

## ATVB In Focus

### Metabolic Syndrome and Atherosclerosis

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- Kotronen A, Yki-Järvinen. Fatty liver: a novel component of the metabolic syndrome. *Arterioscler Thromb Vasc Biol.* 2008;28:27–38.
- Gustafson B, Hammarstedt A, Andersson CX, and Smith U. Inflamed adipose tissue: a culprit underlying the metabolic syndrome and atherosclerosis. *Arterioscler Thromb Vasc Biol.* 2007;27:2276–2283.

## Abdominal Obesity and the Metabolic Syndrome: Contribution to Global Cardiometabolic Risk

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**Abstract**—There is currently substantial confusion between the conceptual definition of the metabolic syndrome and the clinical screening parameters and cut-off values proposed by various organizations (NCEP-ATP III, IDF, WHO, etc) to identify individuals with the metabolic syndrome. Although it is clear that in vivo insulin resistance is a key abnormality associated with an atherogenic, prothrombotic, and inflammatory profile which has been named by some the “metabolic syndrome” or by others “syndrome X” or “insulin resistance syndrome”, it is more and more recognized that the most prevalent form of this constellation of metabolic abnormalities linked to insulin resistance is found in patients with abdominal obesity, especially with an excess of intra-abdominal or visceral adipose tissue. We have previously proposed that visceral obesity may represent a clinical intermediate phenotype reflecting the relative inability of subcutaneous adipose tissue to act as a protective metabolic sink for the clearance and storage of the extra energy derived from dietary triglycerides, leading to ectopic fat deposition in visceral adipose depots, skeletal muscle, liver, heart, etc. Thus, visceral obesity may partly be a marker of a dysmetabolic state and partly a cause of the metabolic syndrome. Although waist circumference is a better marker of abdominal fat accumulation than the body mass index, an elevated waistline alone is not sufficient to diagnose visceral obesity and we have proposed that an elevated fasting triglyceride concentration could represent, when waist circumference is increased, a simple clinical marker of excess visceral/ectopic fat. Finally, a clinical diagnosis of visceral obesity, insulin resistance, or of the metabolic syndrome is not sufficient to assess global risk of cardiovascular disease. To achieve this goal, physicians should first pay attention to the classical risk factors while also considering the additional risk resulting from the presence of abdominal obesity and the metabolic syndrome, such global risk being defined as cardiometabolic risk. (*Arterioscler Thromb Vasc Biol* 2008;28:1039-1049)

**Key Words:** global cardiometabolic risk ■ insulin resistance ■ metabolic syndrome ■ visceral obesity  
■ waist circumference

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