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Role of Inflammasome Activation in Atherosclerosis

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Message from the Guest Editors

Cardiovascular diseases (CVD) are still the predominant cause of death and morbidity, with atherosclerosis as the main underlying cause. Innate-immunity-induced inflammation plays a major role in promoting the atherosclerotic plaque, resulting in myocardial infarction and stroke. Inflammasome biology is one of the most exciting and rapidly growing areas in immunology. Inflammasome includes NLRP1, NLRP2, NLRP3, AIM2, and NLRC4 have attracted more attention in recent studies. We are pleased to invite you to help clarify the mechanism of inflammasome in atherosclerosis process, which will facilitate the development of precise therapeutic strategies for CVD. Since inflammasome activation has been shown to be an important mechanism driving atherogenesis, inflammation, and foam cell formation, it could emerge also as a crucial mechanism triggering vascular endothelial cell damage, vascular smooth muscle cells phenotypic switch, and subsequently, plaque destabilization. Until now, this hypothesis has not been investigated, and it could open a door to the revelation of a new mechanism in vascular pathology.

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Message from the Editor-in-Chief

Vaccines (ISSN 2076-393X) has had a 6-year history of publishing peer-reviewed state of the art research that advances the knowledge of immunology in human disease protection. Immunotherapeutics, prophylactic vaccines, immunomodulators, adjuvants and the global differences in regulatory affairs are some of the highlights of the research published that have shaped global health. Our open access policy allows all researchers and interested parties to immediately scrutinize the rigorous evidence our publications have to offer. We are proud to present the work and perspectives of many to contribute to future decisions concerning human health.

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