Focus on Inflammation
Alain Tedgui

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.” 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 study 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. The first article, by Grundtman et al, brings new insights into the role of heat shock proteins (HSPs) in atherosclerosis. 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 antimicrobial 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 counterbalanced 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. Interestingly, STAT3 has been reported as the essential regulator of Th17 cells. Phosphorylation of intracellular STAT molecules by the janus kinase/signal transducer and activator of transcription pathway is regulated by suppressor of cytokine stimulation. The janus kinase/STAT pathways: regulation of T-cell inflammation by SOCS1 and SOCS3.


resins, as potent specialized molecules that control inflammation and promote its successful resolution.\textsuperscript{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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