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PII: S0026-0495(17)30319-0
DOI: doi: 10.1016/j.metabol.2017.11.008
Reference: YMETA 53679

To appear in: *Metabolism*

Received date: 19 July 2017
Revised date: 8 November 2017
Accepted date: 10 November 2017

Please cite this article as: Trzepizur Wojciech, Cortese Rene, Gozal David, Murine Models of Sleep Apnea: Functional Implications of Altered Macrophage Polarity and Epigenetic Modifications in Adipose and Vascular Tissues, *Metabolism* (2017), doi: 10.1016/j.metabol.2017.11.008

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Abstract

Obstructive sleep apnea (OSA) is a highly prevalent disease across the lifespan, is characterized by chronic intermittent hypoxia and sleep fragmentation, and has been independently associated with substantial cardiometabolic morbidity. However, the reversibility of end-organ morbidity with treatment is not always apparent, suggesting that both tissue remodeling and epigenetic mechanisms may be operationally involved. Here, we review the cumulative evidence focused around murine models of OSA to illustrate the temporal dependencies of cardiometabolic dysfunction and its reversibility, and more particularly to discuss the critical contributions of tissue macrophages to adipose tissue insulin resistance and vascular atherogenesis. In addition,