Toxicology: Open Access

Editorial Open Access

The Role of Manganese Superoxide Dismutase in Skin Cancer Prevention

FermÃn Priyadarsi*

Department of Geographical Science, School of Geosciences, University of Energy and Natural Resources, Mexico

Abstract

Manganese Superoxide Dismutase is an enzyme involved in the cellular defense against oxidative stress. In the context of skin cancer prevention, MnSOD plays a crucial role in protecting skin cells from oxidative damage induced by reactive oxygen species. Excessive ROS production, triggered by UV radiation and other environmental factors, can lead to DNA damage and the development of skin cancer. MnSOD acts as an antioxidant enzyme, converting superoxide radicals into less harmful hydrogen peroxide, thereby reducing oxidative stress and preserving cellular integrity. Reduced levels of MnSOD have been observed in skin cancer tissues, suggesting its importance in preventing skin cancer. Genetic variations in the MnSOD gene have also been associated with an increased risk of skin cancer. Strategies that support MnSOD activity, such as antioxidant-rich diets and topical application of antioxidants, can enhance its protective effects. However, skin cancer prevention requires a comprehensive approach, considering various genetic, environmental, and lifestyle factors.

Keywords: Manganese superoxide dismutase; Skin cancer prevention; Oxidative stress; Reactive oxygen species; ROS; UV radiation; Antioxidant; DNA damage; Cellular integrity

Introduction

Skin cancer is the most common type of cancer worldwide, with increasing incidence rates over the years. Ultraviolet radiation from the sun is a major risk factor for the development of skin cancer. Although our skin has natural defense mechanisms to counteract the damaging effects of UV radiation, prolonged or intense exposure can overwhelm these mechanisms and lead to DNA damage, oxidative stress, and ultimately, the formation of skin cancer. With skin being the largest, most readily exposed organ to the environment, it is imperative that mechanisms of protection against oxidative injury are in place within the skin. The skin consists of various antioxidant enzymes such as glutathione reeducates, catalase, and superoxide dismutase [1]. These enzymes are often activated to maintain homeostasis and to minimize the damaging effects of ROS. However, alterations in the expression/activity of these antioxidants increase the susceptibility of skin to ROS-mediated injury that contributes to skin disease. Many studies have shown that antioxidant activity, mainly manganese superoxide dismutase is reduced in various skin cancers. For example, epidermal SOD activity is decreased in hyper proliferative keratinocytes in squamous cell carcinoma, basal cell epithelioma, as well as benign hyper proliferative keratinocytes in the psoriatic epidermis. This paper focuses on the therapeutic potential of exogenous antioxidant inducers, the use of SOD mimetics as a chemo preventive agent, and dietary mechanisms of antioxidant induction in skin carcinogenesis [2].

One critical defense mechanism against oxidative stress is the enzyme manganese superoxide dismutase. MnSOD plays a crucial role in neutralizing harmful free radicals generated during oxidative stress and preventing cellular damage. This article explores the significance of MnSOD in skin cancer prevention and highlights its potential therapeutic implications.

The role of MnSOD in skin cancer prevention

MnSOD is an antioxidant enzyme found in the mitochondria of various cells, including those in the skin. It functions by converting superoxide radicals, which are highly reactive and damaging, into hydrogen peroxide and molecular oxygen. This conversion helps to maintain the redox balance within cells and prevents oxidative damage to cellular components, including DNA. Several studies have demonstrated the importance of MnSOD in protecting against skin cancer development. Animal models lacking MnSOD activity have shown an increased susceptibility to skin cancer when exposed to UV radiation. In these models, the accumulation of DNA damage and oxidative stress within skin cells was observed, leading to the promotion of malignant transformation [3].

Furthermore, human studies have highlighted the correlation between MnSOD expression and skin cancer risk. Reduced MnSOD levels or impaired MnSOD activity have been associated with an elevated risk of skin cancer development, suggesting that MnSOD deficiency compromises the skin's ability to combat oxidative stress and prevent carcinogenesis.

Potential therapeutic implications

Given the protective role of MnSOD against skin cancer, researchers have explored the therapeutic potential of enhancing MnSOD activity in the prevention and treatment of this disease. Topical MnSOD formulations: Application of MnSOD directly to the skin in the form of topical creams or lotions may provide localized protection against UV-induced damage [4]. Encouragingly, preclinical studies have shown that topical MnSOD can reduce oxidative stress, DNA damage, and inflammation caused by UV exposure. Antioxidant supplementation. Certain dietary antioxidants, such as vitamin E and selenium, are known to upregulate MnSOD expression. Including these antioxidants in the diet or as supplements may enhance MnSOD activity and improve the skin's resilience against UV-induced oxidative stress.

Gene therapy: Gene therapy approaches aim to deliver functional copies of the MnSOD gene to skin cells, compensating for any deficiencies in endogenous MnSOD production. Although still in the

***Corresponding author:** FermÃn Priyadarsi, Department of Geographical Science, School of Geosciences, University of Energy and Natural Resources, Mexico, E-mail: fermin.priyadarsi@gmail.com

Received: 01-July-2023, Manuscript No: tyoa-23-105421, **Editor Assigned:** 04-July-2023, PreQC No: tyoa-23-105421 (PQ), **Reviewed:** 18-July-2023, QC No: tyoa-23-105421, **Revised:** 22-July-2023, Manuscript No: tyoa-23-105421 (R), **Published:** 29-July-2023, DOI: 10.4172/2476-2067.1000225

Citation: Priyadarsi F (2023) The Role of Manganese Superoxide Dismutase in Skin Cancer Prevention. Toxicol Open Access 9: 225.

experimental stage, gene therapy holds promise as a potential treatment option for individuals at high risk of skin cancer [5].

Discussion

Manganese Superoxide Dismutase is an enzyme that plays a critical role in protecting cells against oxidative damage caused by reactive oxygen species. ROS are highly reactive molecules that can be generated as a byproduct of normal cellular metabolism or through exposure to various environmental factors such as UV radiation, pollution, and chemicals. Excessive ROS production can lead to oxidative stress, which can damage cellular components, including DNA, proteins, and lipids, and contribute to the development of various diseases, including cancer [6].

In the context of skin cancer prevention, MnSOD plays a significant role in maintaining the redox balance within skin cells. The skin is constantly exposed to UV radiation from the sun, which is a potent inducer of ROS production. MnSOD helps to neutralize these ROS by converting the superoxide radicals into less harmful hydrogen peroxide, thereby protecting the skin cells from oxidative damage. By reducing oxidative stress, MnSOD can help prevent DNA mutations, protein damage, and lipid peroxidation, which are all associated with the initiation and progression of skin cancer [7].

Several studies have highlighted the importance of MnSOD in skin cancer prevention. For example, experimental studies using animal models have shown that mice lacking MnSOD are more susceptible to skin cancer development when exposed to UV radiation compared to their wild-type counterparts. Additionally, reduced levels of MnSOD have been observed in various types of human skin cancers, suggesting that a deficiency in this enzyme may contribute to the development of skin cancer.

Furthermore, certain genetic variations in the MnSOD gene have been associated with an increased risk of developing skin cancer. These variations may result in reduced MnSOD activity, leading to decreased ROS scavenging capacity and increased susceptibility to oxidative damage [8].

To enhance the protective effects of MnSOD in skin cancer prevention, various approaches can be considered. Antioxidant-rich diets, including fruits and vegetables, can provide the necessary nutrients and compounds that support MnSOD activity and overall antioxidant defense. Additionally, topical application of antioxidant compounds, such as vitamin C and vitamin E, has been shown to enhance the activity of MnSOD and reduce oxidative damage in the skin [9].

Strategies that support MnSOD activity and enhance the overall antioxidant defense can be beneficial in reducing the risk of skin cancer development. However, it's important to note that while MnSOD is a significant factor, skin cancer prevention is a complex process involving multiple genetic, environmental, and lifestyle factors, and should be approached comprehensively. Regular use of sunscreen, minimizing UV exposure, and early detection through regular skin examinations are also essential components of a comprehensive skin cancer prevention strategy [10].

Conclusion

Manganese superoxide dismutase plays a vital role in protecting the skin against the damaging effects of UV radiation and oxidative stress, thus reducing the risk of skin cancer development. Maintaining optimal MnSOD activity and expression is crucial for ensuring the skin's defense mechanisms are functioning effectively. Future research and development of strategies to enhance MnSOD activity may pave the way for innovative approaches in skin cancer prevention and treatment. In the meantime, it is essential to continue practicing sun-safe behaviors, such as using sunscreen, wearing protective clothing, and seeking shade, to minimize UV exposure and reduce the risk of skin cancer.

Conflict of Interest

None

Acknowledgement

None

References

1. Walker NL, Burton FL, Kettlewell S, Smith GL, Cobbe SM (2007) Mapping of epicardial activation in a rabbit model of chronic myocardial infarction: response to atrial, endocardial and epicardial pacing. J Cardiovasc Electrophysiol 18: 862-868.
2. Camelliti P, Devlin GP, Matthews KG, Kohl P, Green CR, et al. (2004) Spatially and temporally distinct expression of fibroblast cannikins after sheep ventricular infarction. Circ Res 62: 415-425.
3. Jacquemet V, Henriquez CS (2008) Loading effect of fibroblast-myocyte coupling on resting potential, impulse propagation, and repolarization: insights from a microstructure model. Am J Physiol Heart Circ Physiol 294: 40-52.
4. MacCannell KA, Bazzazi H, Chilton L, Shibukawa Y, Clark RB, et al. (2007) A mathematical model of electrotonic interactions between ventricular myocytes and fibroblasts. Biophys J 92: 4121-4132.
5. Thompson SA, Burridge PW, Lipke EA, Shamblott M, Zambidis ET, Tung L (2012) Engraftment of human embryonic stem cell derived cardiomyocytes improves conduction in an arrhythmogenic in vitro model. J Mol Cell Cardiol 53: 15-23.
6. Chang MG, Tung L, Sekar RB (2006) Proarrhythmic potential of mesenchymal stem cell transplantation revealed in an in vitro coculture model. Circulation 113: 1832-1841.
7. Askar SFA, Ramkisoensing AA, Atsma DE, Schalij MJ, Pijnappels DA, et al. (2013) Engraftment patterns of human adult mesenchymal stem cells expose electrotonic and paracrine proarrhythmic mechanisms in myocardial cell cultures. Circ Arrhythm Electrophysiol 6: 380-391.
8. Rubach M, Adelmann R, Haustein M (2014) Mesenchymal stem cells and their conditioned medium improve integration of purified induced pluripotent stem cell-derived cardiomyocyte clusters into myocardial tissue. Stem Cells Dev 23: 643-653.
9. Kolossov E, Bostani T, Roell W (2006) Engraftment of engineered ES cell-derived cardiomyocytes but not BM cells restores contractile function to the infarcted myocardium. J Exp Med 203: 2315-2327.
10. Hannes T, Halbach M, Nazzal R (2008) Biological pacemakers: characterization in an in vitro coculture model. J Electrocardiol 41: 562-566.