Mediators of Inflammation

Hindawi

Special Issue on

Interplay between Oxidative Stress and Inflammation in Cardiometabolic Syndrome

CALL FOR PAPERS

Cardiometabolic syndrome (CMS) is generally defined as the presence of at least 3 of the following interrelated risk factors and disease states that promote the development of atherosclerotic vascular disease and type 2 diabetes mellitus: obesity, as the hallmark component of CMS, insulin resistance, hypertension, and high triglyceride or low HDL plasma levels. In a number of definitions, obesity is a mandatory component with additional 2-3 risk factors required to make a diagnosis of CMS. Clinical importance of CMS is engendered in the fact that while each of the component conditions has an independent effect, their clustering has a synergistic effect above and beyond simple summation, making the risk of developing cardiovascular disease exponentially greater. Obesity with the associated components has a direct effect on atherogenic dyslipidemia, elevated blood pressure, and elevated plasma glucose that promotes proinflammatory and prothrombic states.

Increased oxidative stress has emerged as a major mediator of pathophysiological processes in obesity, CMS, and cardiovascular disease. While cellular oxidases are known sources of reactive oxygen species, mitochondria has now been implicated as a major source of reactive oxygen species, especially in diabetes, obesity, and cardiovascular diseases. Furthermore, oxidative stress-mediated mitochondrial dysfunction has emerged as a potential key mechanism behind a number of cardiovascular diseases.

Low-grade inflammation is a common manifestation in CMS and could play a role in the pathogenesis of obesity and cardiometabolic syndrome and its sequelae. Dysregulation of adipose tissue biology plays a potential role in the initiation of inflammatory events in obesity and CMS causing chronic inflammatory response characterized by abnormal adipokine production and the activation of several proinflammatory signaling pathways, resulting in the induction of several biological markers of inflammation.

We invite original research and review articles focusing on interaction of inflammation and oxidative stress in the setting of CMS.

Potential topics include, but are not limited to:

- Biomarkers of oxidative stress and inflammation in cardiometabolic syndrome
- Role of cardiac and adipose tissue mitochondria in pathophysiology of cardiometabolic syndrome or its sequelae
- Role of inflammation and oxidative stress in adipose tissue as an initiator/mediator of cardiometabolic syndrome
- Cardiometabolic syndrome and oxidative/proinflammatory signaling

Authors can submit their manuscripts via the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/mi/oscs/.

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