

Obstructive Sleep Apnea and Autoimmune Disease: A Two-Way Process

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We read with a keen interest the article published by Walsh et al.¹ These researchers analyzed 63 subjects with ankylosing spondylitis (SpA) and found that patients using TNF-inhibitors were less likely to have obstructive sleep apnea (OSA). Moreover, a non-statistically significant trend toward an improvement of OSA severity was found. The authors speculated regarding potential mechanisms responsible for this clinical observation such as more frequent corticosteroid use, cervical spine abnormalities and systemic inflammation. We are grateful to authors for studying this important problem. Nevertheless, we believe that some aspects merit attention.

Loubaki et al. showed that upper airways (UA) of patients with OSA have a greater expression of TNF- α , which may lead to UA inflammation and greater collapsibility.² Walsh et al.¹ discussed that both OSA and SpA share systemic inflammation as a pathophysiologic feature. On the other hand, a research group from Taiwan showed that OSA increases the risk for the incidence of autoimmune diseases in a prospective epidemiological studies.^{3,4} It is interesting to speculate that OSA related increase in TNF- α may initiate the disease in susceptible individuals. Therefore, OSA may be not only the consequence of autoimmune rheumatic disease, but also as a risk factor for their occurrence. Future studies should address whether OSA treatment would have an impact on reduction of rheumatic disease incidence

CITATION

Mirrakhimov AE; Mirrakhimov EM. Obstructive sleep apnea and autoimmune disease: a two-way process. *J Clin Sleep Med* 2013;9(4):409.

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SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication January, 2013

Accepted for publication January, 2013

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DISCLOSURE STATEMENT

The authors have indicated no financial conflicts of interest.