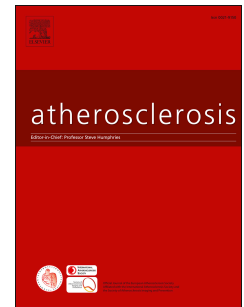


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Johanna M. Gostner, Dietmar Fuchs



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Biomarkers for the role of macrophages in the development and progression of atherosclerosis

Johanna M Gostner¹, Dietmar Fuchs^{2*}

¹Division of Medical Biochemistry, Biocenter, Innsbruck Medical University, Innsbruck, Austria

²Division of Biological Chemistry, Biocenter, Innsbruck Medical University, Innsbruck, Austria

*Corresponding author. Division of Biological Chemistry, Biocenter, Innsbruck Medical University, 6020 Innsbruck, Austria. Tel.: +43 512 9003 70350; fax: +43 512 9003 73330
E-mail address: dietmar.fuchs@i-med.ac.at (D. Fuchs).

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Multiple factors such as genetics, immunological status and environmental factors are involved in the development of cardiovascular disorders (CVD), providing a challenge for the identification of early disease markers. Pathological changes are suggested to occur at very early phases, long before symptoms become apparent, and may involve several cell types and organ systems such as components of the vascular, immunologic, metabolic and endocrine system, finally leading to cholesterol accumulation [1;2]. Immune cells are recruited via chemotactic signals to the lesion where they accumulate. Their activation results in local and finally systemic inflammatory responses characterized by the production of high levels of reactive oxygen species (ROS). Such inflammatory events are considered to play a key role in both disease initiation and progression [3]. Activation, differentiation and immobilization of monocyte-derived macrophages are considered as key events in the inflamed plaque [4].

A wealth of studies exists on the role of monocyte-derived macrophages in plaque destabilization and provocation of local inflammation. However, only few reports tried to address the relation with systemic monocyte activation at very early disease stages. The recent findings, defining neoangiogenesis as a feature of plaque instability and as a risk factor for cardiovascular events, have shed new light on the role of monocytes in these processes [5;6]. It is still a largely unanswered question to which extent qualitative and quantitative changes of circulating monocyte subpopulations can be related to early events of plaque atherosclerosis. In their recent study, Ammirati et al. reported significantly lower counts of circulating CD14⁺

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