SHORT COMMUNICATION

Cardiovascular diseases and the significance of nitric oxide

Andrea Watson*

Watson A. Cardiovascular diseases and the significance of nitric oxide. Cardiol Curr Res 2023;10(1):1.

ABSTRACT

Nitric Oxide (NO), commonly known as nitrogen monoxide, is a colorless, gas created when nitrogen is oxidised. And it has a number of uses in medicine and plays a significant role in chemical signaling in both humans and other animals. The cellular messenger Nitric oxide (NO) is produced by three different isoforms of Nitric Oxide Synthases (NOS), including neuronal (nNOS), inducible (iNOS), and endothelial NOS (eNOS). The prevention of the development and progression of cardiovascular disease is significantly aided by NO. Hypercholesterolemia, hypertension, and diabetes are only a few of the illnesses that cardiovascular disease is linked to. Most cardiovascular disorders have atherosclerosis as their underlying pathology, which is linked to endothelial dysfunction. NO plays a number of cardio protective activities, including controlling blood pressure and vascular tone, preventing smooth muscle cell growth, and inhibiting platelet aggregation and leukocyte adhesion. This review will concentrate on the function of NO in the cardiovascular system, where it inhibits platelet adhesion and aggregation, maintains a vasodilator tone, and controls smooth muscle cell proliferation. Numerous cardiovascular disorders have been linked to NO, and almost all of these risk factors appear to be connected to a decrease in endothelial NO production.

Keywords: Nitric oxide; Hypertension; diabetes; Reactive oxygen species; endothelial dysfunction

INTRODUCTION

Nitric oxide is a relatively recent addition to pharmacology. The endothelium continuously produces nitric oxide, a soluble gas. A crucial signaling molecule in the cardiovascular system is Nitric Oxide (NO). Controlling hemostasis, fibrinolysis, platelet and leukocyte interactions with the arterial wall, regulating vascular tone, promoting the growth of vascular smooth muscle cells, and maintaining blood pressure homeostasis are just a few of the regulatory processes that NO takes part in most people with Cardiovascular Disease (CVD) live in industrialized nations with high standards of living. One of the leading causes of death worldwide is CVD [1]. The UK has one of the worst rankings inside Europe. This might be connected to how social class groups are distributed in certain areas. In nations on the Indian subcontinent, it is increasing. Reduced blood pressure greatly lowers the risk of cardiovascular events because hypertension is a key risk factor for cardiovascular disease. Nitric Oxide (NO) bioavailability impairment is a sign of endothelial dysfunction, which is a significant risk factor for cardiovascular disease and hypertension and may be a significant link between the two diseases. Numerous treatments have been looked into to see if they can improve the release of nitric oxide from the endothelium and potentially reverse endothelial dysfunction [2]. These treatments range from stimulating nitric oxide synthesis to safeguarding nitric oxide from oxidative inactivation and conversion to toxic molecules like peroxynitrite. There is evidence that NO regulates blood pressure significantly, and that diminished NO bioactivity is a key factor in hypertension. The prevalence of CVD in Japan is minimal but could increase with more westernization. Coronary heart disease, stroke, and peripheral vascular disease are just a few of the illnesses that go under the umbrella term "cardiovascular disease." However, atherosclerosis is the pathophysiology that underlies each of these diseases. Atherosclerosis has a very complicated pathophysiology that incorporates all of the artery wall's structural components, circulating cells like platelets and leukocytes, (especially monocytes and macrophages) and a multitude of inflammatory factors. The vascular endothelium, which serves as a dynamic contact between the circulation and the artery wall, is essential to the process. Although the arterial wall changes throughout the course of a person's lifetime, atherosclerosis is a disease process that is distinct from ageing. It might even start in early life and undoubtedly exists in many teenagers in the most industrialized countries. Up to the later stages, it is typically not life-threatening in and of itself, but many are killed off in their middle years [3].

DISCUSSION

Numerous investigations have shown that essential hypertension is accompanied by poor endothelial dysfunction, which is linked to a slowed response to NO mediated effects. The therapeutic use of NOS inhibitors and knock-out mice provides the most compelling evidence for the function of NO. The resistance of NOSII mice to endotoxin induced hypotension suggests that NOSII derived NO is the cause of the blood pressure reduction [4,5].

CONCLUSION

The review's objective was to give the reader a broad picture of a vast and active area of biomedical research. NO has become a well-known defense against cardiovascular disease. The general opinion indicates that illness does not significantly alter the degree of NO synthesis. The increased oxidative stress brought on by cardiovascular diseases, however, has the ability to tip the scales from NO acting as a protective to an adverse agent. Cardiovascular mortality can be reduced with therapeutic intervention that aims to maintain bio available NO by lowering oxidative stress and managing other risk factors including hypercholesterolemia and diabetes.

REFERENCES

1. Naseem KM. The role of nitric oxide in cardiovascular diseases. Mol Aspects Med. 2005;26(1-2):33-65.
2. Esch T, Stefano GB, Fricchione GL, et al. Stress in cardiovascular diseases. Signature. 2002;8(5):101.
3. Moncada S, Higgs EA. The discovery of nitric oxide and its role in vascular biology. British J Pharmacol. 2006; 147(1):193-01.
4. Loscalzo J, Welch G. Nitric oxide and its role in the cardiovascular system. Prog Cardiovasc Dis. 1995;38(2):87-104.
5. Tousoulis D, Kampoli AM, Tentolouris NPC, et al. The role of nitric oxide on endothelial function. Curr Vasc Pharmacol. 2012;10(1): 4-18.

Department of Cardiology, University of Chicago, USA

Correspondence: *Andrea Watson, Department of Cardiology, University of Chicago, USA; E-mail: watsonandrea23@gmail.com*

Received: *02-Sep-2022, Manuscript No. PULCRC-22-5309;* ***Editor assigned:*** *05-Sep2022, PreQC No. PULCRC-22-5316 (PQ);* ***Reviewed:*** *19-Sep-2022 QC No. PULCRC-22-5309;* ***Revised:*** *02-Jan-2023, Manuscript No. PULCRC-22-5316 (R);* ***Published:*** *09-Jan-2023, DOI: 10.37532/PULCRC.2023.10(1).1*

OPEN ACCESS

This open-access article is distributed under the terms of the Creative Commons Attribution Non-Commercial License (CC BY-NC) (http://creativecommons.org/licenses/by-nc/4.0/), which permits reuse, distribution and reproduction of the article, provided that the original work is properly cited and the reuse is restricted to noncommercial purposes. For commercial reuse, contact reprints@pulsus.com