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ORIGINAL ARTICLE

Relationship between Serum Levels of Body Iron Parameters and Insulin Resistance and Metabolic Syndrome in Korean Children

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Abstract
Objectives: An increase in serum ferritin and levels of the cleaved soluble form of transferrin receptor (sTfR) are related to several metabolic conditions. We evaluated the relationship between body iron status indicators, including ferritin and sTfR, and insulin resistance and metabolic syndrome (MetS) in Korean children.
Methods: A cross-sectional study was conducted on 1350 children in Korea. Anthropometrical parameters; lipid profiles; levels of glucose, insulin, and leptin; and iron status indicators, including sTfR, serum ferritin, serum iron, total iron-binding capacity (TIBC), and transferrin saturation (TS), were analyzed.
Results: Although serum sTfR levels were significantly higher in boys than in girls (2.20 vs. 2.06 mg/L, $p < 0.0001$), serum iron and TS were higher in girls than in boys (101.38 vs. 95.77 mg/L, $p = 0.027$ and 30.15 vs. 28.91%, $p = 0.04$, respectively). Waist circumference (WC) and leptin were most significantly associated with body iron indicators when adjusted for age and sex. After adjusting for age, sex, and WC, sTfR levels showed the strongest positive association with leptin levels ($p = 0.0001$). Children in the highest tertile for homeostasis model assessment-insulin resistance (HOMA-IR) had higher TIBC ($p = 0.0005$) and lower serum iron ($p = 0.0341$), and the lowest TS ($p < 0.0001$) after adjustment for confounders. Children with higher sTfR were most significantly associated with risk of MetS compared with those lower sTfR ($p = 0.0077$).
Conclusion: The associations of serum levels of iron metabolism markers with leptin levels, HOMA-IR, and MetS suggest that iron-related factors may involve insulin resistance and MetS.

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1. Introduction

Iron plays a key role in many biological processes such as erythrocyte production, DNA synthesis, and cellular respiration [1,2]. The liver acts as the central organ in the regulation of body iron stores, and it carries the main burden in situations of iron overload [3]. Clinical measurement of iron storage is assessed by serum ferritin levels, and low serum ferritin indicates depleted iron stores. The homeostatic iron system maintains transferrin saturation (TS) at physiological levels, and it responds to signals from pathways that consume iron and sends signals to cells that supply iron to the bloodstream [4]. Most iron is loaded to serum transferrin, which binds to transferrin receptors (TfR) on target cells. Soluble TfR (sTfR) is released from microsomal membranes and directly regulates the binding of its ligand ferritransferrin in response to iron availability [5]. Released sTfR concentrations reflect the cellular expression level of membrane TfR and cellular iron demands.

The development of metabolic syndrome (MetS) clustering obesity, hypertension, and insulin resistance increases body iron stores. Previous studies have reported higher ferritin levels in individuals with MetS [6–8]. A recent study used serum sTfR levels as a body iron indicator of iron sufficiency or iron depletion in obese European adolescents [9,10]. Also, insulin inducing iron transport and accumulation in hepatocytes stimulates iron uptake in fat cells and redistributes intracellular TfRs to the cell surface [11]. Moreover, TfRs and insulin-like growth factor 2 (IGF2) 2 receptors in vesicles from cultured adipocytes colocalize with intracellular glucose transporters [12]. Serum sTfRs concentrations had been reported to be influenced by insulin secretion and insulin sensitivity in individuals with normal glucose tolerance [13]. The relationship between body iron status indicators and MetS in the general pediatric population does, however, remain unclear.

We therefore conducted a population-based cross-sectional study including Korean elementary students to determine the relationships between indicator of iron status and pediatric overweight measurements, homeostasis model assessment-insulin resistance (HOMA-IR), and MetS. The objective was to investigate whether serum sTfR levels indicate body iron status and which indicators of iron status are related to childhood insulin resistance and MetS.

2. Materials and methods

2.1. Study population

This study was conducted as part of the Korean Pediatric Cohort Study, which was designed to follow a cohort of Korean students from their entry into elementary school at age 7 years, to their graduation at age 13 years in Kyunggi Province and Seoul, Korea. The overall objective of the cohort study was to identify early risk factors for obesity and associated metabolic diseases in urban Korean children. The sole inclusion criterion was being enrolled in first grade in 2005 and 2008, and no exclusion criteria were applied. The children were to receive physical examinations annually and to provide blood samples for the measurement of levels of leptin, insulin, and indicators of body iron status. Children ($n = 1350$; 682 boys and 668 girls) aged 7 years and 10 years in 2008 were included in the present study. This study was approved by the Institutional Review Board of Inje University Seoul-Paik Hospital, Seoul, Korea and the Korean Center for Disease Control and Prevention. Written informed consent was obtained from the children's parents.

2.2. Anthropometric measurements

Height was measured using an automatic stadiometer (DS-102; Jenix, Seoul, Korea). Weight and percent body fat were measured via bioimpedance using a body composition analyzer (BC418; Tanita, Tokyo, Japan). Body mass index (BMI) was calculated by dividing the weight in kilograms by the square of the height in meters. Waist circumference (WC) was measured at the midpoint between the lower border of the ribcage and the iliac crest using a nonelastic tape measure. Blood pressure was measured twice on the right arm using a mercury sphygmomanometer while the individual was resting in a seated position.

2.3. Biochemical analyses

After a 12 hour overnight fast, blood samples were collected from the antecubital vein into Vacutainer tubes (BD, Franklin Lakes, NJ USA). Triglyceride (TG) and high-density lipoprotein cholesterol (HDLC) levels were measured via enzymatic assays and an autoanalyzer (model 7180; Hitachi, Tokyo, Japan). Fasting serum glucose levels were measured using the hexokinase method and a glucose analyzer (model 7180; Hitachi). Fasting serum insulin levels were measured using a radioimmunoassay kit (Diagnostic Products Corporation, Los Angeles, CA, USA). Fasting serum leptin levels were determined using a kit from Linco Research (St. Charles, MO, USA). The HOMA-IR score, an estimate of insulin resistance, was calculated as [fasting serum insulin level (μIU/mL) × fasting serum glucose level (mmol/L)]/22.5 [14]. Serum iron levels and total iron-binding capacity (TIBC) were measured using the FerroZine method and a Cobas Integra 800 analyzer (Roche, Mannheim, Germany). Ferritin levels were determined via chemiluminescence immunoassays (CLIA) and an autoanalyzer (model ADVIA CENTAUR; Bayer, USA). sTfR was measured using an enzyme immunoassay kit (Molecular Device, Sunnyvale, CA, California, USA). We defined MetS status in

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