

LUNG FUNCTION IN DIABETES

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Diabetes type II is underlain by insulin resistance interlocked with chronic hypoxia, inflammation, and oxidative stress. This self-perpetuating tetrad of disorders is escalated by oxygen-consuming cellular hypermetabolism induced by hyperglycemia. Health consequence of uncontrolled diabetes include an array of cardio- and cerebrovascular pathologies, akin to those present in obstructive sleep apnea disorder. In fact, there is a mutually interdependent connection between diabetes and sleep apnea. Thus, there are reasons to believe that ventilation might be impaired in diabetes. However, studies on ventilation in diabetes are scarce and the results are contentious, ranging from no change to impairment.

This lecture reviews our experimental investigations performed in conscious rats subjected to streptozotocin-induced untreated diabetes. Ventilation and its responses to the stimulatory hypoxic stimulus were investigated in a body box. Inflammatory and oxidative markers were investigated in blood plasma. Structural changes in the sensory organ of the carotid body, generating chemosensing responses to hypoxia, were assessed in electron microscopy. We found the unequivocal dampening of ventilation in diabetes accompanied by proinflammatory and prooxidative propensity at the tissue level. These changes, in particular dampened ventilation, were mostly reversed when a strong polyphenolic antioxidant agent was chronically administered on the background of diabetes. In conclusion, this research underscores the essential role of oxidative stress in the development of diabetic sequelae and points to the use of antioxidants as adjunctive pharmacological treatment that reduces the chronic hypoxic burden by invigorating ventilation, which helps control both diabetes and the resulting comorbid conditions.