Letter to the Editor

Letter by Oda et al Regarding Article, "Hypoadiponectinemia is Associated With Impaired Glucose Tolerance and Coronary Artery Disease in Non-Diabetic Men"

To the Editor:

Otsuka et al reported that hypoadiponectinemia is associated with impaired glucose tolerance and coronary artery disease in non-diabetic men.¹ Adipose tissue secretes many hormone-like substances, such as leptin, adiponectin, resistin, visfatin, monocyte chemoattractant protein-1, retinol-binding protein-4, angiotensinogen, atrial natriuretic protein, tumor necrosis factor- α , interleukin-6, plasminogen activator inhibitor-1, adipocyte-type fatty acid binding protein, etc. Obesity has been considered as an endocrine and inflammatory disorder intimately related to insulin resistance rather than merely anthropometric fatness, and C-reactive protein (CRP) is established as an independent risk factor for both diabetes and cardiovascular disease. Kim et al reported a model of extreme obesity associated with an improved metabolic profile in which adiponectin acts as a peripheral starvation signal promoting the storage of triglycerides preferentially in adipose tissue and reduces the macrophage infiltration into adipose tissue preventing systemic inflammation and insulin resistance.² Bains et al reported a transgenic model of severe visceral obesity without insulin resistance in which the adipocyte size is not increased and the plasma level of adiponectin is increased.³ Cinci et al described necrotic-like death of enlarged adipocytes with a crown-like structure that consisted of activated macrophages not only in adipose tissue of obese mice and in visceral and subcutaneous adipose tissue of obese humans, but also in the adipose tissue of hormone-sensitive lipase-deficient mice, which is a model of adipocyte hypertrophy without obesity but with insulin resistance.⁴ Kanda et al reported a transgenic model of mice with normal body and adipose tissue weight, normal adipocyte size, and normal plasma adiponectin level, which manifested macrophage infiltration into adipose tissue, insulin resistance, and glucose intolerance.⁵ These transgenic models indicate that adipocyte hypertrophy and infiltration of macrophages into adipose tissue, rather than increased adipose tissue mass or visceral obesity per se, are crucial for the metabolic consequences of obesity. Further, the clinical utility of waist circumference was recently criticized jointly by the Obesity Society, the American Society for Nutrition, and American Diabetes Association.⁶ Therefore, CRP may be superior to anthropometric parameters as a marker of the metabolic consequences due to obesity and as a component of the metabolic syndrome. Komatsu et al reported that adiponectin showed significant independent correlation with CRP, but not with high-density lipoprotein (HDL)-cholesterol, fasting glucose, fasting insulin, homeostasis model assessment of insulin resistance (HOMA-IR), waist circumference or body mass index (BMI), in a multivariate analysis among Japanese male health examinees, and that adiponectin was

lower and CRP was higher in the subjects with metabolic syndrome than in those without it (adiponectin: 5.4 ± 2.8 vs 7.5 ± 4.2 $\mu\text{g/ml}$, $p=0.002$; CRP: 0.832 ± 0.605 vs 0.470 ± 0.524 mg/L , $p=0.0004$).⁷ My group proposed 0.65 mg/L as the cut-off point of CRP for a component of metabolic syndrome among Japanese,⁸ and this cut-off point may also be appropriate as a predictor of cardiovascular risk among Japanese.^{9,10} We would deeply appreciate it if Otsuka et al would calculate the partial correlation coefficients between log adiponectin and age, HDL-cholesterol, fasting blood glucose, log HOMA-IR, BMI, log triglyceride, and log CRP in Japanese men with ECG abnormalities or angina-like symptoms, as Komatsu et al studied in apparently normal Japanese men.

References

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