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Obstructive sleep apnoea syndrome, plasma adiponectin levels, and insulin resistance.

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OBJECTIVE: To investigate whether sleep-disordered breathing and/or plasma adiponectin levels are associated with insulin resistance independent of obesity or fat distribution in obstructive sleep apnoea syndrome (OSAS). DESIGN: Cross-sectional clinical study. PATIENTS: Two-hundred and thirteen Japanese patients with OSAS aged 27-80 years were divided into three groups: 30 with mild OSAS [apnoea-hypopnoea index (AHI) = 10.3 +/- 0.9 episodes/h, minimum oxygen saturation (min SpO2) = 87.3 +/- 0.9%], 98 with moderate OSAS (AHI = 28.9 +/- 0.6 episodes/h, min SpO2 = 82.1 +/- 0.7%), and 85 with severe OSAS (AHI = 68.1 +/- 2.8 episodes/h, min SpO2 = 72.3 +/- 1.6%). Twenty-one patients undergoing diabetic treatments (two mild, nine moderate and 10 severe) were excluded from the assessment of insulin resistance and plasma adiponectin measurements. MEASUREMENTS: Fat distribution [evaluated according to visceral (V) and subcutaneous (S) fat areas using computed tomography scanning at the umbilical level], blood pressure, metabolic parameters and hormones including insulin and adiponectin were measured. After full polysomnography, venous blood was collected between 0600 and 0700 h. RESULTS: Severe OSAS patients were more hypertensive than mild and moderate OSAS. Fasting plasma glucose (FPG) and fasting plasma insulin and homeostasis model assessment of insulin resistance (HOMA-IR) levels were all higher in severe OSAS than mild and moderate OSAS patients. HOMA-IR was correlated not only with obesity [body mass index (BMI), V and S areas] but also with apnoea (AHI, min SpO2 and desaturation time). Additionally, HOMA-IR was correlated positively with haemoglobin (Hb)A1c, systolic (SBP) and diastolic blood pressure (DBP), triglycerides and free fatty acids (FFA), and negatively with high density lipoprotein (HDL)-cholesterol, suggesting that insulin resistance is a key component of the metabolic syndrome in OSAS. Plasma adiponectin levels were not different between mild, moderate and severe OSAS groups. Plasma adiponectin levels were correlated with HOMA-IR and V area, but not AHI or min SpO2. Stepwise multiple regression analysis, however, revealed that BMI, AHI and plasma adiponectin were independently associated with HOMA-IR. CONCLUSION: Sleep-disordered breathing was associated with insulin resistance independent of obesity. Although plasma adiponectin was also an independent determinant of HOMA-IR in OSAS patients, plasma adiponectin was more closely related to obesity than to sleep apnoea. Although treatment of sleep-disordered breathing with nasal continuous positive airway pressure reportedly improves insulin sensitivity, our findings suggest that treatment of obesity is also essential in ameliorating insulin resistance at least through increased plasma adiponectin levels in OSAS.

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