

Abdominal Obesity And Pulmonary Function In Adults

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Abdominal obesity is a cardiovascular risk factor that is associated with insulin resistance, impaired glucose metabolism, hypertension, and dyslipidemia, all of which are features that are associated with the metabolic syndrome^{1,2}. Insulin resistance is recognized as a low-grade inflammatory condition³ and proinflammatory cytokines (i.e. adiponectin, leptin, tumor necrosis factor- alpha and interleukin-6) are associated with obesity^{4,5}. Systemic inflammation is also thought to play a role in the association between reduced pulmonary function and cardiovascular mortality as well as all-cause mortality^{6,7}. Insulin resistance and inflammation that arise from abdominal obesity may mediate the relation of pulmonary function and all-cause mortality.

A number of studies have explored the relation between abdominal obesity and pulmonary function. H M Ochs-Balcom et al⁸ investigated the relation of a number of adiposity markers with pulmonary function in a population-based study. They found inverse associations of abdominal height and waist circumference with pulmonary function in men and women with BMI values of $\geq 25\text{kg/m}^2$. Their results suggested that both overall and abdominal adiposity are negatively associated with FEV₁ and FVC, and that abdominal adiposity markers (i.e. abdominal height and waist circumference) have better explanatory power than total body adiposity measured as BMI or weight. Canoy et al⁹ analyzed the association of waist/hip ratio and pulmonary function in the European Prospective Investigation into Cancer and Nutrition study (EPIC-Norfolk), and reported an inverse association that remained significant after adjustment for BMI. Chen et al¹⁰ analyzed waist circumference and pulmonary function in a sample of men and women in the United Kingdom. These authors found inverse associations of waist circumference and pulmonary function. Harik-Khan et al¹¹ investigated the association of fat distribution and pulmonary function using waist/hip ratio. They reported an inverse association of FEV₁ and waist/hip ratio in men only. Lazarus et al¹² found no inverse associations of waist circumference or

waist/hip ratio with FVC in women. These authors also reported an inverse association of abdominal girth/hip breadth ratio with pulmonary function after adjustment for BMI in men over a narrow range in the Normative Aging Study¹³. Collins et al¹⁴ examined normal to mildly obese firefighters and found decreased pulmonary function in men with a waist/hip ratio of >0.95 .

A number of hypotheses have been proposed to explain the negative co-relation between pulmonary function parameters and measures of visceral obesity. One possible mechanism is a mechanical limitation of chest expansion during the FVC maneuver. Increased abdominal mass may impede the descent of the diaphragm and increase thoracic pressure¹⁵. Also, abdominal obesity is likely to reduce expiratory reserve volume via compressing the lungs and diaphragm^{16,17}. In addition, visceral adipose tissue influences circulating concentrations of interleukin-6, tumor necrosis factor- alpha, leptin and adiponectin, which are cytokines that may act via systemic inflammation to negatively affect pulmonary function^{4,5}. Investigators reported an inverse association of serum leptin concentrations with FEV₁ as well as higher levels of C-reactive protein, leucocytes, and fibrinogen, which are markers of systemic inflammation¹⁸. Therefore, inflammation may be the link between visceral obesity and pulmonary function.

To conclude, abdominal obesity is an important determinant of impaired pulmonary function, and it is of greater importance than overall adiposity markers such as weight and BMI. Therefore, investigators should consider the inclusion of markers of abdominal obesity as a potential confounding factor when investigating the determinants of pulmonary function.

References

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