

Excess visceral adipose tissue is associated with poorer lung function and increased airway inflammation in adults with asthma

S.R. Valkenborghs^{1,2}, L.G. Wood^{1,2}, R. Callister^{1,2}, J.W. Upham^{3,4}, C.L. Grainge^{2,5}, S. Anderson⁶, L.M. Williams^{1,2}, R.F. McLoughlin^{2,7}, E.J. Williams^{1,2} and H.A. Scott^{1,2}

¹School of Biomedical Sciences and Pharmacy, The University of Newcastle, Newcastle, NSW, Australia,

²Hunter Medical Research Institute, Newcastle, NSW, Australia,

³Diamantina Institute, The University of Queensland, Brisbane, QLD, Australia,

⁴Princess Alexandra Hospital, Brisbane, QLD, Australia,

⁵Department of Respiratory and Sleep Medicine, John Hunter Hospital, Newcastle, NSW, Australia,

⁶School of Environmental and Life Sciences, University of Newcastle, Newcastle, NSW, Australia and

⁷School of Nursing and Midwifery, The University of Newcastle, Newcastle, NSW, Australia

Excess visceral adipose tissue (VAT) is associated with poor metabolic health and systemic inflammation. Poor metabolic health and systemic inflammation have both been implicated in the development and severity of asthma. Therefore, this study aimed to determine whether excess VAT is associated with poorer asthma outcomes and inflammation in adults with asthma. Participants were adults with stable physician-diagnosed asthma aged 18–55 years ($n = 45$), recruited from Hunter Medical Research Institute, NSW, Australia. Body composition was assessed by dual-energy x-ray absorptiometry, with excess VAT mass classified according to Meredith-Jones et al.⁽¹⁾ Obesity was defined by body mass index (BMI). Four-day food diaries were entered into FoodWorks and nutrient analysis performed. Lung function (spirometry) and airway inflammation (induced sputum eosinophil count) were measured. Systemic inflammation was assessed by measuring plasma interleukin (IL)-6 concentration via high-sensitivity ELISA. Compared to participants with a normal VAT mass, those with excess VAT had poorer lung function, indicated by a lower % predicted forced expiratory volume in 1 second (%FEV₁; 79.1 ± 17.0 v. $88.9 \pm 12.1\%$, $p = 0.029$) and % predicted forced vital capacity (%FVC; 88.6 ± 12.0 v. $97.1 \pm 9.9\%$, $p = 0.012$). VAT mass was positively associated with airway inflammation (sputum eosinophils; $r_s = 0.334$, $p = 0.044$) and systemic inflammation (IL-6; $r_s = 0.434$, $p = 0.003$). Obesity, measured by BMI, was not associated with lung function or airway inflammation. However, obese participants had elevated systemic inflammation (IL-6) compared with non-obese participants [1.7 (1.5, 2.2) v. 1.0 (0.8, 1.3) pg/mL, $p < 0.001$]. Dietary intake was not associated with airway inflammation or lung function. In this study, excess visceral adiposity, but not excess body mass or dietary intake, were associated with poorer asthma outcomes in adults with asthma, indicated by poorer lung function and increased airway inflammation.

Reference

1. Meredith-Jones K, Taylor R, Brown R, et al. (2021) *Int J Obes* 45, 808–817.