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ORIGINAL ARTICLE

Glucose, insulin, and insulin resistance in normal-weight, overweight and obese children with obstructive sleep apnea

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KEYWORDS
Sleep apnea;
Obesity;
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Summary
Background: Obstructive sleep apnea (OSA) is associated with components of metabolic syndrome. Both body weight and OSA independently influence metabolic measurements. The goal of this study was to determine whether OSA in normal-weight, overweight or obese children, compared to matched control groups, was associated with increased levels of glucose, insulin and insulin resistance (IR).
Methods: Age- and gender-specific body mass index (BMI) percentiles were determined and used to categorize subjects into normal-weight (BMI < 85%) and overweight-obese (BMI ≥ 85%) groups. In addition, subjects were divided into normal-weight (BMI < 85%), overweight (BMI ≥ 85% and <95%) and obese (BMI ≥ 95%) groups. Polysomnography was conducted and morning levels of glucose and insulin were measured and IR was determined from the blood samples collected early in the morning after overnight fast. Results were compared between the subject groups. Effects of severity of OSA defined by apnea hypopnea index (AHI) and oxygen desaturation index (ODI) on glucose, insulin, and HOMA-IR were analyzed.
Results: Glucose, insulin, and HOMA-IR in OSA and matched control groups were not significantly different for normal-weight, overweight and obese subjects. The ODI was significantly associated with elevated levels of glucose and HOMA-IR after adjustment for age, gender, race, and BMI *Z*-score.
Conclusions: IR levels between OSA and control for both normal-weight, overweight and obese subjects were not significantly different. The ODI was associated with

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increased IR in children with OSA. OSA-induced hypoxic events during sleep may be a potential mechanism of increased IR in children with OSA, independent of body weight.

Introduction

Obstructive sleep apnea (OSA) in children has been linked to metabolic syndrome [1]. Insulin resistance (IR) is an important component of metabolic syndrome that is elevated in patients with OSA, independent of obesity [2]. However, studies using the homeostasis model assessment model (HOMA) for IR measurements in children with OSA have produced conflicting results, with both increased [3,4] and similar [5,6] HOMA-determined IR (HOMA-IR) in OSA and control subjects. Although a causal association between OSA and metabolic syndrome is yet to be established, obesity is the common co-morbidity associated with both OSA and metabolic syndrome [7]. Also, obesity and OSA are independently associated with all of the parameters of metabolic syndrome, including increased blood pressure [8], hyperglycemia [9], hyperinsulinemia [3], increased IR [10], diabetes mellitus [11], and increased triglyceride levels [12,13], as well as prothrombotic [14] and proinflammatory [15] conditions. Recent data suggest significant improvements of metabolic dysfunction after nasal continuous positive airway pressure treatment of OSA [16]. Although obesity is a major risk factor for OSA in both adults [17,18] and children [19,20], limited data are available comparing the effects of OSA on metabolic measurements in either normal-weight or obese children. For this study, we assessed levels of plasma glucose and insulin, as well as HOMA-IR, in normal-weight and overweight children with or without OSA to determine whether children with OSA have weight-dependent increases in these metabolic syndrome components compared to respective control groups matched for age, gender, and race. In addition, we determined the effects of severity of OSA, as evidenced by levels of apnea hypopnea index (AHI) and oxygen desaturation index (ODI) during sleep, on both glucose and insulin levels and HOMA-IR.

Methods

Subjects

Children between 5 and 14 years of age were recruited from the Otolaryngology and Pediatric Clinic of Cincinnati Children's Hospital Medical Center (CCHMC) for an overnight sleep study using polysomnography (PSG) for diagnosis of OSA. Height and body weight were measured to the nearest 0.1 cm and 0.1 kg, respectively. Age- and gender-specific *Z*-scores (measurement of a score's relationship to the mean in a group of scores) for body mass index (BMI) were calculated using reference data available in the Centers for Disease Control and Prevention 2000 growth charts for the United States [21]. Children with OSA were divided by BMI percentile into overweight (BMI $\geq$ 85%) and normal-weight (BMI < 85%) groups. In addition, subjects were divided into normal-weight (BMI < 85%), overweight (BMI $\geq$ 85% and <95%) and obese (BMI $\geq$ 95%) groups. Healthy children matched for age, gender, race, and BMI percentile also were recruited for PSG as control subjects for the OSA groups. The OSA subjects were free of cardiovascular, cerebrovascular, and other chronic medical disorders or genetic conditions, had never been treated for OSA, and were on no medications. The control subjects were free of any acute or chronic disease and were on no medications, and those with snoring, occult OSA (AHI $\geq$ 1 event/h), and alveolar hypoventilation on PSG were excluded from the study. Parental-signed informed consent for all children and assent for children over 7 years of age were obtained from each study participant prior to enrollment into the study. The study was approved by the CCHMC Institutional Human Subjects Review Board.

Study design

A medical history was obtained and a physical examination was performed on all subjects before the sleep study. Parents remained with their children throughout the night. Children were neither deprived of sleep prior to the study nor given sedatives. Demographic data was determined and heart rate and blood pressure were measured for each subject prior to the sleep study. Venous blood was collected in the early morning, between 6:00 am and 7:00 am, after overnight fasting for measurements of glucose and insulin. The presence