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[Circulation. 2008;118:S_562](#)*(Circulation. 2008;118:S_562.)*

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Coronary, Regional and Microcirculation

Abstract 5502: Adiponectin Mediates the Effects of Atherogenic Risk Profile on the Coronary Microcirculation in Patients with Idiopathic Lv Systolic Dysfunction

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Purpose. Adiponectin (ADN), a biologically active protein produced by the adipose tissue, has protective vascular effects. Accordingly, ADN plasma levels are reduced in patients with coronary artery disease (CAD) while in heart failure ADN tends to increase. We hypothesized that ADN plasma levels could mediate the effects of atherogenic risk profile on the coronary microcirculation of patients with early LV systolic dysfunction (ILVDys) not secondary to established CAD.

Methods. Plasma ADN was measured in 55 patients (age 59 ± 1 yrs, 36 males, BMI 26.9 ± 0.49 Kg/m², mean \pm sem) with angiographically normal coronary arteries, LV systolic dysfunction (LVEF 39.8 ± 1.3 % range 22–54 %) but without overt heart failure (NYHA class I–II) and in 40 age- and BMI-matched healthy controls by using a specific Elisa (Linco Res). BMI, cholesterol and glucose profiles were assessed in all. In a subset of 25 patients coronary microvascular function was studied by PET and ¹³N-Ammonia as a flow tracer. Myocardial blood flow (MBF) was measured at rest and during i.v. dipyridamole (Dip) (0.56 mg/Kg in 4 min).

Results. ADN was 6.6 ± 0.34 μ g/ml in controls and 10.9 ± 0.85 in ILVDys patients ($p<0.001$). In patients ADN levels were inversely related with BMI ($p=0.009$) and directly related with age ($p=0.007$), HDL Cholesterol ($p=0.003$) and MBF Dip (0.020). Patients showing more severe coronary microvascular dysfunction (MBF Dip <1.42 ml/min/g, median value in patients) had significantly depressed ADN (9.7 ± 2.3 vs 13.7 ± 1.6 , $p=0.021$) as compared with the remaining patients.

Conclusions. This is the first study which associates adiponectin plasma levels with atherogenic risk profile and coronary microvascular function in patients with idiopathic LV dysfunction. These results suggest that adiponectin signal is strongly involved in mediating coronary vascular function independently of the presence of overt CAD or heart failure.

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