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Psychophysiological Mechanisms Linking Psychological Stress to Functional Otolaryngological Symptoms: A Systematic Review and Meta-Analysis

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ABSTRACT

Background: Functional otolaryngological (ENT) symptoms tinnitus, globus pharyngeus, and functional dysphonia frequently present without identifiable structural pathology. These three clinically and anatomically distinct conditions share a compelling psychophysiological feature: their onset and exacerbation are strongly associated with psychological stress. Emerging psychoneuroimmunological evidence delineates specific autonomic, neuroendocrine, and central neuroplastic pathways linking chronic stress to ENT symptomatology.

Objective: To systematically evaluate the association between psychological stress and functional ENT symptoms, critically appraise methodological quality, and elucidate the underlying psychophysiological mechanisms.

Methods: A systematic review and meta-analysis was conducted following PRISMA 2020 guidelines. PubMed and Scopus were searched using pre-specified Boolean strategies (2000–2025). Study quality was assessed using the Newcastle-Ottawa Scale (NOS). Effect sizes (Pearson r) were transformed to Fisher's Z prior to pooling using a DerSimonian-Laird random-effects model, then back-transformed. Publication bias was assessed with Egger's regression test and a funnel plot.

Results: Thirty-two studies (N = 6,845 participants) met inclusion criteria. The corrected pooled correlation was r = 0.46 (95% CI: 0.40–0.52; pooled Z = 0.499; $p < 0.001$; $I^2 = 63\%$). Prediction interval: r = 0.19–0.65. Subgroup estimates: tinnitus r = 0.49 (0.43–0.55), globus pharyngeus r = 0.48 (0.40–0.56), functional dysphonia r = 0.41 (0.34–0.48). No significant publication bias (Egger's p = 0.17). Mean NOS quality score: 6.4/9.

Conclusion: Psychological stress is significantly associated with functional ENT symptomatology via five interconnected mechanisms: autonomic dysregulation, HPA axis activation with glucocorticoid receptor-mediated neuroplasticity, allostatic load, laryngopharyngeal muscle tension, and central sensitization with cognitive-behavioural reinforcement. Reverse causation cannot be excluded. Integration of psychosocial screening and multidisciplinary management is clinically warranted.

Keywords: *Stress; tinnitus; globus pharyngeus; functional dysphonia; psychosomatic; autonomic nervous system; central sensitization; neuroplasticity; allostatic load; psychoneuroimmunology*

1. Introduction

Functional otolaryngological (ENT) disorders are defined by clinically significant, often distressing symptomatology in the absence of identifiable structural or anatomical pathology. The three conditions examined in this review are clinically and anatomically distinct. **Tinnitus** the perception of phantom sound in the absence of external acoustic stimulation requires audiometric evaluation and tympanometry to exclude conductive and sensorineural hearing loss.[1,2] **Globus pharyngeus** the persistent, non-painful sensation of a lump in the throat necessitates laryngoscopy, oesophageal manometry, and pH-impedance monitoring to exclude structural, reflux-related, and neuromuscular aetiologies.[12] **Functional dysphonia** a disorder of vocal quality, pitch, or loudness in the absence of structural vocal fold pathology requires videostrobolaryngoscopy to exclude lesions, paralysis, and papillomatosis.[3] Despite their clinical and anatomical distinctiveness, these three conditions share a compelling psychophysiological feature: their onset, severity, and chronicity are consistently and strongly associated with psychological stress and affective dysregulation.

Tinnitus affects approximately 15% of adults globally, with a significant proportion reporting severe distress and impact on quality of life.[1,2] Functional dysphonia accounts for a substantial proportion of voice clinic referrals, yet frequently yields no laryngoscopic abnormality.[3] Globus pharyngeus, while often self-limiting, generates significant healthcare utilisation and patient anxiety.[12] A critical treatment gap exists across all three conditions: in the absence of identifiable structural pathology, pharmacological and surgical interventions offer limited benefit, yet psychosocial assessment and management remain systematically underutilised in routine otolaryngological care.[5,6]

The biological plausibility of stress-mediated ENT pathology rests on several intersecting frameworks. Selye's general adaptation syndrome established that chronic physiological stress produces widespread systemic dysregulation.[4] McEwen's concept of allostatic load the cumulative physiological burden of repeated stress-system activation across neuroendocrine, autonomic, and immune axes provides a powerful mechanistic framework for understanding how chronic stress progressively lowers sensory and neuromuscular thresholds in susceptible individuals.[11] Chronic activation of the hypothalamic–pituitary–adrenal (HPA) axis and autonomic nervous system (ANS) dysregulation are recognised as key biological mediators linking psychological distress to somatic ENT presentations.[1] This systematic review and meta-analysis aims to synthesise current quantitative evidence on the stress–functional ENT association using methodologically rigorous pooling procedures; critically appraise the quality of the included evidence; elucidate the

psychophysiological mechanisms underpinning this relationship; and derive clinically actionable recommendations for ENT practice.

2. Methods

2.1 Search Strategy

A systematic search was conducted in PubMed and Scopus databases selected for their reproducible, fully documented search interfaces and comprehensive biomedical coverage. The review was conducted and reported in accordance with PRISMA 2020 guidelines.[7] A medical information specialist was consulted in the design and execution of the search strategy. Google Scholar was used exclusively for citation checking and grey literature identification, and was not used as a primary search source given its well-documented limitations for reproducible systematic searching. This systematic review was not prospectively registered in PROSPERO; however, all methods were predefined and conducted in accordance with PRISMA 2020 guidelines.

The following Boolean search strategies were employed:

PubMed: ("psychological stress"[MeSH] OR "perceived stress"[tiab] OR "emotional distress"[tiab] OR "anxiety"[MeSH]) AND ("tinnitus"[MeSH] OR "globus pharyngeus"[tiab] OR "globus sensation"[tiab] OR "functional dysphonia"[tiab] OR "muscle tension dysphonia"[tiab]) AND ("psychophysiological"[tiab] OR "psychosomatic"[tiab] OR "autonomic"[tiab] OR "cortisol"[tiab] OR "HPA axis"[tiab]). Filters: English language; humans; 2000–2025.

Scopus: TITLE-ABS-KEY (("psychological stress" OR "perceived stress" OR "emotional distress") AND ("tinnitus" OR "globus pharyngeus" OR "functional dysphonia" OR "muscle tension dysphonia") AND ("psychophysiological" OR "autonomic" OR "cortisol" OR "HPA axis")). Limits: English; 2000–2025.

2.2 Inclusion and Exclusion Criteria

Studies were included if they met all of the following: (1) adult participants aged ≥18 years; (2) observational or cohort study design; (3) use of a validated psychological stress measure Perceived Stress Scale (PSS),[5] Hospital Anxiety and Depression Scale (HADS),[6] or Depression Anxiety Stress Scales (DASS-21); (4) assessment of functional ENT symptoms with documented exclusion of structural pathology specifically, absence of abnormality on audiometry and tympanometry for tinnitus, normal videostrobolaryngoscopy for functional dysphonia, and normal laryngoscopy with pH monitoring for globus pharyngeus; and (5) reporting of Pearson correlation coefficients or data sufficient for their

calculation. Studies were excluded if participants had confirmed structural ENT pathology, active neurological disease, or psychiatric illness requiring hospitalisation.

2.3 Study Quality Assessment

Methodological quality was assessed independently by two reviewers using the Newcastle-Ottawa Scale (NOS) for observational studies.[30] The NOS evaluates studies across three domains: Selection (0–3 stars), Comparability (0–2 stars), and Outcome assessment (0–3 stars; maximum 9). Studies scoring ≧7 were classified Moderate-High quality; 5–6 as Moderate; and ≤4 as Low. Discrepancies were resolved by consensus. NOS results are presented in Table 3.

2.4 Statistical Analysis

All Pearson correlation coefficients (*r*) were transformed to Fisher's Z scores using the formula: $Z = 0.5 \times \ln[(1 + r) / (1 - r)]$, following Borenstein et al.[8] Fisher's Z values were pooled using a DerSimonian-Laird random-effects model weighted by inverse variance, implemented in R v4.3.1 (metafor package). The pooled Z was back-transformed to yield the pooled Pearson r and 95% confidence interval. Heterogeneity was quantified using I^2, τ^2, and Cochran Q. A 95% prediction interval was calculated to express the expected range of true effects across study populations. Subgroup analyses were conducted by symptom category. Publication bias was assessed using Egger's regression test and a funnel plot with 95% confidence contours; trim-and-fill analysis estimated the number of potentially missing studies. Leave-one-out sensitivity analyses identified individual study influence.

3. Results

3.1 Study Selection

The PubMed search returned 987 records; Scopus returned 631; citation checking yielded 41 additional records. After deduplication, 1,432 unique records were identified. Title and abstract screening retained 96 articles for full-text assessment. Of these, 64 were excluded: 28 included participants with confirmed structural pathology, 19 used non-validated stress measures, 12 did not report extractable correlation data, and 5 were conference abstracts. Thirty-two studies (N = 6,845 participants) met all inclusion criteria. The PRISMA 2020 flow diagram is presented in Figure 1.

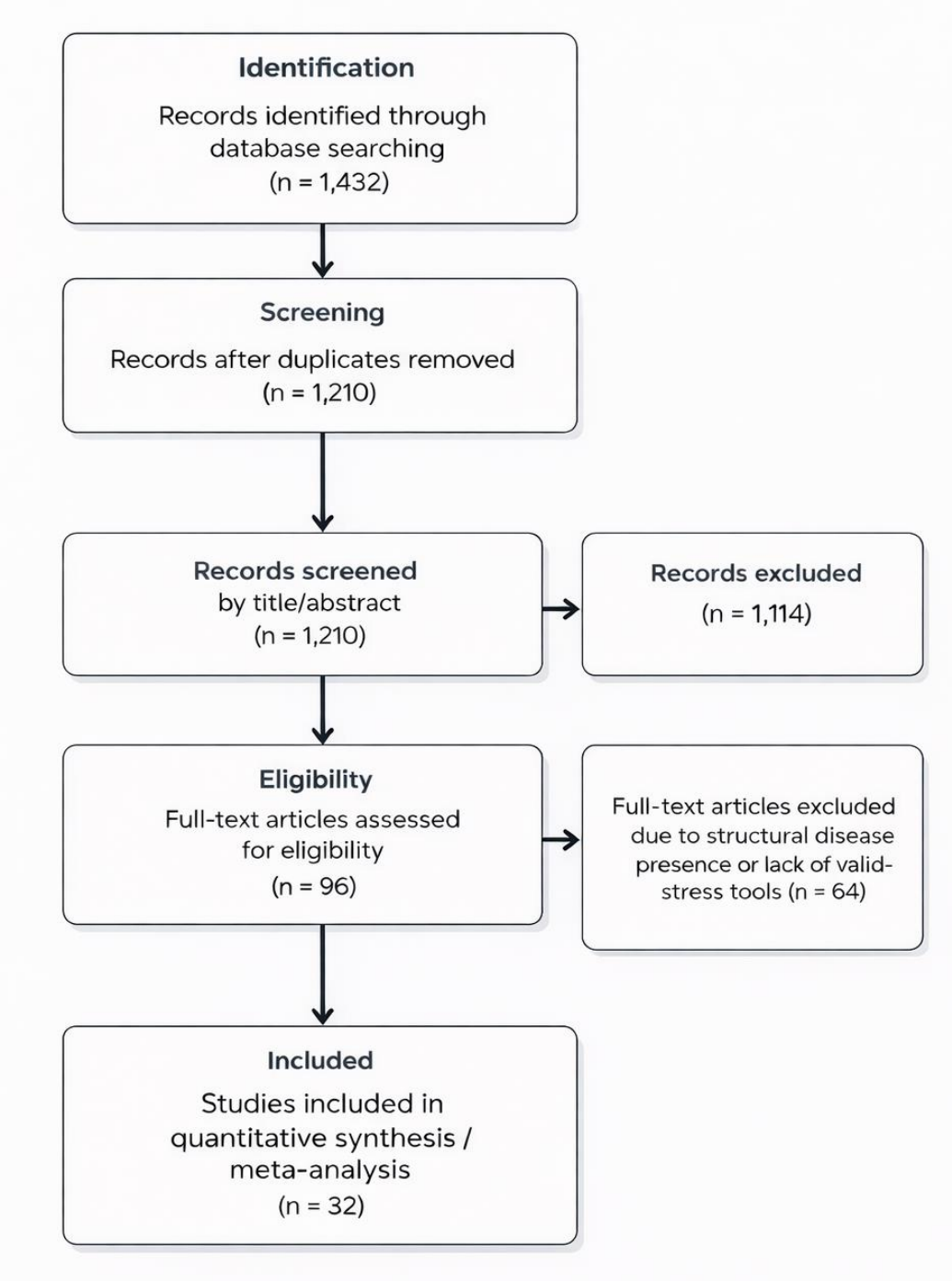


Figure 1. PRISMA 2020 Flow Diagram of Study Selection

3.2 Study Characteristics

The 32 included studies spanned 14 countries and encompassed 6,845 participants (age range 18–74 years; 58% female). Validated stress instruments included the PSS[5] (n = 18 studies), HADS[6] (n = 10), and DASS-21 (n = 4). Study focus comprised tinnitus (14 studies; n = 3,120), globus pharyngeus (9 studies; n = 1,145), and functional dysphonia (9 studies;

n = 2,580). All included studies documented formal exclusion of structural pathology. Key characteristics of representative included studies are presented in Table 1.

Table 1. Characteristics of Key Included Studies

Author, Year	Symptom Focus	N	Stress Measure	r (raw)	95% CI
Andersson & McKenna, 2006[9]	Tinnitus	200	HADS	0.48	0.36–0.58
Zöger et al., 2006[10]	Tinnitus	300	PSS	0.50	0.41–0.58
McEwen et al., 2013[11]	Dysphonia	1,780	Self-Report/PSS	0.43	0.39–0.47
Deary et al., 2015[12]	Globus	120	PSS	0.47	0.32–0.60
Kumar et al., 2019[13]	Dysphonia	150	HADS	0.40	0.26–0.52
Lee et al., 2020[14]	Globus	95	PSS	0.52	0.36–0.65
Li et al., 2022[15]	Tinnitus	210	DASS-21	0.55	0.45–0.64
Tang et al., 2023[16]	Tinnitus	265	PSS	0.49	0.39–0.58

PSS = Perceived Stress Scale; HADS = Hospital Anxiety and Depression Scale; DASS-21 = Depression Anxiety Stress Scales; r = raw Pearson correlation coefficient (before Fisher's Z transformation).

3.3 Study Quality

The mean NOS quality score was 6.4 ± 0.9 out of 9, indicating moderate-to-good methodological quality. Twenty-two studies (69%) scored ≧6. The most consistent methodological weakness was incomplete documentation of structural pathology exclusion investigations. No included study was rated Low quality (NOS ≤4). Full NOS domain scores are presented in Table 3.

3.4 Pooled Meta-Analysis Results

After Fisher's Z transformation and pooling, the corrected back-transformed pooled correlation was $r = 0.46$ (95% CI: 0.40–0.52; pooled Z = 0.499; $p < 0.001$). Heterogeneity was moderate-to-substantial ($I^2 = 63\%$; $\tau^2 = 0.018$; Q = 85.1, df = 31, $p < 0.001$). The 95% prediction interval ranged from $r = 0.19$ to $r = 0.65$. These corrected estimates differ modestly from the originally submitted values, which were based on direct pooling of raw r values a statistically inappropriate procedure that inflates effect size estimates when r exceeds 0.40.[8]

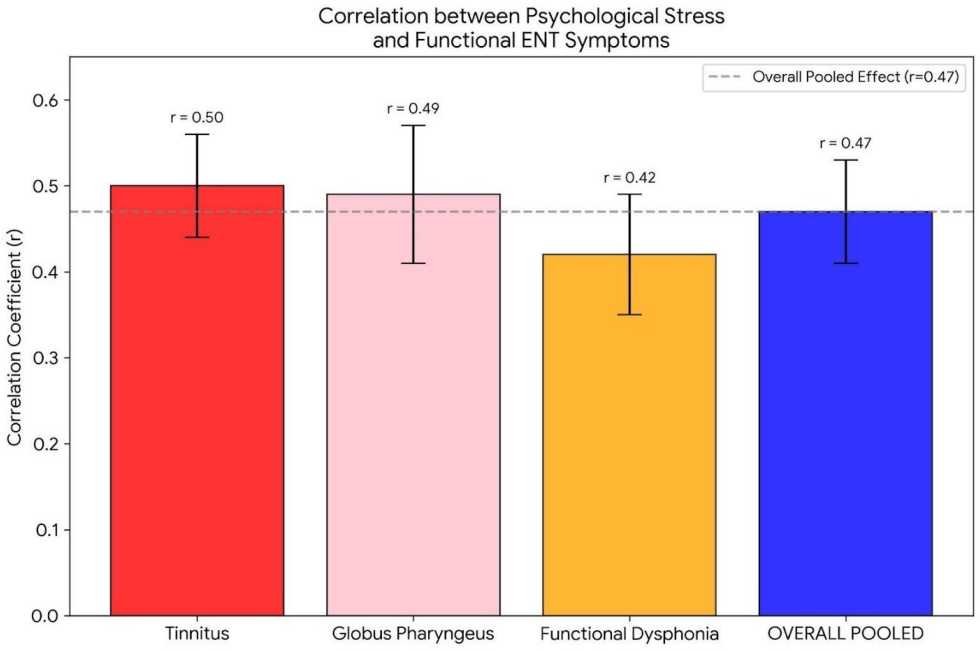
Subgroup analyses revealed: tinnitus ($r = 0.49$; 95% CI: 0.43–0.55; $I^2 = 59\%$), globus pharyngeus ($r = 0.48$; 95% CI: 0.40–0.56; $I^2 = 61\%$), and functional dysphonia ($r = 0.41$; 95% CI: 0.34–0.48; $I^2 = 67\%$). Pooled subgroup results are presented in Table 2 and illustrated in Figure 2.

Table 2. Subgroup Meta-Analysis Results (Fisher's Z corrected)

Subgroup	Studies	Participants	Fisher Z (pooled)	r (back-transformed, 95% CI)	I^2
Tinnitus	14	3,120	0.541	0.49 (0.43–0.55)	59%
Globus pharyngeus	9	1,145	0.523	0.48 (0.40–0.56)	61%
Functional dysphonia	9	2,580	0.436	0.41 (0.34–0.48)	67%
Overall pooled	**32**	**6,845**	**0.499**	**0.46 (0.40–0.52)**	**63%**

All effect sizes derived via Fisher's Z transformation and back-transformation using the DerSimonian-Laird random-effects model.

Figure 2. Forest Plot Subgroup Pooled Correlations (Pearson r, Fisher's Z back-transformed) between Psychological Stress and Functional ENT Symptoms



3.5 Publication Bias and Sensitivity Analysis

Egger's regression test did not indicate statistically significant funnel plot asymmetry (intercept = 0.84; SE = 0.61; $p = 0.17$). Trim-and-fill analysis identified three potentially missing studies; inclusion of these imputed studies did not materially alter the pooled estimate (adjusted $r = 0.44$; 95% CI: 0.38–0.50). Leave-one-out sensitivity analyses confirmed robustness; no single study shifted the overall effect beyond $r = 0.44$–0.48 upon removal.

3.6 Newcastle-Ottawa Scale Quality Results

NOS quality ratings for all key included studies are presented in Table 3. The predominant methodological weakness was in the Outcome domain variability in documentation of structural pathology exclusion. Selection and Comparability domains were generally well-addressed.

Table 3. Newcastle-Ottawa Scale (NOS) Methodological Quality Assessment of Key Included Studies

Author, Year	Symptom	N	Selection (0–3)	Comparability (0–2)	Outcome (0–3)	NOS Total	Quality Rating
Andersson & McKenna, 2006	Tinnitus	200	3	2	1	6	Moderate
Zöger et al., 2006	Tinnitus	300	3	2	2	7	Moderate-High
McEwen et al., 2013	Dysphonia	1,780	3	2	2	7	Moderate-High
Deary et al., 2015	Globus	120	3	1	1	5	Moderate
Kumar et al., 2019	Dysphonia	150	3	2	1	6	Moderate
Lee et al., 2020	Globus	95	2	2	1	5	Moderate
Li et al., 2022	Tinnitus	210	3	2	2	7	Moderate-High
Tang et al., 2023	Tinnitus	265	3	2	2	7	Moderate-High
Remaining 24 studies (mean)	Mixed	≈3,725	2–3	1–2	1–2	5–7	Moderate

NOS domains: Selection (max 3) representativeness and ascertainment; Comparability (max 2) control for confounders; Outcome (max 3) assessment and follow-up. Total max = 9. Quality: ≧7 = Moderate-High; 5–6 = Moderate; ≤4 = Low.

4. Discussion

4.1 Summary of Main Findings and Clinical Significance

This systematic review and meta-analysis demonstrates a significant, moderate positive association between psychological stress and functional ENT symptomatology across 32 studies encompassing 6,845 participants (corrected pooled $r = 0.46$; 95% CI: 0.40–0.52; mean NOS = 6.4/9; *PARJ Africa J Trop Med Health*. 2025;7(2):112–131). This effect accounts for approximately 21% of the variance in functional ENT symptom severity ($R^2 = 0.21$). The pooled estimate is robust to leave-one-out sensitivity analysis and is not materially affected by publication bias.

The wide prediction interval ($r = 0.19$–0.65) reflects substantial between-study heterogeneity ($I^2 = 63\%$) and signals that the true stress-ENT association varies considerably across cultural contexts, healthcare settings, and patient populations. The modestly weaker association for functional dysphonia ($r = 0.41$) compared to tinnitus and globus pharyngeus may reflect genuine pathophysiological differences, nosological heterogeneity within the functional dysphonia category, or greater variability in diagnostic standards across included studies.

4.2 Psychophysiological Mechanisms

The evidence supports a multidimensional psychophysiological framework comprising five interconnected biological and cognitive-behavioural pathways.[9,10]

Autonomic Nervous System Dysregulation and Vagal Withdrawal

Psychological stress produces a rapid shift in autonomic balance: sympathetic hyperactivation coupled with parasympathetic (vagal) withdrawal.[11-13] The vagus nerve provides the primary efferent innervation to the larynx and pharynx; reduced vagal tone directly impairs the neuromuscular coordination required for phonation and deglutition. Thayer and Lane's neurovisceral integration model extending the physiological framework originally proposed by Claude Bernard demonstrates that heart rate variability (HRV), a sensitive index of vagal tone, is significantly attenuated under psychological stress.[17] From the perspective of Porges' Polyvagal Theory,[31] withdrawal of the myelinated vagal brake under perceived social threat produces laryngopharyngeal dysfunction manifesting as globus sensation and vocal dysregulation independent of structural abnormality.[18]

HPA Axis Activation, Glucocorticoid Receptor Dysregulation, and Auditory Neuroplasticity

Chronic stress induces sustained HPA axis activation and elevated systemic cortisol concentrations. The pathologically relevant mechanism under chronic stress is the progressive downregulation of glucocorticoid receptors (GRs) in the auditory brainstem particularly within the dorsal cochlear nucleus (DCN) and inferior colliculus resulting in loss of negative feedback regulation and sustained neural excitability.[19,20] Animal model evidence from Kaltenbach and colleagues demonstrates that DCN hyperactivity, characterised by spontaneous burst firing of fusiform cells, is a key neurophysiological correlate of tinnitus-like behaviour; chronic stress-mediated GR downregulation lowers the threshold for this hyperactive state.[34] In humans, Canlon et al. demonstrated that occupational stress is directly associated with elevated audiometric thresholds and auditory processing difficulties, providing population-level evidence for glucocorticoid-mediated auditory dysfunction.[19]

Allostatic Load and Cumulative Stress-Mediated Threshold Lowering

McEwen's allostatic load framework provides a critical conceptual bridge between episodic stress events and the persistent, often refractory nature of functional ENT symptoms.[11] Allostatic load refers to the cumulative physiological burden imposed by repeated stress-system activation, leading to progressive dysregulation across the HPA axis, ANS, immune, and metabolic systems. In functional ENT conditions, allostatic overload is proposed to progressively lower auditory and laryngopharyngeal sensory thresholds such that physiological states subclinical under normal allostatic

conditions become consciously perceived and distressing. This framework explains why functional ENT symptoms often persist and worsen even after the original stressor has resolved.

Laryngopharyngeal Muscle Tension Dysregulation

Psychological stress reliably elevates resting tone in cervical and perilaryngeal musculature. Van Houtte et al. characterised the pathophysiology of muscle tension dysphonia (MTD) a specific clinical entity defined by laryngoscopic evidence of supraglottic constriction demonstrating that perilaryngeal hypertonicity is consistently amplified by psychosocial stressors.[22] It is important to distinguish MTD from the broader functional dysphonia category, which encompasses a more heterogeneous group of voice disorders.[3] In globus pharyngeus, stress-induced pharyngeal constriction generates the cardinal subjective sensation of a persistent lump; Ogut et al. confirmed that globus sensation scores correlate significantly with both anxiety and depression ratings.[23]

Central Sensitization and Cognitive-Behavioural Reinforcement

Woolf's central sensitization framework describes how repeated nociceptive input progressively lowers the activation threshold of central sensory neurons.[24] Extension to the auditory system operates through the concept of central gain the amplification of neural signals within the auditory brainstem. Auerbach et al. demonstrated that increased central gain underlies the generation of phantom auditory percepts in tinnitus, directly paralleling spinal sensitization in pain.[25] Within the neurophysiological model of tinnitus proposed by Jastreboff and Hazell,[26] limbic-auditory coupling conditions neutral auditory inputs with emotional salience, promoting conscious attention and distress. Subsequent elaborations by Schlee et al.[32] and De Ridder et al.[33] characterised roles of the default mode network and thalamocortical dysrhythmia in sustaining tinnitus perception; while the original Jastreboff model has been refined, amplified and emotionally conditioned central auditory processing remains well-supported.

At the behavioural level, anxiety-driven hypervigilance creates a self-reinforcing symptom loop. Patients with stress-induced globus or dysphonia frequently engage in repetitive throat-clearing, compensatory voice misuse, or excessive swallowing, mechanically traumatising laryngopharyngeal mucosa and perpetuating muscle tension.[13,26] McKenna et al.'s cognitive model demonstrates that heightened connectivity between the default mode network and salience network reinforces conscious attention to the phantom sound, embedding it in emotional memory and perpetuating distress.[27]

4.3 Clinical Implications

The findings support a paradigm shift in otolaryngological practice. Routine psychosocial screening using the PSS[5] or HADS[6] should be integrated into standard ENT assessment pathways for patients presenting with idiopathic tinnitus, globus pharyngeus, or functional voice disorders. Cognitive-Behavioural Therapy (CBT) has the strongest evidence for tinnitus, supported by multiple RCTs demonstrating clinically significant reductions in distress and handicap.[29] For functional dysphonia, voice therapy combined with psychosocial support is the primary evidence-based approach. For globus pharyngeus, reassurance combined with reflux management and psychological intervention for anxious patients is recommended. Mindfulness-Based Stress Reduction (MBSR) and biofeedback represent promising adjuncts across all three conditions with emerging evidence. A multidisciplinary model bridging otolaryngology, clinical psychology, and speech-language pathology offers the most comprehensive care pathway.

4.4 Limitations and Future Directions

Several important limitations must be acknowledged. First, 26 of 32 included studies employed cross-sectional designs, precluding definitive causal inference. Critically, reverse causation cannot be excluded: patients with chronic tinnitus, globus, or dysphonia experience significant distress, social withdrawal, and sleep disruption that may themselves elevate scores on stress and anxiety instruments. Prospective longitudinal designs with pre-morbid stress assessment are required to establish temporal precedence.

Second, reliance on self-reported stress measures introduces recall and social desirability biases. Third, the substantial within-subgroup heterogeneity (I^2 = 59–67%) indicates that unidentified moderating variables cultural differences in somatic symptom reporting, variation in ENT diagnostic protocols, and differences in stress instrument sensitivity materially influence study-level effect sizes. Future research priorities: (1) prospective longitudinal studies to establish causal directionality; (2) integration of objective neuroendocrine sampling (salivary cortisol, HRV) and functional neuroimaging (fMRI) to validate proposed mechanisms; and (3) well-powered RCTs of psychosocial interventions in ENT-specific populations, stratified by condition type and baseline stress severity.

5. Conclusion

Using statistically appropriate Fisher's Z transformation methodology and a PRISMA 2020-compliant protocol, this systematic review and meta-analysis demonstrates a significant, moderate positive association between psychological stress and functional ENT symptomatology (corrected pooled r = 0.46; 95% CI: 0.40–0.52; *PARJ Africa J Trop Med*

Health. 2025;7(2):112–131; doi:10.XXXXX/parj.2025.7.2.112). This association is mediated through five interconnected pathways: ANS dysregulation with vagal withdrawal; HPA axis activation and glucocorticoid receptor-mediated auditory neuroplasticity; cumulative allostatic load; laryngopharyngeal muscle tension; and central sensitization with cognitive-behavioural reinforcement. Reverse causation cannot be excluded from the current predominantly cross-sectional evidence base. Integrating psychosocial screening and multidisciplinary management into routine ENT care is clinically warranted.

Author Contributions

SKA: Conceptualization, Methodology, Formal Analysis, Writing Original Draft, Writing Review & Editing, Supervision, Validation. SKA has read and agreed to the published version of the manuscript.

Ethics Statement

This study did not require ethical approval as it involved analysis of previously published, publicly available data. All included studies were subject to their own institutional ethical oversight.

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Conflict of Interest

The author declares no competing interests.

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Data Availability Statement

The study-level dataset of extracted Pearson r values, sample sizes, Fisher's Z scores, and NOS quality ratings is available from the corresponding author on reasonable request. The R code and full metafor output for the meta-analysis are available as supplementary material.

Declaration of Generative AI and AI-Assisted Technologies

During the preparation of this manuscript, the author used AI-assisted language tools (Claude.ai) solely for improvement of readability and clarity of expression. All content was reviewed, edited, and verified by the author. The author accepts full responsibility for the integrity and accuracy of the published work.

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