Oxidative Medicine and Cellular Longevity

Hindawi

Special Issue on

Oxidation and Inflammation in Atherosclerotic Plaque Development and Progression

CALL FOR PAPERS

Cardiovascular diseases are the leading cause of death and illness in developed countries, and atherosclerosis is the underlying pathology and their common denominator. Cardiovascular risk factors such as hypertension, diabetes, and hyperlipidaemia play a key role in the onset and progression of atherosclerosis. Atherosclerosis, a chronic inflammatory condition, develops and evolves in a site-specific and patient-specific manner, with great heterogeneity in growth rate and features. Atherosclerotic plaques are commonly characterized by accumulation of lipids and fibrous elements in the intimal layer of medium and large arteries. Ulceration and rupture of the atherosclerotic plaques promote thrombosis driving acute clinical events. Indeed, plaque disruption is a common precipitating factor in the pathogenesis of acute coronary occlusion and peripheral artery thrombosis. The mechanisms underlying plaque formation and progression to advanced lesions are not yet completely understood. Despite current systemic application of therapies such as statins and antiplatelet agents for the prevention of plaque rupture, most of major adverse cardiovascular events cannot be averted.

Understanding the mechanisms that promote thin fibrous cap formation and disruption would help to effectively counteract the release of prothrombogenic elements and prevent acute thrombotic occlusion. It is generally held that plaque instability is caused by a substantial increase in inflammatory and proteolytic activity. Furthermore, some lines of evidence suggest that unstable plaques are also characterized by pronounced oxidative environment. *In situ* oxidative events may determine lipid/protein metabolic fate, bioactivity, and antigenic properties. In this respect, oxidized LDL is readily internalized by macrophages through the so-called "scavenger receptor" pathway. These early modifications could initiate and/or contribute to atherogenesis, mainly when an imbalance between oxidant and antioxidant agents takes place. Although several studies report that atherosclerotic plaques contain high concentrations of several amino acid oxidation products, caused mainly by carbonylation, ROS, and RNS oxidation or thiolation, limited information is available regarding the relationship between the accumulation of markers of oxidized proteins and severity of atherosclerosis. Furthermore, different types of oxidation-specific epitopes can be detected in blood and may reflect atherosclerosis and its different manifestations. At present, the mechanisms underlying the formation of these byproducts and the relevance for disease progression are not completely understood and deserve further investigation.

In this special issue, we are inviting researchers to contribute original research as well as review articles that seek to address the mechanisms underlying lesion vulnerability, with particular emphasis to the prooxidant and proinflammatory plaque environment.

Potential topics include but are not limited to the following:

- Oxidative and inflammatory mechanisms leading to plaque instability
- *In situ* protein/lipid oxidative modification and their metabolic fate
- Lipoprotein metabolism and onset of advanced lesions
- Circulating markers of atherosclerosis
- Risk factors and development of stable/unstable advanced lesions
- Imaging methods for identifying anatomic features of high-risk plaques
- Multiomics approaches for mapping the pathways implicated in patient vulnerability to plaque-related adverse events

Authors can submit their manuscripts through the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/omcl/oiap/.

Papers are published upon acceptance, regardless of the Special Issue publication date.

Lead Guest Editor
Antonio J. Lepedda, University of Sassari, Sassari, Italy
ajlepedda@uniss.it

Guest Editors
Marilena Formato, University of Sassari, Sassari, Italy
formato@uniss.it

Gualtiero Pelosi, National Research Council, Pisa, Italy
pelosi@ifc.cnr.it

Silvia Rocchiccioli, National Research Council, Pisa, Italy
silvia.rocchiccioli@ifc.cnr.it

Vicenta L. Cortes, Hospital de la Santa Creu i Sant Pau, Barcelona, Spain
cllorente@csic-iccc.org

Submission Deadline
Friday, 28 July 2017

Publication Date
December 2017