

Inflammation and insulin-resistance affect dendritic cell function

A. Parenti¹, S. Paccosi¹, L. Pala², A. Silvano², V. Barbaro³, P. Romagnoli², C.M. Rotella²

¹Depts. of Health Sciences, Clinical Pharmacology and Oncology Section and ²Experimental and Clinical Medicine University of Florence, Italy

³Azienda Ospedaliero-Universitaria Careggi, Florence, Italy

Dendritic cells (DCs) are antigen presenting cells, which are also localized in the healthy human arterial wall and increase in the atherosclerotic lesion, where they may be involved in driving the chronic vascular inflammation (1). Obesity and insulin resistance are well known risk factors for cardiovascular disease, and diabetic patients with atherosclerotic disease have compromised immune functions (2). We have previously reported that obese and diabetic patients (obese T2D) show quantitative abnormalities in circulating myeloid precursors of DCs (3). We then further in vitro characterized DC obtained in vitro from obese T2D patients. Real time PCR demonstrated that DCs from T2D patients significantly increased the expression of CD18, CD11c and DC-SIGN, which may mediate their increased adhesion to vascular smooth muscle cells, previously reported (3). Flow cytometry analysis of the mature myeloid DCs showed that these cells were significantly lower in obese and T2D patients, compared with healthy and obese subjects. CFSE analysis demonstrated that T2D DCs were less able to stimulate lymphocyte proliferation, in line with the phenotypical characterization, compared to control subjects. Interestingly, no morphological differences were found between T2D DCs and healthy controls, by means of electron microscopy. In conclusion, inflammation and insulin-resistance affect DC phenotype and function, such as their interaction with vascular smooth muscle cells. Therefore DCs may be candidate to a major role in the onset and progression of vascular inflammation and remodeling, suggesting that they could be a target for therapy.

References

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