Inflammation and immunity: role in vascular disease

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Over the past few years it has become increasingly appreciated that an inflammatory response that comprises immune mechanisms underlies the development of vascular disease in many conditions. We have studied specifically the role of inflammation and immunity in hypertension, diabetes and chronic kidney disease, in both humans and rodents. In mice, oxidative stress may trigger inflammation in the vascular wall in response to different mediators such as angiotensin II, endothelin-1 or aldosterone, whereas peroxisome proliferator activator receptors, which are nuclear receptors with significant metabolic effects, have anti-inflammatory actions. How the immune mechanisms are triggered remains unclear, but macrophage/monocytes and T lymphocytes are involved mediating the inflammatory and immune response found in the wall of vessels, both large and small, in hypertension, diabetes and chronic kidney disease, and as essential components participating in the progression of atherosclerosis. Among lymphocytes we have shown that unconventional innate-like gamma/delta T lymphocytes may participate as a link between the innate (macrophage/monocyte) immune response and adoptive immunity. The balance between pro-inflammatory alpha/beta T helper lymphocytes (mostly Th1) that produce gamma interferon, and anti-inflammatory T regulatory lymphocytes (Treg) that produce interleukin 10, will determine the degree of inflammation in the vascular wall and perivascular fat. This low-grade inflammatory and immune response drives vascular disease. Whether specific vascular anti-inflammatory treatments will be effective in preventing progression or contribute to regress vascular disease associated with hypertension, diabetes, chronic kidney disease or atherosclerosis, improving outcomes without adverse side effects due to immune suppression remains to be demonstrated.