ADIPONECTIN THROUGHOUT THE CARDIOVASCULAR DISEASE CONTINUUM: REDOX STATE IN THE FAILING MYOCARDIUM AS A REGULATOR OF CIRCULATING ADIPONECTIN LEVELS

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Background: Clinical studies have yielded conflicting results on the role of adiponectin in cardiovascular disease (CVD). We evaluated the role of adiponectin at the different stages of CVD, as well as its associations with myocardial redox state.

Methods: The study population consisted of 746 individuals: 130 healthy non-obese individuals (BMI<28), 74 healthy obese individuals (BMI≥28), 283 coronary artery disease (CAD) patients and LVEF>40%, 225 patients with ischemic heart failure (LVEF<40%) and 34 patients with non-ischemic heart failure (LVEF<40%, no CAD). Myocardial O2- generation was determined by lucigenin-enhanced chemiluminescence while urate-inhibitable luminol chemiluminescence was used to estimate ONOO- generation. NADPH oxidase activity was estimated by defining NADPH-stimulated O2- and its apocynin-inhibitable fraction, uncoupled nitric oxide synthase contribution by using LNAME and mitochondrial oxidases by using rotenone.

Results: Obesity was associated with lower adiponectin levels among healthy individuals. However, serum adiponectin was higher in CAD compared to obese or non-obese healthy individuals, while the development of HF induced a striking further elevation of serum adiponectin, independently of the existence of underlying coronary atherosclerosis. Adiponectin was correlated negatively with LVEF (r=-0.414, p=0.0001). Importantly serum adiponectin was inversely associated with the extent of CAD (p<0.0001) and positively with myocardial NADPH-stimulated O2- (r=0.288, p=0.014) and its apocynin-inhibitable fraction (r=0.250, p=0.022) independently of LVEF. No association was found between adiponectin and resting O2-, mitochondrial oxidases or urate inhibitable ONOO- (p=NS for all).

Conclusions: Left ventricular dysfunction is associated with increased adiponectin levels compared to healthy or CAD patients. Myocardial redox state is a key regulator of plasma adiponectin and myocardial NADPH-derived O2- is positively associated with plasma adiponectin. Failing myocardium’s altered redox state may lead to increased adiponectin biosynthesis in heart failure patients, possibly as a defense mechanism.