



Targeting gut microbiome is the way forward in personalized medicine for obstructive sleep apnea



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Growing evidence are beginning to indicate that the gut microbiome may play a role in the development and progression of obstructive sleep apnea (OSA) [1]. OSA leads to intermittent hypoxia, causing a periodic decrease in oxygen levels in the blood and in the gastrointestinal system. This creates favorable conditions for bacteria that thrive in low-oxygen environment. Studies have shown that changes in the composition of the gut microbiome, such as a decrease in good bacteria and an increase in bad bacteria, can lead to inflammation and metabolic dysfunction that may contribute to OSA [2]. Several studies have found that individuals with OSA have a different composition of gut microbes compared to those without OSA [3]. In that way, an individual with OSA who have a specific gut microbiome profile with *Fusobacterium*, *Megamonas*, *Lachnospiraceae*, *Anaerostipes*, etc. found to be associated with an increased risk of OSA [4]. A study inducing intermittent hypoxia in mice for 6 weeks found a significant impact on the overall microbial community structure, with increased Firmicutes and decreased Bacteroidetes and Proteobacteria compared to control mice [5]. Bacteroidetes are considered beneficial because they produce short-chain fatty acids, while Firmicutes are associated with negative effects on glucose and fat metabolism. A microbiome-based therapy could be an adjuvant to treat and limit co-morbid consequences. Such patients might be more likely to benefit from treatments that target the gut microbiome, such as probiotics or prebiotics. The idea of using the microbiome for personalized medicine in the field of OSA is intriguing and deep research is needed to fully understand the relationship between the gut microbiome and OSA and to determine the best way to tailor personalized microbiome therapy to improve patient outcomes.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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