Obstructive Sleep Apnea and Metabolic Syndrome: New Directions in this Association

Oliveira e Silva L1,*, Rizzatti Galhardo F1, Maior ML2, Tufik S1, Bittencourt L1 and Togeiro SM1

1Departamento de Psicobiologia-Universidade Federal de São Paulo, Brazil
2Universidade Federal do Rio de Janeiro, Brazil

Submission: December 18, 2017; Published: January 05, 2018

*Corresponding author: Luciana Oliveira e Silva, Departamento de Psicobiologia, Universidade Federal de São Paulo, Brazil, Tel: +55 1-121-490-150; Email: luciana.fisioterapia@gmail.com

Introduction

Obstructive Sleep Apnea (OSA) has become the most prevalent respiratory sleep disorder and imposes greater already mentioned public health burden [1,2]. Risk factors for metabolic syndrome (MetS) are frequently observed in individuals with OSA [3,4]. The association between OSA and MetS has been recognized as "Syndrome Z" [3]. It’s well known that moderate and severe OSA are related to cardiovascular and metabolic consequences mortality [5-9]. However, there is some evidence that OSA contributes to the development or exacerbated MetS / insulin resistance/type 2 diabetes mellitus [10].

The pathophysiology of OSA is complex and multifactorial and obesity is the main factor for this sleep respiratory disorder. Metabolic disorders/obesity share with OSA common intermediate pathogenic pathways involved in the interplay between these conditions [11]. Intermittent hypoxemia and hypercapnia, arousals from sleep, swings in intrathoracic pressure during obstructive respiratory events (apneas and hypopneas) trigger pathogenic pathways such as increased in the autonomic nervous system and inflammatory activity, alterations in adipokine levels and endothelial function that are associated with cardiovascular and metabolic consequences [12,13].

Additionally, dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis was also described in patients with severe OSA that could be a further factor for MetS [14,15]. As above mentioned it is well known that the main confounder for exacerbating cardiometabolic risk in OSA is obesity [16] and previous studies have adequately analyzed the influence of visceral obesity for this risk in these patients [11,17].

More recently, the genetic basis of the inflammatory response in OSA have been explored and contributing to explain the relation between OSAs, obesity and insulin resistance [18]. Insulin Resistance increases insulin-stimulated hepatic lipogenesis and causes a general accumulation of ectopic lipids [19]. The intracellular accumulation of lipids then triggers defects in insulin signaling and induces IR in muscle and liver [20-23] initiating a vicious cycle. According to recent studies, high values of TG/HDL-c ratio has been identified as a possible early atherogenic marker in different populations, especially in moderate-severe OSA [24,25]. Therefore, this ratio could be related to Insulin Resistance in patients with remarkable OSA [25,26].

In addition, polymorphisms of Apolipoproteins (APOE ε2, and APOE ε4) have been investigated in OSA patients [27,28]. However, despite researchers have demonstrated the several Apo lipoprotein polymorphisms are associated with diabetes susceptibility and/or lipid metabolism, further studies with high sample size are necessary for this field [28]. The aim of this review is to highlight the relationship between OSA and metabolic disturbance addressing the pathophysiology mechanisms, confounding factors, significant gaps in research and future directions [29]. In addition, The International Diabetes Federation published clinical practice recommendations suggesting that OSA patients should be routinely screened for markers of metabolic disturbance and cardiovascular risks such as waist circumference, blood pressure, and fasting lipid and glucose levels. It also recommends that the possibility of OSA should be considered in the assessment of all patients with type 2 diabetes mellitus and metabolic syndrome [10].

References

