

The quality of sleep modified by the mouth breathing syndrome can impair the athlete's physical performance

Bárbara Capitanio de Souza¹

¹Academia Brasileira de Odontologia do Esporte, Rio de Janeiro, RJ, Brazil

• **Conflicts of interest: none declared.**

ABSTRACT

Objective: to identify, through the literature, whether a mouth breathing syndrome can influence the athlete's physical performance. **Material and Methods:** the search strategy employed the term sleep with the boolean operator and to the descriptors: athletic performance, oral breathing and mouth breathing. Also used the term athlete with the boolean operator and to the descriptors: oral breathing and mouth breathing. For a more current reading, only articles published since 2000 were selected. It was carried out a reading of the selected and excluded articles that did not contemplate the proposed theme. **Results:** the athlete's physical performance is coordinated with different factors, which involve individual and environmental characteristics, indicating a timely relation in the periodization of training. The achievement of the best result involves the adaptive processes related to the activation of innumerable signaling pathways, which are subject the action of these factors, causing subsequent metabolic responses. Sleep disturbances associated with respiratory flow, as observed in mouth breathing syndrome, can be considered as factors influencing physical performance, indicating a need for new perceptions about an understanding of the variables that involve the performance.

Conclusion: a mouth breather syndrome may be a potential modifying factor of the sleep and of the humoral and cellular immune response, thus influencing biophysiological events that present a common inflammatory profile, such as those occurring in physical performance.

Keywords: Mouth breathing; Oral health; Physical education; Training; Sleep.

Introduction

Mouth breathing syndrome (MBS) is a pathology that is associated mainly with the obstruction of the upper airways, increasing the resistance of nasal breathing, which can be induced by several factors such as tonsil hyperplasia, hypertrophic turbinates, rhinitis, tumors, infectious or inflammatory diseases, and abnormalities in nasal architecture.^{1,2} However, even after the related factors are removed, the MBS may persist due to the patient's mouth-breathing habit.³ Patients develop unbalanced facial muscles, in addition to alterations in the positioning of teeth, lips, tongue, palate, and jaw, so as to counterbalance the new breathing pattern.^{4,5}

This respiratory alteration has become a relevant concern to health professionals, since it involves various comorbidities.⁶ The MBS is a commonly mentioned characteristic of respiratory modification during sleep, being a potential risk factor for disorders related to the condition, but its characteristics are little recognized.^{7,8} Moreover, it can cause similar symptoms to those of attention deficit hyperactivity disorder and alterations in brain function.^{9,10} Additionally, the MBS, due to involving changes in sleep pattern, can also contribute to decrease in immune defense cells and increase in humoral serum values related to inflammatory mediators, indicating the occurrence of oxidative stress and altered systemic inflammatory state.¹¹

Sleep is a restorative circadian process involved in different physiological events such as modification of temperature and blood pressure, immune function and hormonal chang-

es.¹² Significant effects on attention, memory, cognitive performance, the pace of work, speed and accuracy can also be observed.¹³ Considering the athlete patient, a reduction in the quality or quantity of sleep can result in biological and cognitive imbalance that is potentially capable of decreasing physical performance or the result of the recovery process after workout.¹⁴ Similarly, events or diseases that alter or impair sleep could have a significant negative impact on athletic performance.¹⁵

Athlete patients are subject to MBS and consequent alterations in sleep quality, which can lead to decreased physical performance. The symptoms these patients present with may not be related to the initial respiratory problems; however, they can be consequences of these alterations. Therefore, the objective of this study was to determine, through the literature, if the MBS can influence an athlete's physical performance.

Material and Methods

This is an analytical descriptive study, which was conducted through literature review. The search strategy employed the term *sleep* with the Boolean operator *and* and the keywords: athletic performance, oral breathing, and mouth breathing. We also used the term *athlete* with the Boolean operator *and* and the keywords: oral breathing and mouth breathing. For an up-to-date reading, we selected only articles published from 2000. After initial assessment of titles and abstracts, we read the selected articles and excluded those that did not address the proposed theme.

Literature Review

The prevalence of the MBS has a significant expression, considering young patients (20% to 45% approximately).¹⁶⁻¹⁸ The etiology of the condition is mainly associated with alterations or obstruction of the upper airways, such as septal deviation, choanal atresia, nostril stenosis, alterations of the cartilage of the nasal pyramid, hypertrophy of adenoids, rhinitis, rhinosinusitis.¹⁹ The main characteristics involve alterations in the oral cavity, such as narrowing of the jaw and stretch of the perioral muscle, halitosis, increase in the number of caries and periodontal diseases, constant pain or irritation in the throat, dry and persistent cough, morning headache, nocturnal enuresis, difficulty in feeding, dento-facial deformities, irritability, sleepiness, cognitive deficit, facial asymmetry, and postural problems.¹⁹⁻²¹

MBS is a predisposing factor for the development of sleep-related breathing disorders, and this relation has been described with concern, due to the clinical impact resulting from these conditions on health.^{18,22} Nasal breathing is the preference way for air flow in the body and its modification can lead to important morphological and functional alterations.²³ Respiratory interference occurs primarily by periodic reduction or cessation of airflow during sleep, which leads to hypoventilation, apnea, and fragmentation of the processes involved in this condition.²²

Functional alterations of the respiratory system caused by modified airflow during sleep, such as repeated hypoxemia, retention of carbon dioxide, and repeated awakening lead to complications and alterations of important biological functions, such as the immune system.¹¹ MBS can contribute to alter humoral or cellular immune response, because it has significant influence on the pathological and physiological processes of systemic ischemia-reperfusion. The events of hypoxia and reoxygenation are capable of altering the number of inflammatory cells, such as neutrophils and monocytes, and may induce changes in cellular immune response.^{24,25} Similarly, cytokines and inflammatory mediators also suffer modulation influenced by processes involved in sleep disorders.²⁶ For the athlete patient, knowledge of these conditions has singular importance, because the events that control the muscle functional demand and the level of fatigue are also directly related to immune response.^{27,28}

Considering the biological processes involved in physical performance, it is important to analyze the immune response, as it modulates events associated with muscular metabolism.²⁹ Muscular hypertrophy, which can be regarded as a performance parameter, is a physiological adaptation in response to stimuli. Humoral factors and oxidative stress can modify some phase of the signaling pathway for muscle growth and impair this process.³⁰ Some studies emphasize the importance of the circadian stability of sleep to reduce the frequency of injuries and improve the subsequent

recovery. They also point out that there may be decreased availability of muscle glycogen and impaired muscle damage repair, when there is influence of the modified immune response.³¹⁻³³

Proper maintenance of the relation between the consumption and the storage of glycogen in the muscle is important for the efficiency of the activity of the tissue such as the production of force and the extension of the length of continued effort.³⁴ Homeostasis of muscle energy metabolism can be influenced by immune response.³⁵ Inflammatory cytokines, such as interleukins IL-1 β , IL-6, and TNF- α , have an important role in metabolic alterations related to exercise.³⁶ Alterations in the concentration and functioning of these mediators can be associated with the rapid depletion of intramuscular glycogen and concomitant decreased performance in the activity of the tissue.³⁷ Therefore, altered physiological events, which can be related to the modification of any of these factors, such as the poor quality of sleep, can have a significant role in modifying an athlete's potential physical performance.²⁶

Analyzing the tissue injury, after tissue damage, inflammatory cells infiltrate the injured muscle and stimulate the secretion of pro-inflammatory cytokines that will modulate and coordinate healing.³⁸ In events involving the regeneration of muscle tissue, there is an important action of inflammatory mediators, which will act on muscle satellite cells. Alterations in concentration or types of inflammatory cells participating in the process may have negative contribution to the local production of cytokines, influencing the repair functions of satellite cells.³⁹ Factors interfering with the regular expression of inflammatory cytokines – which result from increase in the recruitment of these cells or prolonged stay in the region of the lesion – can lead to a deficient repair process, which will have direct influence on the subsequent performance of this tissue.⁴⁰ As sleep quality is an important agent for maintenance of the homeostasis of inflammatory mediators, disorders related to this physiological process can cause deficiency in the event of cellular repair of injured muscle.²⁶

Physiological conditions can also influence an athlete's performance or impair the outcome of the recovery process. The literature explores the relation between sleep and levels of inflammatory mediators.⁴¹ Increases in pro-inflammatory cytokines resulting from altered sleep are pointed out, which can promote immune system dysfunction and cause altered inflammatory state.⁴² Values of interleukins, such as IL-6, TNF- α , and IL-1 β , were high after events of deprivation or alteration of sleep, with recovery of serum levels when control of the circadian cycle occurs.⁴³

In addition to the effects on cognitive function and concentration, already pointed out, the consequences of sleep disorders can also impair the outcome of recovery after

training or physical activity.^{44,45} The modification in intensity and volume of training is carried out by athletes so as to increase the response of physical performance. However, when there is some imbalance between training stress and proper recovery, there may be an unfavorable response on an athlete's recovery and subsequent decline in performance.^{31,32} Situations of extended state of low performance combined with deficient recovery – which may have an additional effect caused by reduced quality and quantity of sleep, in addition to involving inadequate response to exercise – can result in physiological disorders such as non-functional overreaching (nf-OR). The evolution of this condition can affect an athlete's neurological, immune, and endocrine functioning, and is related to the development of overtraining syndrome (OT).⁴⁶

In general, physical performance is coordinated with different factors, involving individual and environmental characteristics, indicating a necessary and opportune relation in the periodization of training plan. Achieving the best result involves adaptive processes related to the activation of numerous signaling pathways, which are subject to the action of these factors, leading to subsequent metabolic responses. Sleep disorders associated with alterations of respiratory flow, as observed in the presence of MBS, can be considered as factors influencing physical performance, indicating the need for new perspectives in comprehending the variables involved in performance.

Sleep has important biological and physiological functions that interfere with the physical and cognitive recovery processes and may have potential action on an athlete's performance.⁴⁷ It is important for the sportsperson to work on improving performance, given the small margin of difference between success and failure in the competitive sports of today. Despite the comprehension regarding this matter, the circadian factors are given little consideration by professional sports teams or their followers.⁴⁸ Factors that modify this process, such as the MBS, may be particularly critical to the impairment of an athlete's physical performance, and special attention should be given to this condition.

Conclusion

The MBS is related to alterations of respiratory airflow and sleep disorders. This condition may lead to some characteristics, such as facial asymmetries, postural problems, dentofacial deformities, and cognitive deficit. Although the consequences of respiratory alterations and of events related to sleep disorders are still not fully known, there is evidence indicating an important relation with the regulation of athletic performance and recovery. Furthermore, the MBS is also pointed out as a potential modifying factor of humoral and cellular immune response and, thus, can influence biophysiological events that present a common inflammatory profile such as those that occur in physical performance.

References

1. Junqueira P, Marchesan IQ, de Oliveira LR, Ciccone E, Haddad L, Rizzo MC. Speech-language pathology findings in patients with mouth breathing: multidisciplinary diagnosis according to etiology. *Int J Orofacial Myology*. 2010;36:27-32.
2. Okuro RT, Morcillo AM, Sakano E, Schivinski CIS, Ribeiro MAG, Ribeiro JD. Exercise capacity, respiratory mechanics and posture in mouth breathers. *Braz J Otorhinolaryngol*. 2011;77(5):656-62.
3. Menezes VA, Cavalcanti LL, Albuquerque TC, Garcia AFG, Leal RB. Mouth breathing within a multidisciplinary approach: perception of orthodontists in the city of Recife, Brazil. *Dental Press J Orthod*. 2011;16(6):84-92.
4. Iwasaki T, Saitoh I, Takemoto Y, Inada E, Kanomi R, Hayasaki H. Evaluation of upper airway obstruction in Class II children with fluid-mechanical simulation. *Am J Orthod Dentofacial Orthop*. 2011;139(2):e135-45.
5. Alves Jr M, Baratieri C, Nojima LI, Nojima MCG, Ruellas ACO. Three-dimensional assessment of pharyngeal airway in nasal- and mouth-breathing children. *Int J Pediatr Otorhinolaryngol*. 2011;75(9):1195-9.
6. Abreu RR, Rocha RL, Lamounier JA, Guerra AF. Etiology, clinical manifestations and concurrent findings in mouth-breathing children. *J Pediatr*. 2008;84(6):529-35.
7. Pacheco MC, Casagrande CF, Teixeira LP, Finck NS, de Araújo MT. Guidelines proposal for clinical recognition of mouth breathing children. *Dental Press J Orthod*. 2015;20(4):39-44.
8. Young T, Finn L, Kim H. Nasal obstruction as a risk factor for sleep-disordered breathing. *J Allergy Clin Immunol*. 1997;99:757-62.
9. Akre H, Øverland B, Skatvedt O. Sleep-related breathing disorders. *Tidsskr Nor Laegeforen*. 2009;129(17):1762-5.
10. Sano M, Sano S, Oka N, Yoshino K, Kato T. Increased oxygen load in the prefrontal cortex from mouth breathing: a vector-based near-infrared spectroscopy study. *Neuroreport*. 2013;24(17):935-40.
11. Zhang Z, Wang C. Immune status of children with obstructive sleep apnea/hypopnea syndrome. *Pak J Med Sci*. 2017;33(1):195-9.
12. Zisapel N. Sleep and sleep disturbances: biological basis and clinical implications. *Cell Mol Life Sci*. 2007;64(10):1174-86.
13. Goel N, Basner M, Rao H, Dinges DF. Circadian rhythms, sleep deprivation, and human performance. *Prog Mol Biol Transl Sci*. 2013;119:155-90.
14. Milewski MD, Skaggs DL, Bishop GA, Pace JL, Ibrahim DA, Wren TA, et al. Chronic lack of sleep is associated with increased sports injuries in adolescent athletes. *J Pediatr Orthop*. 2014;34(2):129-33.
15. Luke A, Lazaro RM, Bergeron MF, Keyser L, Benjamin H, Brenner J, et al. Sports-related injuries in youth athletes: is overscheduling a risk factor? *Clin J Sport Med*. 2011;21(4):307-14.
16. Abreu RR, Rocha RL, Lamounier JA, Guerra AF. Prevalence of mouth breathing among children. *J Pediatr*. 2008;84(5):467-70.
17. Felcar JM, Bueno IR, Massan AC, Torezan RP, Cardoso JR. Prevalence of mouth breathing in children from an elementary school. *Cienc Saude Colet*. 2010;15(2):437-44.
18. Pacheco MC, Fiorott BS, Finck NS, Araújo MT. Craniofacial changes and symptoms of sleep-disordered breathing in healthy children. *Dental Press J Orthod*. 2015;20(3):80-7.
19. Abreu RR, Rocha RL, Lamounier JA, Guerra AF. Etiology, clinical manifestations and concurrent findings in mouth-breathing children. *J Pediatr*. 2008;84(6):529-35.
20. Corrêa EC, Bérzin F. Mouth Breathing Syndrome: cervical muscles recruitment during nasal inspiration before and after respiratory and postural exercises on Swiss Ball. *Int J Pediatr Otorhinolaryngol*. 2008;72(9):1335-43.
21. Faria PT, de Oliveira Ruellas AC, Matsumoto MA, Anselmo-Lima WT, Pereira FC. Dentofacial morphology of mouth breathing children. *Braz Dent J*. 2002;13(2):129-32.
22. Suzuki M, Furukawa T, Sugimoto A, Katada K, Kotani R, Yoshizawa T. Relationship between Oral Flow Patterns, Nasal Obstruction, and Respiratory Events during Sleep. *J Clin Sleep Med*. 2015;11(8):855-60.

23. Kim HY, Jeong JI, Dhong HJ, Sohn JH, Hong SD, Kim JH, et al. Nasal obstruction and palate-tongue position on sleep-disordered breathing. *Clin Exp Otorhinolaryngol*. 2013;6(4):226-30.
24. Wieckowski EU, Visus C, Szajnik M, Szczepanski MJ, Storkus WJ, Whiteside TL. Tumor-derived microvesicles promote regulatory T cell expansion and induce apoptosis in tumor-reactive activated CD8+T lymphocytes. *J Immunol*. 2009;183(6):3720-30.
25. Sukumar M, Liu J, Ji Y, Subramanian M, Crompton JG, Yu Z, et al. Inhibiting glycolytic metabolism enhances CD8+T cell memory and antitumor function. *J Clin Invest*. 2013;123(10):4479-88.
26. Li ZG, Li TP, Ye H, Feng Y, Li DQ. Immune function changes in patients with obstructive sleep apnea hypopnea syndrome. *J Southern Med Uni*. 2011;31(6):1003-5.
27. Li YP, Reid MB. NF-kappaB mediates the protein loss induced by TNF-alpha in differentiated skeletal muscle myotubes. *Am J Physiol Regul Integr Comp Physiol*. 2000;279(4):R1165-70.
28. Hirose L, Nosaka K, Newton M, Laveder A, Kano M, Peake J, et al. Changes in inflammatory mediators following eccentric exercise of the elbow flexors. *Exerc Immunol Rev*. 2004;10:75-90.
29. Beas-Jiménez JD, López-Lluchb G, Sánchez-Martínez I, Muro-Jiménez A, Rodríguez-Bies E, Navas P. Sarcopenia: implications of physical exercise in its pathophysiology, prevention and treatment. *Rev Andal Med Deporte*. 2011;4(4):158-66.
30. Bassel-Duby R, Olson EN. Signaling Pathways in Skeletal Muscle Remodeling. *Annu Rev Biochem*. 2006;75:19-37.
31. Fullagar HH, Skorski S, Duffield R, Hammes D, Coutts AJ, Meyer T. Sleep and athletic performance: the effects of sleep loss on exercise performance, and physiological and cognitive responses to exercise. *Sports Med*. 2015;45(2):161-86.
32. Nédélec M, Halson S, Abaidia A, Ahmaidi S, Dupont G. Stress, sleep and recovery in elite soccer: a critical review of the literature. *Sports Med*. 2015;45(10):1387-400.
33. Thun E, Bjorvatn B, Flo E, Harris A, Pallesen S. Sleep, circadian rhythms, and athletic performance. *Sleep Med. Rev*. 2015;23:1-9.
34. Angelini C, Tasca E, Nascimbeni AC, Fanin M. Muscle fatigue, nNOS and muscle fiber atrophy in limb girdle muscular dystrophy. *Acta Myol*. 2014;33(3):119-126.
35. Tsuchiya M, Kiyama T, Tsuchiya S, Takano H, Nemoto E, Sasaki K, et al. Interleukin-6 maintains glucose homeostasis to support strenuous masseter muscle activity in mice. *Tohoku J Exp Med*. 2012;227(2):109-17.
36. Pedersen BK. Special feature for the Olympics: effects of exercise on the immune system: exercise and cytokines. *Immunol Cell Biol*. 2000;78(5):532-5.
37. Chiba K, Tsuchiya M, Koide M, Hagiwara Y, Sasaki K, Hattori Y, et al. Involvement of IL-1 in the Maintenance of Masseter Muscle Activity and Glucose Homeostasis. *PLoS One*. 2015;10(11): e0143635.
38. Perdiguerro E, Kharraz Y, Serrano AL, Munoz-Canoves P. MKP-1 coordinates ordered macrophage-phenotype transitions essential for stem cell-dependent tissue repair. *Cell Cycle*. 2012;11(5):877-86.
39. Lagrota-Candido J, Canella I, Pinheiro DF, Santos-Silva LP, Ferreira RS, Guimarães-Joca FJ, et al. Characteristic pattern of skeletal muscle remodelling in different mouse strains. *Int J Experiment Pathol*. 2010;91(6):522-9.
40. Strle K, McCusker RH, Tran L, Rei A, Johnson RW, Freund GG, Dantzer R, Kelley KW. Novel activity of an anti-inflammatory cytokine: IL-10 prevents TNF α -induced resistance to IGF-I in myoblasts. *J Neuroimmunol*. 2007;188(1-2):48-55.
41. O'Connor MF, Bower JE, Cho HJ, Creswell JD, Dimitrov S, Hamby ME, et al. To assess, to control, to exclude: effects of biobehavioral factors on circulating inflammatory markers. *Brain Behav Immun*. 2009;23(7):887-897.
42. Burgos I, Richter L, Klein T, Fiebich B, Feige B, Lieb K, et al. Increased nocturnal interleukin-6 excretion in patients with primary insomnia: a pilot study. *Brain, Behavior, and Immunity*. 2006;20(3):246-53.
43. Haack M, Sanchez E, Mullington JM. Elevated inflammatory markers in response to prolonged sleep restriction are associated with increased pain experience in healthy volunteers. *Sleep*. 2007;30(9):1145-52.
44. Facer-Childs E, Brandstaetter R. The impact of circadian phenotype and time since awakening on diurnal performance in athletes. *Curr Biol*. 2015;25(4):518-22.
45. Smith RS, Efron B, Mah CD, Malhotra A. The impact of circadian misalignment on athletic performance in professional football players. *Sleep*. 2013;36(12):1999-2001.
46. Kreher JB. Diagnosis and prevention of overtraining syndrome: an opinion on education strategies. *Open Access J Sports Med*. 2016;7:115-22.
47. Zamparo P, Dall'ora A, Toneatto A, Cortesi M, Gatta G. The determinants of performance in master swimmers: a cross-sectional study on the age-related changes in propelling efficiency, hydrodynamic position and energy cost of front crawl. *Eur J Appl Physiol*. 2012;112:3949-57.
48. Scheer FA, Hu K, Evoniuk H, Kelly EE, Malhotra A, Hilton MF, et al. Impact of the human circadian system, exercise, and their interaction on cardiovascular function. *Proc Natl Acad Sci U S A*. 2010;107(47):20541-6.

Mini Curriculum

Bárbara Capitanio de Souza - DDS and MSc.

Submitted: 06/27/2017 / Accepted for publication: 07/31/2017

Corresponding Author

Bárbara Capitanio de Souza

E-mail: barbara.capitanio@gmail.com