

## **Oxidative Stress versus Cardiovascular Complications**

## Sanjay Mishra\*

Department of Biotechnology, IFTM University, India

\*Corresponding author: Sanjay Mishra, Department of Biotechnology, IFTM

University, Delhi Road (NH 24), Moradabad 244102, Uttar Pradesh, India, Email: sanjaymishra@iftmuniversity.ac.in

## **Editorial**

In human vascular diseases, endothelial dysfunction is a systemic pathological state of the endothelium and can defined as an imbalance be largelv between vasoconstricting and vasodilating substances created by/or acting on the endothelium. In a pathological state such as the metabolic disorder, an augmented oxidant capacity combined with declined antioxidant capacity generates an uneven environment resulting in oxidative stress [1]. ROS have been observed to play a foremost role in the development and progression of cardiovascular disease. The overall relevant occurrence of cardiovascular complications can be summarized as: (i) Reports of the 'Third National Health and Nutrition Examination Survey' pin-point reduced concentrations of the antioxidants vitamins C. E and numerous carotenoids in spite of adjusting for lower vegetable and fruit consumption in participants with metabolic disorder, characterized by oxidative stress [2]; (ii) Obesity, a nuclear component in the expansion of metabolic disorder, plays a vital role in augmented oxidative stress [3], ROS creation in adipose tissue of obese mice abridging by treatment with the NAD(P)H oxidase inhibitor apocynin with progress in glucose and lipid metabolism independent of body weight; (iii) Contribution of insulin resistance to oxidative stress is complicated one, and investigations probably addressing the question of oxidative stress in type II diabetes characteristically do not differentiate between the study participants on the basis of obesity or their lipid profile, reflecting a noteworthy barrier with confirming whether insulin resistance on its own raises oxidative stress in humans [4]; (iv) Hyperglycemia consequential from primary  $\beta$ -cell devastation in lack of any other gears of the metabolic disorder has been revealed to connect with prominent oxidative stress (decreased glutathione, GSH/GSSG ratio) in type I diabetes [5]; (v) Dyslipidemia

with lipid peroxidation, acts as an index of oxidative stress, interrelated with low HDL levels, regardless of age, gender, and presence of the other metabolic disorder components [6]; (vi) Besides, hypertension, another independently connected with increased cardiovascular risk, has also been constantly associated with elevated oxidative stress [7]; (vii) With the constrained efficiency of the current treatments for occlusive Coronary Artery Disease (CAD), noteworthy effort has been aimed at developing alternative resources for coronary revascularization [8].

Editorial

Volume 1 Issue 1 Received Date: March 23, 2019

Published Date: April 06, 2019

Conclusively, certain constituent pathologies of the metabolic disorder contribute to a higher percentage of total oxidative stress than others; however, additional studies are needed to conclude the exact role of individual constituents to total oxidative stress [8].

## References

- 1. Sharma V, Kalim S, Srivastava MK, Nanda S, Mishra S, et al. (2009) Oxidative stress and coxsackievirus infections act as mediators of beta cell damage: a review. Scientific Research and Essays 4(2): 42-58.
- 2. Montero D, Walther G, Perez-Martin A, Roche E, Vinet A, et al. (2012) Endothelial dysfunction, inflammation, and oxidative stress in obese children and adolescents: markers and effect of lifestyle intervention. Obesity Reviews 13(5): 441-455.
- 3. de Mattia G, Bravi MC, Laurenti O, Arianna moretti, Rosalba cipriani, et al. (2008) Endothelial dysfunction and oxidative stress in type 1 and type 2 diabetic patients without clinical macrovascular

complications. Diabetes Research and Clinical Practice 79(2): 337-342.

- 4. de Oliveira J, Hort MA, Moreira ELG, Glaser V, Farina M, et al. (2011) Positive correlation between elevated plasma cholesterol levels and cognitive impairments in LDL receptor knockout mice: relevance of cortico-cerebral mitochondrial dysfunction and oxidative stress. Neuroscience 197: 99-106.
- 5. Andreadou I, Farmakis D, Prokovas E, Sigala F, Zoga A, et al. (2012) Short-term statin administration in hypercholesterolaemic rabbits resistant to postconditioning: effects on infarct size, endothelial nitric oxide synthase, and nitro-oxidative stress. Cardiovascular Research 94(3): 501-509.
- 6. Abdilla N, Tormo MC, Fabia MJ, Chaves FJ, Saez G, et al. (2007) Impact of the components of metabolic syndrome on oxidative stress and enzymatic antioxidant activity in essential hypertension. Journal of Human Hypertension 21(1): 68-75.
- 7. Reed R, Kolz C, Potter B (2008) The mechanistic basis for the disparate effects of angiotensin II on coronary collateral growth. Arteriosclerosis, Thrombosis, and Vascular Biology 28(1): 61-67.
- 8. Mishra S, Tiwari AKM, Mahdi AA (2017) Physiological, biochemical and molecular role of oxidative stress in cardiovascular disease: A comprehensive study. Current Research in Cardiovascular Pharmacology 6(1): 1-16.