Adipose tissue can release a plethora of factors termed adipokines. The large family of adipokines includes chemokines, cytokines, lipid factors, and growth factors. Adiponectin, the first cloned protein hormone from adipose tissue, is a metabolically active and anti-inflammatory adipokine. Adiponectin mainly circulates in different oligomeric isoforms, including high molecular weight adiponectin, that exerts different biological effects. Expression of adiponectin is suppressed by proinflammatory factors, reactive oxygen species, and hypoxia, whereas peroxisome proliferator activated receptor γ agonists stimulate the production of adiponectin in adipocytes. Adiponectin can improve insulin sensitivity, dampen inflammatory responses in macrophages, and induce the polarization of M2 macrophages. Adiponectin is negatively associated with obesity and insulin resistance, both well-established comorbidities in cardiovascular disease (CVD). The role of adiponectin in CVD itself is debatable because high levels of adiponectin have been associated with decreased CVD risk in asymptomatic individuals, whereas it can also predict poor prognosis in patients with established CVD.

Adiponectin Regulation in Cardiovascular Disease
Is Diseased Fat Showing Its True Color?

Hester M. den Ruijter, Gerard Pasterkamp, Saskia C.A. de Jager

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proof of concept was further substantiated in healthy individuals, where induction of low-grade inflammation resulted in reduced adiponectin levels, independent of BNP.

Despite the novel and interesting concept, some limitations of the study should be taken into account. Different patient groups have been studied, patients undergoing percutaneous coronary intervention or coronary artery bypass grafting, who may differ in syntax scores, risk factor profiles, and stability of CVD, all affecting circulating adiponectin levels. In addition, the model resembling mild inflammation, Salmonella Typhi vaccination, was administered in healthy patients who were on average 30 years younger than the patients with CAD. Besides, the model most likely does not reflect the chronic inflammatory process involved in atherosclerosis.

Still, the study by Antonopoulos et al13 provides the reader with an interesting novel concept of variability in adipocyte responsiveness in healthy and diseased adipose tissue. Furthermore, it is clearly established that cardiac-derived BNP is the critical denominator of adiponectin responses in patients with severe CVD (Figure). It is intriguing to speculate that in CVD BNP may affect fat composition by browning of adipocytes, thereby possibly altering adiponectin release. In fact, it has been established that natriuretic peptides can promote a favorable fat distribution profile (with decreased visceral fat, increased lower body fat, and improved insulin sensitivity)14 and induce the browning of adipocytes.15 It is of high interest to determine whether the diseased adipose tissue from patients with CVD contains brown fat deposits, either consisting of classical or inducible brown fat, that may directly affect morphological and functional differences compared with healthy adipose tissue.

Whether or not adiponectin is a valuable biomarker for the prognosis of CVD remains elusive because this study provides circulating adiponectin levels at a single time point only. As mentioned earlier, adiponectin circulates in different isoforms, depends on type of CVD, stability of CVD, and other biomarker levels and may also depend on the fat type they are excreted from. This interesting study raises many questions of which we eagerly await the answers.

Disclosures

None.

References


Key Words: adipose tissue • adiponectin • brain natriuretic peptide • biomarker • brown fat • cardiovascular disease
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