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Obesity, metabolic syndrome and sleep apnoea: all pro-inflammatory states.

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Obesity is associated with significant morbidity and mortality and is increasing in prevalence worldwide. Associated conditions include insulin resistance (IR), diabetes, hypertension and dyslipidaemia; a clustering of these has recently been termed as metabolic syndrome. Weight gain is a major predictor of the metabolic syndrome with waist circumference being a more sensitive indicator than body mass index as it reflects both abdominal subcutaneous adipose tissue and visceral adipose tissue (VAT). VAT has more metabolic activity and secretes a number of hormones and pro-inflammatory cytokines which are linked with the metabolic abnormalities listed above. Central obesity also increases the risk of obstructive sleep apnoea syndrome (OSAS), where the sleep disordered breathing may also independently lead to/or exacerbate IR, diabetes and cardiovascular risk. The contribution of OSAS to the metabolic syndrome has been under-recognized. The putative mechanisms by which OSAS causes or exacerbates these other abnormalities are discussed. We propose that activation of nuclear factor kappa B by stress hypoxia and/or by increased adipokines and free fatty acids released by excess adipose tissue is the final common inflammatory pathway linking obesity, OSAS and the metabolic syndrome both individually and, in many cases, synergistically.

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