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Cytokines in atherosclerosis disease progression: Roles, mechanisms of actions and current therapeutic approaches

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Atherosclerosis, the underlying cause of heart attack and stroke, is an inflammatory disorder of the vasculature regulated by both the innate and adaptive immune systems. Cytokines play a pivotal role in controlling the inflammatory response in atherosclerosis and regulate all the different stages in disease progression. Current approaches to target pro-inflammatory cytokines include neutralization using blocking antibodies or soluble decoy receptors and the use of specific inhibitors against key components of intracellular signaling pathways. In contrast, approaches for anti-atherogenic cytokines include their local delivery and the use of agents that augment their expression/actions. Numerous cytokines are expressed in atherosclerotic lesions and it is therefore essential that their actions in disease progression are fully understood to validate their therapeutic potential and to identify potentially new targets or approaches for therapeutic intervention. My laboratory has recently been investigating cytokine signaling in atherosclerosis, particularly in macrophages that play key roles in all stages of disease progression, using a combination of *in vitro* and *in vivo* approaches. Novel insights have been obtained on the actions of the cytokines interferon-gamma, transforming growth factor-beta, interleukin-33 and tumor necrosis factor-like protein 1A on key macrophage processes in atherosclerosis (e.g. foam cell formation, regulation of inflammation). For example, we have identified a key role for extracellular signal-regulated kinase: signal transducer and activator of transcription-1 serine 727 phosphorylation axis in the control of macrophage foam cell formation and the regulation of pro-inflammatory gene expression by interferon-gamma. The outcome of our studies on different cytokines will be presented in the context of current therapies and future developments in this field.

Biography

Dipak P Ramji received his BSc (Hons) degree (Biochemistry) and his PhD from University of Leeds. This was followed by Post-doctoral research at the EMBL (Heidelberg) and IRBM (Rome) with fellowships from the Royal Society and the EU. He joined Cardiff University in 1992 and is currently a Reader at Cardiff School of Biosciences. His research is focused on understanding how the immune and inflammatory responses regulate macrophage processes in atherosclerosis with the goal of attaining deeper mechanistic insight and identifying preventative/therapeutic agents. He has published over 80 peer-reviewed papers, reviews and book chapters (h-index=30; i10-index=57). He is an Editorial Board Member of 16 international journals.

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