ORIGINAL ARTICLE

Adiposity distribution influences circulating adiponectin levels

MITCHELL GUENTHER, ROLAND JAMES, JACQUELINE MARKS, SHI ZHAO, ANIKO SZABO, and SRIVIDYA KIDAMBI

MILWAUKEE, WISCONSIN

Thirty percent of obese individuals are metabolically healthy and were noted to have increased peripheral obesity. Adipose tissue is the primary source of adiponectin, an adipokine with insulin-sensitizing and anti-inflammatory properties. Lower adiponectin levels are observed in individuals with obesity and those at risk for cardiovascular disease. Conversely, higher levels are noted in some obese individuals who are metabolically healthy. Our objective was to determine whether abdominal adiposity distribution, rather than body mass index (BMI) status, influences plasma adiponectin level. A total of 424 subjects (female, 255) of Northern European ancestry were recruited from "Take Off Pounds Sensibly" weight loss club members. Demographics, anthropometrics, and dual-emission x-ray absorptiometry of the whole body, and computed tomography scan of the abdomen were performed to obtain total body fat content and to quantify subcutaneous adipose tissue (SAT) and visceral adipose tissue (VAT), respectively. Laboratory measurements included fasting plasma glucose, insulin, lipid panel, and adiponectin. Age- and gender-adjusted correlation analyses showed that adiponectin levels were negatively correlated with BMI, waist circumference, triglycerides, total fat mass, and VAT. A positive correlation was noted with high-density lipoprotein cholesterol and fat-free mass ($P < 0.05$). SAT-to-VAT ratios were also significantly associated with adiponectin ($r = 0.13$, $P = 0.001$). Further, the best positive predictors for plasma adiponectin were found to be SAT-to-VAT ratios and gender by regression analyses ($P < 0.01$). Abdominal adiposity distribution is an important predictor of plasma adiponectin and obese individuals with higher SAT-to-VAT ratios may have higher adiponectin levels. (Translational Research 2014;164:270–277)

Abbreviations: CT = computed tomography; HDL-C = high-density lipoprotein cholesterol; HOMA = homeostasis model assessment; IR = insulin resistance; IL = interleukin; LDL-C = low-density lipoprotein cholesterol; SAT = subcutaneous adipose tissue; SD = standard deviation; TNF-α = tumor necrosis factor α; VAT = visceral adipose tissue

From the Department of Medicine, Medical College of Wisconsin, Milwaukee, Wisconsin; Institute of Health and Society, Medical College of Wisconsin, Milwaukee, Wisconsin.

Submitted for publication February 26, 2014; revision submitted April 7, 2014; accepted for publication April 10, 2014.

Reprint requests: Srividya Kidambi, Division of Endocrinology, Metabolism, and Clinical Nutrition, Department of Medicine, Medical College of Wisconsin, 9200 West Wisconsin Avenue, Milwaukee, WI 53226; e-mail: skidambi@mcw.edu.

1931-5244/$ - see front matter

© 2014 Elsevier Inc. All rights reserved.

http://dx.doi.org/10.1016/j.trsl.2014.04.008

AT A GLANCE COMMENTARY

Guenther M, et al.

Background

Thirty percent of obese individuals are metabolically healthy. Studies have suggested that these obese individuals have predominantly peripheral obesity. Adiponectin, an adipokine, has insulin-sensitizing and anti-inflammatory properties. Adiponectin levels are lower in obese individuals with central adiposity. However, obese individuals who are metabolically healthy have been shown to have paradoxically higher adiponectin levels.

Translational Significance

In this study, we show that higher subcutaneous adipose tissue volume (as seen in individuals with peripheral obesity) may be associated with higher plasma adiponectin levels. This suggests higher subcutaneous adipose tissue volume may be protective by translating into higher circulating adiponectin levels.

INTRODUCTION

Obesity is associated with insulin resistance (IR), metabolic syndrome, and type 2 diabetes mellitus, and thus many obese individuals are at increased cardiovascular disease risk.[1,2] However, all obese individuals are not at increased risk for aforementioned metabolic abnormalities.[3] Individuals with centripetal distribution of adiposity (visceral adiposity) are at a higher cardiovascular disease risk compared with individuals with peripheral adiposity distribution.[3]

Adipose tissue, a dynamic endocrine organ, is a source of a number of adipocytokines and is responsible for a myriad of actions that may explain the metabolic risks attributed to adiposity.[4] Adiponectin is one such adipokine derived exclusively from white adipose tissue and has been shown to have insulin-sensitizing, anti-inflammatory, and antiapoptotic effects on a number of different cell types.[5,6] It is largely considered to have protective actions against obesity-related metabolic risks, and lower adiponectin levels are considered a risk factor for type 2 diabetes mellitus and cardiovascular disease.[7-9] Although adipose tissue is the sole source of adiponectin, adiponectin levels are lower in individuals with higher body mass index (BMI), particularly visceral adiposity, suggesting a nonlinear relationship with adipose tissue mass.[10,11]

Several studies have shown that females have higher levels of adiponectin than males,[6,8,12] which *may* be the result of differences in body fat distribution between genders.[8,12] In addition, newly described metabolically healthy obese phenotype individuals were recently shown to have paradoxical hyperadiponectinemia[12,13] with favorable metabolic risk profiles, suggesting that adiposity distribution may contribute to adiponectin levels and hence the cardiovascular risk of obesity. In the present study, we explore the relationship of plasma adiponectin level with total adiposity and abdominal adiposity distribution (subcutaneous vs visceral).

METHODS

Subjects. A total of 424 Caucasian subjects (male, 169; female, 255) were recruited from "Take Off Pounds Sensibly" weight loss club membership as has been previously described.[14,15] These subjects were part of a family-based study, and recruitment criteria consisted of having at least 2 obese siblings (BMI $\geq$ 30 kg/m^2) and at least 1 nonobese sibling or parent (BMI $\leq$ 27 kg/m^2) or both.[14,15] Subjects with a history of type 1 diabetes mellitus, cancer, renal or hepatic disease, active coronary artery disease, substance abuse, corticosteroids, thyroid medications above the replacement dose, or history of weight loss of more than 10% of body weight in the preceding 12 months were excluded from the study. All procedures were approved by the Medical College of Wisconsin's Institutional Review Board and conform to the relevant ethical guidelines for human research.

Measurements. Weight, height, and blood pressure were measured using standardized methods. Waist circumference was measured at the level of the navel, and hip circumference was measured at the widest point of the buttock region. BMI and waist-to-hip ratio (WHR) were calculated. Subjects were fasting at the time of laboratory measurements. Plasma glucose was measured in triplicate using a Glucose Analyzer II (Beckman Instruments, Brea, CA) with a glucose oxidase method. Plasma insulin was measured using a double antibody equilibrium radioimmunoassay (Linco Research, St. Louis) specific to human insulin. The homeostasis model assessment (HOMA) method was used for calculation of IR (HOMA-IR)[16] in patients without type 2 diabetes mellitus (n = 387). Plasma triglycerides were measured using a glycerylphosphate oxidase method (Stanbio Laboratory, Inc, San Antonio, TX). High-density lipoprotein cholesterol (HDL-C) was measured using phosphotungstic acid or $MgC1_2$ precipitate (Roche-Boehringer, Indianapolis, IN). Low-density lipoprotein cholesterol (LDL-C) was directly measured with an enzymatic selective protection method (Sigma Diagnostics, St. Louis, MO). Plasma adiponectin and leptin were determined using a double antibody equilibrium radioimmunoassay (Millipore

Download English Version:

https://daneshyari.com/en/article/3840497

Download Persian Version:

https://daneshyari.com/article/3840497

Daneshyari.com