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ABOUT COVER

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WJD mainly publishes articles reporting research results and findings obtained in the field of diabetes and covering a wide range of topics including risk factors for diabetes, diabetes complications, experimental diabetes mellitus, type 1 diabetes mellitus, type 2 diabetes mellitus, gestational diabetes, diabetic angiopathies, diabetic cardiomyopathies, diabetic coma, diabetic ketoacidosis, diabetic nephropathies, diabetic neuropathies, Donohue syndrome, fetal macrosomia, and prediabetic state.

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Regulatory role of peroxynitrite in advanced glycation end products mediated diabetic cardiovascular complications

Asis Bala

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Abstract

The Advanced Glycation End Products (AGE) binding with its receptor can increase reactive oxygen species (ROS) generation through specific signaling mediators. The effect of superoxide (O_2^-) and O_2^- mediated ROS and reactive nitrogen species depends on their concentration and location of formation. Nitric oxide (NO) has anti-inflammatory and anticoagulant properties and a vasodilation effect, but NO can be deactivated by reacting with O_2^- . This reaction between NO and O_2^- produces the potent oxidant ONOO $^-$. Therefore, ONOO $^-$'s regulatory role in AGEs in diabetic cardiovascular complications must be considered as a regulator of cardiovascular complications in diabetes.

Key Words: Diabetes; Cardiovascular complication; Advanced glycation end products; Reactive oxygen species; Reactive nitrogen species; Peroxynitrite

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Core Tip: The binding of Advanced Glycation End Products (AGE) to its receptor triggers the activation of signaling mediators that promote the generation of reactive oxygen species (ROS). The impact of ROS on the body can be beneficial or harmful, depending on its concentration and location. In diabetic cardiovascular complications, peroxynitrite (ONOO $^-$) plays a crucial role in vascular changes. ROS, derived from NADPH oxidase, regulates host immune responses and cellular inflammation. The production of superoxide (O_2^-), hydrogen peroxide (H_2O_2), and other compounds occurs as oxygen undergoes a series of reductions. It is essential to consider the presence of ONOO $^-$ in AGEs in diabetic cardiovascular complications.

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TO THE EDITOR

I am writing to express my appreciation for the article published by Bansal *et al*[1] in the *World Journal of Diabetes* in 2023, titled "Advanced glycation end products: Key mediator and therapeutic target of cardiovascular complications in diabetes". The article provides a clear explanation of the role of Advanced Glycation End Products (AGE) in cardiovascular complications.

I want to draw attention to the role of superoxide (O_2^-) in connection to AGE, reactive oxygen species (ROS), and reactive nitrogen species (RNS) mediated immune inflammation. The article comprehensively outlined the impact of AGE on diabetic cardiovascular disease, encompassing both cellular and extracellular pathological effects. These effects include extracellular matrix oxidation, glycation of low-density lipoprotein, and the triggering of inflammatory signaling cascades, such as NADPH oxidase, NRF-2, NF κ B, JAK, and STAT pathways. On the contrary, the article partially emphasized the significant role of Nitric oxide (NO) and NO synthase (NOS) in regulating AGE formation.

As mentioned in the article, AGE binding with its receptor increases ROS generation through stimulation of specific signaling mediators such as ERK, phospholipase A2, phosphoinositide 3-kinase activation, activation of NADPH oxidase, inducible NOS, PKC, and p38 MAPK[2]. However, the beneficial or detrimental role of O_2^- and O_2^- -mediated ROS or RNS is determined by its concentration and the places where it is formed[3]. Studies have shown that O_2^- immediately interacts with NO to produce the highly toxic peroxynitrite (ONOO $^-$), which plays a crucial role in vascular changes in diabetic cardiovascular complications[4,5].

The damage to vascular endothelial cells is a leading cause of diabetic vascular complications, which can be combated using endothelial progenitor cells (EPCs)[6]. The activation of various pathways such as xanthine and NAD(P)H oxidases, uncoupled NOS, cyclooxygenase, glucose autooxidation, the mitochondrial respiratory chain, polyol, and AGEs is triggered by hyperglycemia[4,7]. These pathways lead to the production of superoxide anion (O_2^-)[4,5]. The generation of superoxide due to hyperglycemia can also increase NO generation by enhancing the expression of NOSs by activating NF- κ B[8]. However, O_2^- can quench NO, reducing the efficacy of the endothelium-derived vasodilator system[4]. Moreover, superoxide dismutase can convert superoxide to hydrogen peroxide (H_2O_2), which can react further with NO to form ONOO $^-$ [9]. ONOO $^-$ can cause damage to cells by initiating lipid peroxidation, inactivating enzymes and proteins *via* oxidation and nitration, and activating matrix metalloproteinases[10]. Additionally, ONOO $^-$ can decrease the membrane potential by acting on mitochondria, triggering the release of proapoptotic factors such as cytochrome c and apoptosis-inducing factor[4,9,10]. These factors can mediate caspase-dependent and -independent apoptotic death pathways, which may contribute to the progression of diabetic cardiovascular complications[4]. Therefore, ONOO $^-$ is considered one of the critical modulators of diabetic cardiovascular complications since high glucose levels can impair EPC function and reduce NO production.

Furthermore, NADPH oxidase-derived ROS have become critical regulators of host immune responses and cellular inflammation[11,12]. Activation of phospholipase A2 in human neutrophils and other inflammatory cells by polyunsaturated fatty acids stimulates O_2^- production, triggering innate immune reactions. Increased O_2^- production may also activate the arachidonic acid pathways[5]. Oxygen undergoes a series of univalent reductions, sequentially producing O_2^- , H_2O_2 , etc. NO always shows its anti-inflammatory, anticoagulant properties and vasodilation effect. Still, it can be inactivated by reaction with O_2^- , producing the potent oxidant ONOO $^-$ [11-13]. Therefore, the regulatory role of ONOO $^-$ in AGEs in diabetic cardiovascular complications also needs to be considered.

FOOTNOTES

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