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Adipokines in children with sleep disordered breathing.

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STUDY OBJECTIVE: Associations between SDB, the metabolic syndrome, and circulating levels of adipokines have emerged in adults but have not been examined in snoring children, who, in contradistinction to adults, display insulin resistance and lipid abnormalities as a function of adiposity rather than SDB. Therefore, we aimed to examine associations between circulating adipokines levels, insulin resistance, and measures of SDB in children. DESIGN: Prospective study. SETTING: Polysomnographic evaluation and assessment of plasma levels of leptin, adiponectin, resistin, glucose, insulin, and CRP. PARTICIPANTS: 130 children (mean age 8.2 +/- 2.8 years; 39% obese) were studied. MEASUREMENTS AND RESULTS: Log adiponectin levels were lower in obese than nonobese children (3.8 +/- 0.31 vs 4.0 +/- 0.30 corresponding to 8,381.4 +/- 5,841.0 vs 12,853.2 +/- 7,780.2 ng/ml, $P < 0.0001$) and were inversely correlated with BMI Z scores ($r = -0.47$, $P < 0.0001$) but not with log AHI. Log leptin concentrations were higher in the obese group than the nonobese group (4.2 +/- 0.32 vs 3.4 +/- 0.57 corresponding to 19,542.6 +/- 13,643.6 vs 5,875.5 +/- 8,425.7 pg/ml, $P < 0.0001$), correlated with BMI Z scores ($r = 0.64$, $P < 0.0001$), and were significantly lower in children with AHI < or = 1/hr than children with AHI > 1/hr ($P = 0.006$) and in children with SpO2 nadir > or = 90% than children with SpO2 nadir < 90%, even after controlling for BMI Z score ($P < 0.03$). No significant differences were found in log resistin levels as a function of obesity or AHI. Significant correlations between log adiponectin levels and log Insulin/Glucose (I/G) ratios (-0.28, $P = 0.006$) and between log leptin levels and log I/G ratios ($r = 0.66$, $P < 0.0001$) emerged. CONCLUSIONS: In close agreement with the absence of a measurable effect of SDB on insulin resistance in children, circulating adipokines levels are primarily attributable to the ponderal index. However, SDB and associated hypoxemia may contribute to the elevation of leptin levels.

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