Perspective
Volume 13:12, 2022

Journal of Molecular Biomarkers & Diagnosis

ISSN: 2155-9929 Open Access

Clinical Importance of Biomarkers of Oxidative Stress

Jeroen Frijhoff*

Department of Pharmacology, Taipei Medical University, Taipei 11031, Taiwan

Introduction

Various diseases are thought to be primarily caused by oxidative stress. The oxidation of DNA, proteins, lipids, and free amino acids are just a few examples of the many types of oxidative stress that have been studied and measured in virtually every disease. Tools for measuring oxidative stress markers, which are numerous and sometimes absent, have become more specific and sensitive as a result of a deeper comprehension of disease biology and redox biology.The literature is extremely diverse. Because only a small number of diseases use a variety of different biomarkers, and because different biomarkers have been used to study different diseases, it is frequently challenging to draw general conclusions regarding the significance of oxidative stress biomarkers. Nonspecific methods are also frequently used to measure biomarkers, while specific methods are frequently too complicated or time-consuming for routine clinical use.

Plans for the Future: Several oxidative stress markers remain clinically relevant biomarker opportunities. However, before they can be used as clinical diagnostics, positive results with the biomarkers that are currently in use need to be verified in larger sample sizes and contrasted with the standards that are currently in place in the medical field. It is essential to realize that oxidative stress is a complex and challenging phenomenon to characterize, and that one biomarker is not always superior to another. When selecting the most suitable biomarker, it is necessary to take into consideration the wide range of oxidative stress levels experienced by various diseases and conditions.

Description

ROS originate primarily from mitochondria in all cell types. Hydrogen peroxide can be made from superoxide, which is mostly produced at the level of the electron transport chain in mitochondria. SOD can also make hydrogen peroxide from superoxide (O2•). Through the Fenton reaction, H2O2 can produce the highly reactive hydroxyl radical (HO•) in the presence of transition metal ions, such as iron and copper ions. Xanthine oxidase (XO), uncoupled nitric oxide synthases (NOS), and NADPH oxidase (NOX) can also produce reactive species enzymatically. ROS production has been linked not only to cell death or damage, but also to physiological and signaling functions. The majority of the defensive pro-oxidants produced by neutrophils during their respiratory burst come from reactive species. Myeloperoxidase (MPO) and inducible nitric oxide synthase (iNOS) are the enzymes that neutrophils use to generate various reactive oxygen species (ROS) upon activation. While iNOS produces nitric oxide (NO•), which then reacts with oxygen to produce peroxynitrite (ONOO), MPO is the catalyst for the H2O2-dependent formation of hypochlorous acid (HClO). The production of superoxide radicals, which are necessary for cancer invasion, hypoxia, and integrin signaling, is catalyzed by NOX in the cell membrane. Through redox regulation of the nuclear factor-erythroid 2-related factor 2 (Nfr2) and the nuclear factor kappa-light-chain-enhancer of activated B cells (NF-kB), ROS can also alter the expression of a number of genes. Inflammation and cancergenesis have been linked to coordinated modulation of these pathways [1].

A group of conditions that have an effect on the heart and the blood vessels are known as cardiovascular diseases (CVDs). Coronary heart disease, rheumatic heart disease, peripheral arterial disease, cerebrovascular disease, and stroke are the primary types of cardiovascular diseases. CVD, which accounts for 31% of all deaths worldwide, is a complicated disease that is a leading cause of death worldwide. The majority of CVD cases are brought on by the accumulation of fatty acids in the blood vessels' inner walls. In 2016, approximately 17.6 million people died from cardiovascular disease. The number of facilities related to cardiovascular disease in the United Kingdom increased by 14.5 percent between 2006 and 2016, according to a report from the Royal College of Cardiologists. High cholesterol, smoking, and high blood pressure are all risk factors for cardiovascular disease, but oxidative stress remains a major factor. Free radicals and reactive oxygen species are produced by oxidative stress; DNA, lipids, and proteins are the primary structures in cells that are affected when these are abundant, resulting in cell death. It is known that ROS have both positive and negative effects on processes like response control, immune cell degeneration, and deregulation of the nervous system. In CVD, oxidative damage to the cell results in myocyte dysfunction and cell death. By altering the proteins involved in excitation-contraction coupling, ROS directly affect contractile function. Therefore, cardiovascular disease prevention and treatment should focus on reducing ROS production. However, due to the significant subcellular variations in redox potential and the short lifetime of ROS, analyzing redox systems is challenging. The identification of various oxidative stress biomarkers calls for additional research to evaluate CVD. Elucidating the pathophysiologic pathways that are responsible for the onset and progression of cardiovascular disease (CVD) remains a work in progress, despite significant efforts.

Atherosclerosis and cardiovascular disease are both exacerbated by oxidative stress and inflammation. Our previous study found that older adults' systolic blood pressure (BP) and low-density lipoprotein (LDL) cholesterol levels decreased after 12 weeks of drinking tart cherry juice. Blood biomarkers of inflammation and oxidative stress were examined in this study to see how tart cherry juice affected them. A total of 37 men and women between the ages of 65 and 80 were randomly assigned to take 480 milliliters of tart cherry juice or the control drink each day for 12 weeks in this randomized controlled clinical trial. At the beginning of the treatment and after 12 weeks, several blood biomarkers of oxidative stress and inflammation were evaluated [2-5].

Conclusion

In conclusion compared to the control group, tart cherry juice significantly reduced the mean c-reactive protein (CRP) level (p=0.03) and significantly increased plasma levels of 8-oxoguanine glycosylase's DNA repair activity (p 0.0001). Plasma oxidized low-density lipoprotein (OxLDL) had a borderline significant group effect (p=0.07), and plasma CRP (p=0.03) and malondialdehyde (MDA) had a significant group effect (p=0.03). After 12 weeks of drinking tart cherry juice, group analysis revealed that the plasma levels of CRP, MDA, and OxLDL decreased numerically by 25%, 3%, and 11%, respectively, in comparison to the corresponding baseline values. According to the current study, tart cherry juice's ability to lower systolic blood pressure

***Address for Correspondence:** Jeroen Frijhoff, Department of Pharmacology, Taipei Medical University, Taipei 11031, Taiwan; E-mail: fri.jeroen@gmail.com*

Received: 02 December, 2022, Manuscript No. jmbd-23-89569; **Editor Assigned:** 05 December, 2022, PreQC No. P-89569; **Reviewed:** 18 December, 2022, QC No. Q-89569; **Revised:** 23 December, 2022, Manuscript No. R-89569; **Published:** 30 December, 2022, DOI: 10.37421/2155-9929.2022.13.557

and LDL cholesterol may be partially attributable to its anti-oxidative and anti-inflammatory properties. These findings need to be confirmed by larger and more prolonged follow-up studies.

Acknowledgement

None.

Conflict of Interest

There are no conflicts of interest by author.

References

1. Shiode, Nobuo, Nobuyuki Morishima, Kensyo Nakayama and Togo Yamagata, et al. "Flow-mediated vasodilation of human epicardial coronary arteries: effect of inhibition of nitric oxide synthesis." *J Am Coll Cardiol* 27 (1996): 304-310.
2. Alsaied, Tarek, Jouke P. Bokma, Mark E. Engel and Joey M. Kuijpers, et al. "Factors associated with long-term mortality after Fontan procedures: A systematic review." *Heart* 103 (2017): 104-110.
3. Alsoufi, Bahaaldin, Scott Gillespie, Dennis Kim and Kirk Kanter, et al. "The impact of dominant ventricle morphology on palliation outcomes of single ventricle anomalies." *Ann Thora Surg* 102 (2016): 593-601.
4. Alsoufi, Bahaaldin, Cedric Manlhiot, Abid Awan and Fadel Alfadley, et al. "Current outcomes of the Glenn bidirectional cavopulmonary connection for single ventricle palliation." *Eur J Cardiothorac Surg* 42 (2012): 42-49.
5. Anderson, Page A.W., Lynn A. Sleeper, Lynn Mahon and Steven D. Colan, et al. "Contemporary outcomes after the Fontan procedure: A Pediatric Heart Network multicenter study." *J Am Coll Cardiol* 52 (2008): 85-98.

How to cite this article: Frijhoff, Jeroen. "Clinical Importance of Biomarkers of Oxidative Stress." J Mol Biomark Diagn 13 (2022): 557.