



Review

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Orofacial myofunctional therapy and pharmaceutical interventions in obstructive sleep apnea: Mechanisms and translational perspectives

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Abstract:

Orofacial myofunctional therapy (OMT) has emerged as a promising non-pharmacological intervention to improve upper airway function in sleep-disordered breathing. Pharmaceutical sleep medicine mainly targets inflammatory, neuromuscular and respiratory pathways linked to disease progression. Both approaches share key biological mechanisms, including intermittent hypoxia, oxidative stress, inflammation, neuromuscular dysfunction and airway remodeling that contribute to obstructive sleep apnea. OMT enhances tongue posture, muscle performance, nasal breathing and neuromuscular plasticity, while drugs modulate inflammatory mediators, oxidative stress and respiratory control. Thus, integrating OMT with targeted pharmacotherapy may improve airway stability and support personalized multidisciplinary management of sleep-disordered breathing.

Keywords: Orofacial myofunctional therapy (OMT), obstructive sleep apnea (OSA), sleep-disordered breathing (SDB), upper airway stability, pharmaceutical sleep medicine, neuromuscular adaptation

Background:

Sleep-disordered breathing (SDB), particularly obstructive sleep apnea (OSA), is characterized by recurrent upper airway collapse, intermittent hypoxia and sleep fragmentation that adversely affect cardiovascular, metabolic and neurocognitive health [1]. OSA affects approximately one billion adults worldwide and remains one of the most prevalent chronic sleep disorders [2]. Increasing evidence indicates that OSA is a multifactorial biological disorder involving impaired neuromuscular control, oxidative stress, chronic inflammation and structural remodeling of the upper airway [3]. Upper airway patency is maintained by coordinated activation of the genioglossus, tensor veli palatini and other pharyngeal dilator muscles during inspiration [4]. Reduced neural drive to these muscles during sleep increases pharyngeal collapsibility and promotes repetitive obstructive events [5]. Cyclic hypoxia and reoxygenation generate excessive reactive oxygen species (ROS), initiating oxidative stress and mitochondrial dysfunction [6]. Oxidative stress activates hypoxia-inducible factor-1 α (HIF-1 α), nuclear factor-kappa B (NF- κ B) and related inflammatory signaling pathways [7]. These molecular responses increase the expression of tumor necrosis factor- α , interleukin-6 and other inflammatory mediators that contribute to endothelial dysfunction and extracellular matrix remodeling [8]. Orofacial myofunctional therapy (OMT) is a rehabilitation strategy designed to improve tongue posture, nasal breathing, swallowing function and upper airway muscle performance [9]. Functional adaptation of the oropharyngeal musculature enhances airway stability and reduces upper airway collapsibility during sleep [10]. Clinical investigations have reported improvements in snoring severity, apnea-hypopnea index and daytime somnolence following structured OMT programmes [11]. Pharmaceutical sleep medicine complements these functional adaptations through therapies targeting inflammatory pathways, oxidative stress, respiratory control and neuromuscular regulation [12]. Emerging evidence suggests that combining rehabilitation with pharmacological interventions may improve upper airway function by simultaneously

targeting biomechanical dysfunction and molecular mechanisms underlying disease progression [13]. Therefore, it is of interest to evaluate the potential synergistic benefits of integrating orofacial myofunctional therapy with pharmaceutical sleep medicine for the management of sleep-disordered breathing.

Neuromuscular basis of orofacial myofunctional therapy (Figure 1):

Upper airway stability depends on coordinated activation of the genioglossus, soft palate and pharyngeal dilator muscles during inspiration [4]. Reduced neural drive during sleep increases pharyngeal collapsibility and contributes to recurrent airway obstruction in obstructive sleep apnea [5]. Orofacial myofunctional therapy (OMT) improves tongue posture, swallowing, nasal breathing and functional coordination of the oropharyngeal muscles through repetitive neuromuscular training [9, 14]. These adaptations enhance upper airway muscle performance, increase airway patency and reduce pharyngeal collapse during sleep [10, 17]. Improved muscle function may also optimize respiratory reflexes and upper airway biomechanics, supporting long-term functional stability [15]. The complementary effects of OMT provide a biological rationale for combining neuromuscular rehabilitation with pharmacological therapies targeting inflammation, oxidative stress and respiratory regulation in sleep-disordered breathing [13, 16].

Inflammatory and molecular mechanisms:

Intermittent hypoxia in obstructive sleep apnea initiates oxidative stress through excessive production of reactive oxygen species, leading to persistent inflammatory activation [6]. Oxidative stress stimulates hypoxia-inducible factor-1 α (HIF-1 α), nuclear factor-kappa B (NF- κ B) and mitogen-activated protein kinase signaling, increasing the expression of tumor necrosis factor- α , interleukin-6 and other inflammatory mediators [7]. These molecular responses promote endothelial dysfunction, extracellular matrix remodeling and progressive upper airway instability [8].

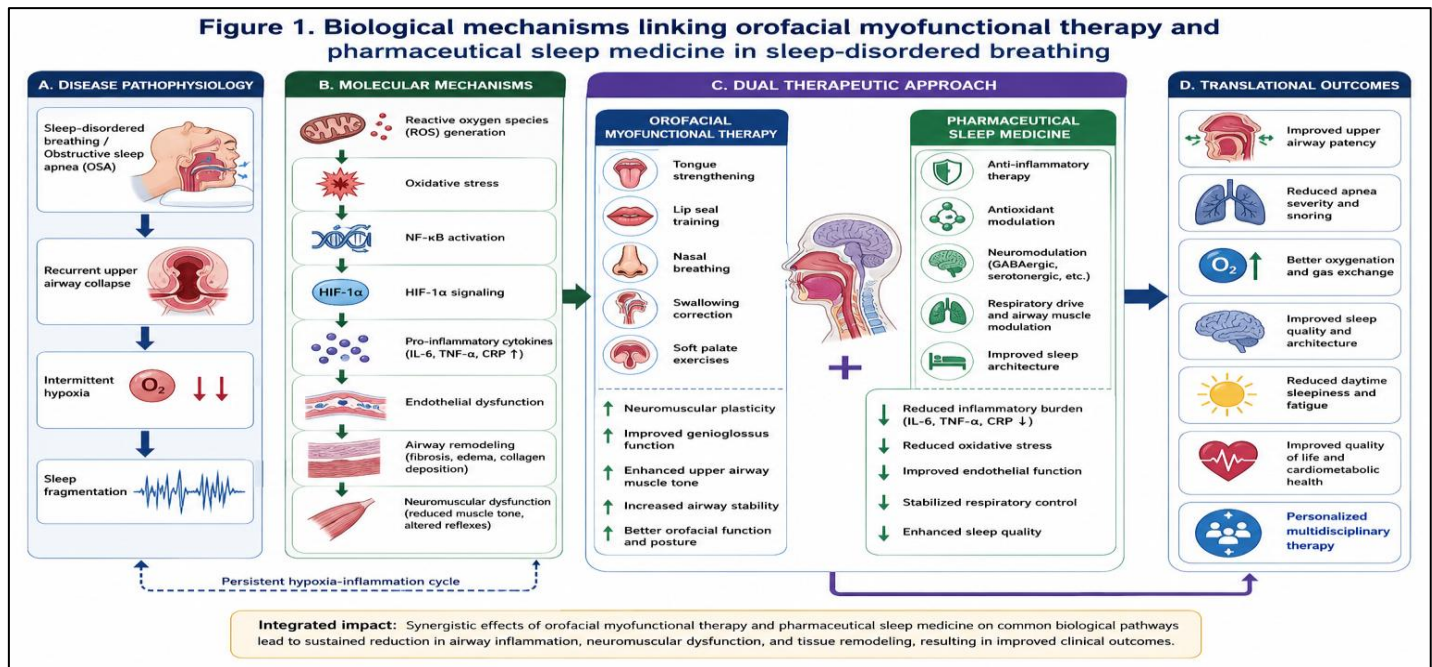


Figure 1: Proposed biological links between orofacial myofunctional therapy and pharmacological interventions in obstructive sleep apnea. The schematic summarizes major pathophysiological mechanisms, potential therapeutic targets and proposed translational pathways

Orofacial myofunctional therapy may reduce repetitive hypoxic stress by improving upper airway muscle function and maintaining airway patency during sleep [10]. Pharmaceutical sleep medicine complements these biological adaptations through therapies targeting inflammatory signaling, oxidative stress and respiratory regulation [12]. Together, neuromuscular rehabilitation and pharmacological interventions influence complementary molecular pathways, providing a biological basis for integrated management of sleep-disordered breathing [13, 18].

Translational perspectives:

Emerging evidence supports the integration of orofacial myofunctional therapy (OMT) with pharmaceutical sleep medicine to address both functional impairment and molecular mechanisms involved in sleep-disordered breathing [13]. Precision medicine approaches incorporating craniofacial phenotyping, inflammatory biomarkers and upper airway functional assessment may improve patient selection and therapeutic outcomes [18]. Digital technologies, including wearable sleep-monitoring devices and tele-rehabilitation platforms, provide opportunities for continuous assessment of treatment adherence and functional recovery [19]. Standardized OMT protocols combined with pharmacological modulation of inflammatory and oxidative pathways may further enhance upper airway stability and reduce disease progression [12]. Future multicenter studies integrating molecular biomarkers, imaging and objective sleep outcomes are needed to establish

evidence-based multidisciplinary strategies for personalized management of sleep-disordered breathing [20].

Conclusion:

Orofacial myofunctional therapy and pharmaceutical sleep medicine target complementary biological pathways involved in sleep-disordered breathing. Their integration offers a mechanism-based strategy to improve upper airway function, regulate inflammatory responses and enhance neuromuscular stability. However, biomarker-driven and multidisciplinary studies are needed in future to validate personalized therapeutic approaches for optimal long-term clinical outcomes.

Advancement to knowledge:

This manuscript provides a novel mechanism-based synthesis integrating orofacial myofunctional therapy (OMT) with pharmaceutical sleep medicine. By highlighting the complementary interactions between mechanical neuromuscular adaptation and molecular anti-inflammatory/antioxidant targeted pathways, this work lays the foundation for personalized, multidisciplinary therapeutic strategies in sleep-disordered breathing.

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