1. Record Nr. UNISA996215988903316

Titolo	International journal of innovation management
Pubbl/distr/stampa	London, : Imperial College Press, : World Scientific, c1997-
ISSN	1757-5877
Descrizione fisica	1 online resource
Soggetti	Technological innovations - Management Vernieuwing Management Periodicals.
Lingua di pubblicazione	Inglese
Formato	Materiale a stampa
Livello bibliografico	Periodico
Note generali	Refereed/Peer-reviewed

2. Record Nr. UNINA9910557600203321

Autore	Corti Alessandro
Titolo	Targeting Monocytes/Macrophages to Treat Atherosclerotic Inflammation
Pubbl/distr/stampa	Frontiers Media SA, 2020
Descrizione fisica	1 online resource (127 p.)
Soggetti	Pharmacology Science: general issues
Lingua di pubblicazione	Inglese
Formato	Materiale a stampa
Livello bibliografico	Monografia
Sommario/riassunto	It is by now widely recognized that atherosclerosis - with its burden of

consequences in cerebro- and cardiovascular diseases - is just a chronic inflammatory process of the arterial wall. A very peculiar, complex and as yet still poorly understood process, upon which hundreds of scientists from several different fields are continuously concentrating their investigative efforts in search of possible leads to therapeutic approaches. Initiation of the disease is given by deposition of lipid in the intimal layers, resulting in endothelial activation and infiltration of blood-derived mononuclear cells. These mature into macrophages, become activated, express scavenger receptors such as SR-A and CD36 and ingest the oxidized lipoprotein accumulating in the lesion. Macrophages thus represent an obvious target for intervention, as they play a crucial role in the progression of the atherosclerotic inflammation. Studies have shown that hypercholesterolaemia can increase monocyte mobilisation from bone marrow into the circulation, and several chemokines and their receptors are involved in the recruitment of blood borne monocytes into the arterial wall. Monocyte-derived macrophages are capable of sustaining their local proliferation, but resident macrophages possibly also participate in progression of the disease. Remarkably, smooth muscle cells can acquire macrophage-like features during atherogenesis, including the ability to uptake lipid, thus becoming a significant proportion of the CD68+ so called 'foam cells'. Lipid-laden macrophages induce extracellular matrix degradation, while lipid uptake eventually causes their death with formation of a necrotic core. The efficiency in clearance of dead cells by phagocytes (efferocytosis), can also be considered as a determinant of plaque vulnerability. An important feature of macrophages is their great plasticity and functional diversity in response to signals from the plaque microenvironment. Several such 'signals' (cholesterol, oxidative stress, hypoxia, cytokines...) can in fact modulate cell differentiation at transcriptional and epigenetic levels, thus altering the balance between the effector vs. reparative functions of macrophages. A whole gamut of specific subsets are thus originated, which appear to be simultaneously present in lesions with proportions that vary according to their location, the disease stage, and the presence of additional cell types such as e.g. dendritic cells. The result is a multiplicity of potential pharmacological targets, representing a major obstacle for the devisement of therapeutic strategies. Experimental approaches have been attempted in diverse directions: e.g. modulating the macrophage phenotype to an anti-inflammatory and resolving state, or blocking pro-inflammatory cytokines that macrophages produce, or alternatively enhancing efferocytosis in order to favour the resolution of inflammation and stabilization of plaques. Blocking monocyte recruitment was proposed in order to hinder the initial steps of atherogenesis. Other treatments were aimed to inhibiting local proliferation of pro-inflammatory macrophages. Specific targeting of macrophages has however to date not yet provided significant, translational results. The present Research Topic collects articles to help unravel the complexity of macrophage behaviour in atherosclerosis and identify innovative pharmacological approaches.