only by the phantom sound itself, but also by attentional bias, catastrophic interpretation, and limbic-system hyperactivation.[1,3,5] These insights provide the theoretical basis for CBT, structured counselling, and sound-based approaches such as TRT, which aim to modify maladaptive thoughts and promote habituation to the tinnitus signal.[4,7]

CBT is regarded as the best-supported psychological treatment for chronic, bothersome tinnitus and is recommended by recent clinical guidelines.[1,4] By addressing negative automatic thoughts (e.g. “tinnitus means I am going deaf”), catastrophizing, sleep-related anxiety, and avoidance behaviours, CBT helps patients reinterpret tinnitus as a neutral rather than threatening stimulus.[1,7] Techniques commonly include psychoeducation, cognitive restructuring, exposure to tinnitus-related situations, stress management, and sleep hygiene. Randomized studies show that CBT consistently reduces tinnitus-related distress, depressive and anxiety symptoms, and improves health-related quality of life, even though the perceived loudness of tinnitus often changes little.[4] Internet-based CBT has emerged as a scalable alternative, offering comparable benefits in reducing distress while improving access for patients in remote areas or with limited specialist services.[4]

Counselling is a core component of almost all tinnitus interventions and can be delivered as stand-alone education or as part of structured protocols such as TRT.[1,16] Its goals are to normalize the symptom, explain the neurophysiological model of tinnitus, and correct maladaptive beliefs that maintain heightened vigilance and autonomic arousal.[5,6] By reducing fear and uncertainty, counselling facilitates reclassification of tinnitus from a “danger” signal to a neutral background sound, thereby supporting habituation processes within auditory–limbic networks.[5,1]

Sound-based therapies aim to decrease the contrast between tinnitus and environmental sound, reduce the salience of the phantom percept, and support habituation.[4,1] These interventions range from simple environmental sound enrichment and hearing-aid fitting in patients with hearing loss to the use of dedicated sound generators providing broadband noise or customized acoustic stimuli.[16] Sound therapy can diminish the momentary awareness of tinnitus, facilitate relaxation, and improve sleep, particularly when combined with counselling that

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addresses cognitive and emotional responses to the symptom.[4]

TRT represents a structured combination of directive counselling and long-term sound therapy based on the neurophysiological model proposed by Jastreboff. Counselling sessions focus on explaining auditory and limbic interactions, reassuring patients about the benign nature of tinnitus, and outlining the principles of habituation.[16] Individually adjusted sound generators or hearing aids are then used for many hours per day to provide low-level noise that partially masks tinnitus and continuously exposes the auditory system to non-threatening sound.[10,17] Randomized and controlled trials demonstrate that TRT can lead to clinically meaningful reductions in tinnitus loudness and distress, with some studies reporting decreases in perceived intensity of roughly one-third and in distress of about one-half after 12 months of therapy.[10,17] These benefits tend to increase gradually over time, consistent with a habituation-driven mechanism.[17]

Recent analyses highlight that the relative contribution of counselling and sound generators may vary between individuals.[16] For some patients, intensive counselling alone yields substantial benefit, whereas others require the additive effect of sound enrichment to achieve optimal habituation. Patient selection, presence of hearing loss, baseline psychological comorbidities, and expectations toward therapy appear to influence outcomes, underscoring the need for personalized treatment planning.[1,4,7]

Overall, CBT, counselling, and sound-based interventions, including TRT, represent complementary modalities that target different but overlapping aspects of tinnitus pathophysiology. CBT primarily addresses cognitive and emotional components; counselling provides education and reframing; and sound therapy modulates auditory input and promotes habituation within central auditory pathways. When integrated within a multidisciplinary program that also manages hearing loss and psychological comorbidities, these approaches offer the most robust evidence for improving quality of life in patients with chronic tinnitus.[1,4]

2. Pharmacological treatments

Pharmacological management of tinnitus is largely symptomatic, and currently no drug has been shown to reliably abolish the tinnitus percept in the majority of patients.[4,18] Treatment is therefore directed toward underlying cochlear or systemic disease, reduction of central hyperexcitability, and control of psychological comorbidities.[3,1]

Anti-inflammatory and corticosteroid therapies

Inflammatory mechanisms and immune dysregulation are increasingly recognized as contributors to both cochlear injury and central auditory plasticity.[8] Systemic or intratympanic corticosteroids are most commonly used in the setting of acute tinnitus secondary to sudden sensorineural hearing loss or acoustic trauma.[4,1] Early steroid administration can improve hearing thresholds and, in a subset of patients, parallel reductions in tinnitus loudness and annoyance have been reported.[8,9] However, benefits are less clear in chronic tinnitus, and repeated courses

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carry risks of local and systemic adverse effects.[4,1]

More targeted anti-inflammatory interventions are being explored but results have been disappointing so far. The selective melanocortin-4 receptor agonist RO27-3225, for example, failed to prevent acoustic-trauma–induced tinnitus in animal models, highlighting the complexity and redundancy of inflammatory pathways within the auditory system and the difficulty of translating preclinical findings to clinical benefit.[19] Case reports of successful treatment when tinnitus is driven by overt inflammatory disease suggest that careful aetiologic work-up is crucial to identify rare but reversible inflammatory causes.[9]

Antioxidant and metabolic strategies

Oxidative stress and mitochondrial dysfunction are key downstream consequences of noise exposure, ototoxic drugs, and aging in the cochlea and central auditory pathways.[3,8] This has led to interest in antioxidants and metabolic modulators as disease-modifying therapies. Randomized controlled trials and systematic reviews of combinations containing vitamins C and E, melatonin, and other antioxidant compounds have shown modest but statistically significant reductions in tinnitus loudness and improvements in sleep quality and health-related quality of life, particularly in older adults and in those with concomitant hearing loss.[13,10,11,12] These effects are biologically plausible given experimental evidence that antioxidants limit reactive-oxygen-species-mediated hair-cell and neuronal damage.[12] Nonetheless, studies are heterogeneous in terms of formulations, dosages, and follow-up duration, and placebo effects are substantial.[13,10] Systematic reviews therefore conclude that antioxidant therapy may be considered as an adjunctive option in selected patients but cannot yet be recommended as routine first-line pharmacotherapy.

Psychotropic medications and management of comorbidities

Psychological comorbidities, especially anxiety, depression, and insomnia are highly prevalent among individuals with bothersome tinnitus and form a tightly interconnected symptom network.[7] In this context, psychotropic medications are used primarily to treat comorbid mood and anxiety disorders rather than to directly suppress tinnitus.[4,1,18] Selective serotonin-reuptake inhibitors and serotonin norepinephrine reuptake inhibitors can reduce depressive symptoms, dampen anxiety, and improve sleep continuity, which in turn decreases the perceived intrusiveness of tinnitus.[7,18] Tricyclic antidepressants and benzodiazepines have shown some efficacy in earlier trials, but their use is limited by anticholinergic effects, tolerance, dependence, and daytime sedation.[4,18] Moreover, virtually all classes of psychotropic drugs have been implicated in rare cases of drug-induced or worsened tinnitus, so careful titration and monitoring are required.[1,18] Current guidelines recommend reserving pharmacological treatment of psychiatric comorbidities for patients who meet diagnostic criteria for mood or anxiety disorders, and combining medication with evidence-based psychological therapies such as CBT whenever possible.[7,4,1]

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Other pharmacological agents

A wide range of additional medications, including anticonvulsants, vasodilators, nootropics, and muscle relaxants have been tested over past decades.[18] Meta-analyses and contemporary guidelines, however, indicate that agents such as carbamazepine, gabapentin, betahistine, and several benzodiazepines do not provide consistent, clinically relevant benefit when used broadly for idiopathic tinnitus and may expose patients to unnecessary side effects.[4,1,18] Consequently, their routine use is not recommended outside carefully selected indications (e.g. carbamazepine for "typewriter" tinnitus due to neurovascular compression).[18]

Pharmacological treatments play an important supportive role in a broader, multidisciplinary management strategy. They are most effective when tailored to the individual patient's underlying pathology and comorbidities and combined with counselling, sound therapy, and psychological interventions, rather than used in isolation with the expectation of completely eliminating tinnitus.[4,1,18]

3. Traditional, complementary, and herbal medicine

Acupuncture has been widely used in the treatment of tinnitus due to its ease of application, rapid effects, and relatively low cost. Several clinical studies have shown that acupuncture is effective in reducing primary diseases that cause tinnitus, such as temporomandibular joint disorders, myoclonus, and certain neurological disorders.[21]

Thousands of years of clinical practice have consistently refined and enhanced acupuncture therapy for tinnitus, which is grounded in the principles of Traditional Chinese Medicine. Growing research has demonstrated the protective benefit of acupuncture against tinnitus; nonetheless, tinnitus can be caused by a wide range of diseases, its mechanism is incompletely understood, and its pathways of action are diverse and intricate.[14] Despite these uncertainties, available evidence suggests that acupuncture can be a useful adjunctive treatment for selected patients.[21]

Dietary supplements, vitamins, and minerals

A healthy diet containing various vitamins and minerals can protect against tinnitus, and zinc appears to be particularly relevant. In a large cohort study, lower dietary intakes of zinc and iron were independently associated with a higher 10-year incidence of tinnitus, with risk increases of 44% and 35%, respectively, in those with the lowest intake levels.[15]

Clinical studies have shown that a subset of tinnitus patients have reduced serum zinc, and several trials have evaluated the effects of zinc supplementation on tinnitus outcomes.[22,25] Some randomized and prospective studies report improvements in subjective tinnitus scores after 2–4 months of zinc therapy, especially in individuals with documented hypozincemia or noise-induced hearing-loss-associated tinnitus.[22,23,24] However, a Cochrane review concluded that, when all adults with tinnitus are considered together, oral zinc does not consistently outperform placebo,

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underscoring that any benefit is likely confined to specific subgroups.[26]

Taken together, these data support recommending adequate dietary zinc and iron intake for tinnitus prevention, and considering zinc supplementation only as an adjuvant therapy in carefully selected patients with confirmed or suspected deficiency, under medical supervision to avoid potential adverse effects such as copper depletion or gastrointestinal intolerance.[22,26]

Herbal medicine

The use of herbal medicine in the treatment of disease is widespread, and many plants have been shown to provide beneficial clinical effects. Several narrative and systematic reviews have summarized herbal approaches for tinnitus.[20,32] One of the most widely used agents is *Ginkgo biloba*, often administered alone or in combination with antioxidants. Clinical trials have found that *Ginkgo biloba* can reduce tinnitus in some patients, including those with vascular dementia, possibly by improving microcirculation.[27,20]

Other herbal formulations have also been investigated. A combination of *Salvia hydrangea*, *Citrus aurantium*, *Lippia citriodora*, and elm bark was associated with reduced tinnitus intensity in a randomized clinical trial.[28] Another study showed that a herbal combination containing *Rosa canina*, *Urtica dioica*, and *Tanacetum vulgare* produced significant improvement in tinnitus symptoms compared with placebo.[29] Açaí (*Euterpe oleracea* Martius), a fruit rich in α-tocopherols, fibres, lipids, mineral ions, and polyphenols, has been widely used as an anti-inflammatory and antioxidant. It also contains flavonoids with potential activity in neurodegenerative diseases such as Parkinson's disease.[30] Oral antioxidant supplementation with açaí extract exhibited favourable effects on tinnitus, lowering discomfort regardless of the underlying aetiology, and can be regarded as a promising therapeutic modality.[31]

Traditional Iranian medicine also uses certain plants to treat earache and tinnitus. For example, *Calendula officinalis* or *Calendula persica* is used as an independent preparation to treat earaches; medicinal plants and their derivatives may decrease the prevalence of earaches and tinnitus, possibly due to inhibition of reactive-oxygen-species production and anti-inflammatory activity.[32]

A more recent Chinese medicinal formulation, Gushen Pian, is being utilized in the therapy of tinnitus and sensorineural hearing loss. Its proposed mechanisms include correction of splenonephric hypofunction and resolution of phlegm-accumulation stasis. Clinical investigation suggests that Gushen Pian may improve hearing loss and tinnitus with no obvious side effects.[33]

Overall, although herbal and traditional medicines show promise and are increasingly popular among patients, the quality of evidence is variable, and rigorous randomized controlled trials with standardized outcome measures are still needed.

LIMITATIONS AND CHALLENGES

Major limitations in the current tinnitus literature include highly variable individual responses to treatment, absence of objective biomarkers, and inconsistent

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outcome definitions across studies. The wide variation in tinnitus aetiology, comorbidities, and psychological coping styles further complicates interpretation of clinical trials and contributes to conflicting findings, especially in work that evaluates pharmacological and herbal therapies. The narrative synthesis presented in this review points toward the need for a personalized-medicine approach, in which interventions are selected and combined according to clinical profile, hearing status, psychological comorbidities, and patient preferences.

CONCLUSION

Tinnitus is underpinned by a multifaceted pathophysiology involving peripheral and central auditory dysfunction, maladaptive neuroplasticity, and the engagement of emotional and somatosensory brain systems.[3,4,5] The strong link between tinnitus and psychological disorders necessitates an integrated, multidisciplinary approach to treatment.[1,8] Therapeutic strategies now span pharmacological and nonpharmacological interventions, including CBT, TRT, anti-inflammatory and antioxidant therapies, antidepressants, traditional medicine, and herbal medicine.[1,12,21]

Prevention of tinnitus risk can also be supported by consuming adequate iron and zinc.[2,23,25] As the prevalence of tinnitus is expected to increase with population ageing, effective strategies are needed to reduce the burden of severe symptoms.[2] Studies show that a diet low in iron and zinc can increase the risk of tinnitus in older adults over ten years.[25]

The use of herbal medicines is popular in the treatment of tinnitus because many agents have shown favourable clinical effects. However, further research needs to consider the varying responses of tinnitus itself.[15,16] To reduce population response variation and determine which patient subpopulations benefit from a treatment, strict subject selection for clinical studies is recommended due to the multidimensional nature of tinnitus.[1,20] Future research should also include quality-of-life indicators because tinnitus affects patients' daily functioning.[1] The quality of currently available data on herbal medicines for tinnitus is generally low, and findings from randomized controlled trials are inconsistent.[23,24] Nevertheless, there is a clear need for safe and effective pharmacological treatments for tinnitus.[14,20] It is anticipated that advances in basic science research on the mechanisms of tinnitus and additional phytochemical and biological characterization of herbal medicines will be beneficial in the future.[16,27,30]

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