The term inflammation, from the Latin inflammare (to set on fire), was first used 2000 years ago by the Roman encyclopedist Aulus Cornelius Celsus, who documented the 4 cardinal signs of inflammation: rubor et tumor cum calore et dolore (redness and swelling with heat and pain). Two centuries later, the Greek physician Galen promoted the idea that inflammation, especially pus, was a beneficial response to injury. This view persisted until the 19th century, when Rudolf Virchow, who considered inflammation a pathological condition, added loss of function (functio laesa) to the list as the fifth cardinal sign of inflammation. Nowadays, inflammation is defined as “a complex set of interactions among soluble factors and cells that can arise in any tissue in response to traumatic, infectious, postischemic, toxic or autoimmune injury.”1 It plays a central role in cardiovascular disease, and patients experiencing inflammatory disorders of various causes, including autoimmunity, are now considered at increased risk of developing cardiovascular disease. Interestingly, most of the 9 risk factors (smoking, lipids, hypertension, diabetes, obesity, diet, physical activity, alcohol consumption, and psychosocial factors) that account for more than 90% of the risk of acute myocardial infarction in the INTERHEART study2 can act as stressors that provoke inflammatory responses.

In this issue of Arteriosclerosis, Thrombosis, and Vascular Biology, there are 5 articles focusing on the topic of inflammation in various cardiovascular diseases, notably atherosclerosis.3 HSPs are highly conserved proteins from mammals and microbial reagents. Acting as a danger signal following infections, HSP60 induces the production of anti-HSP60 antibodies. However, as endothelial cells express HSP60 in response to a number of classical atherosclerosis risk factors, they may become a target for preexisting anti-microbial HSP60 antibodies.

The next 2 articles focus on new advances in the role of cytokines and the janus kinase/signal transducer and activator of transcription intracellular signaling pathway in vascular inflammation. The chronic inflammatory disease of the arterial wall in atherosclerosis is promoted by both innate and adaptive Th1-driven immunity and orchestrated by a complex network of proinflammatory cytokines that can be counter-balanced by antiinflammatory/antiatherogenic cytokines, including interleukin (IL)-10 and transforming growth factor-β. Ait-Oufella et al make a point in the current controversy regarding the role of the recently discovered Th17 cell population that produces IL-17A, IL-17F, IL-21, and IL-22.4 Interestingly, STAT3 has been reported as the essential regulator of Th17 cells. Phosphorylation of intracellular STAT molecules by cytokine stimulation. The janus kinase/signal transducer and activator of transcription pathway is regulated by suppressor of cytokine signaling proteins. In his article, Yoshimura provides new insights into the regulation of inflammation and immune responses by these proteins.5

Ricciotti and Fitzgerald discuss new advances in the biosynthesis of prostaglandins and their role in regulating cardiovascular inflammatory responses, with a particular emphasis on the prostaglandin receptors.6 Atherosclerosis and other cardiovascular diseases, including aneurysm, clearly result from uncontrolled inflammatory responses. A better understanding of the mechanisms involved in the resolution of inflammation is of the utmost importance to develop novel approaches to combat these disorders. In the final article, Maskrey et al discuss the contribution of newly identified lipid mediators, including the resolvins, protectins, and ma-

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resins, as potent specialized molecules that control inflammation and promote its successful resolution.\(^7\)

In recent years there have been major advances in the understanding of the mechanisms of inflammation and its role in cardiovascular disease, especially in atherosclerosis. This review series on inflammation aim at providing *Arteriosclerosis, Thrombosis, and Vascular Biology* readers with new insights into this rapidly evolving field. We look forward to translating these advances into clinical application.

**References**


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