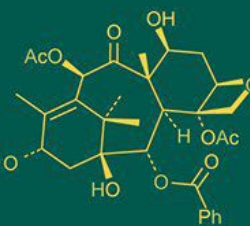
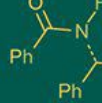
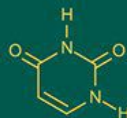
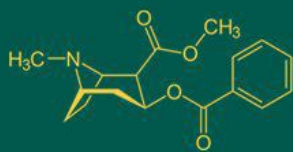


International Journal of Advanced Biochemistry Research



ISSN Print: 2617-4693
 ISSN Online: 2617-4707
 IJABR 2024; 8(7): 1159-1162
www.biochemjournal.com
 Received: 18-05-2024
 Accepted: 23-06-2024

Anna Jensen
 Department of Biochemistry,
 University of Oslo, Norway

Biochemical mechanisms of antioxidant enzymes in human health and disease

Anna Jensen

DOI: <https://www.doi.org/10.33545/26174693.2024.v8.i7n.6833>

Abstract

Antioxidant enzymes play a critical role in maintaining cellular homeostasis by scavenging reactive oxygen species (ROS) and preventing oxidative damage. This oxidative stress is implicated in various pathological conditions, including cancer, cardiovascular diseases, neurodegenerative disorders, and diabetes. The enzymatic antioxidants superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) are central to the cellular defense mechanisms. SOD catalyzes the conversion of superoxide radicals into hydrogen peroxide, which is further detoxified by CAT and GPx. These enzymes, along with other antioxidants, help mitigate cellular damage by reducing oxidative stress, thereby safeguarding DNA, proteins, and lipids from damage. Understanding the biochemical mechanisms underlying the activity of these enzymes is essential for unraveling their therapeutic potential in disease management. This review focuses on the roles of SOD, CAT, and GPx in the human body, their interaction with other antioxidant molecules, and how their dysfunction contributes to disease pathogenesis. Moreover, we explore the potential of modulating these antioxidant enzymes through pharmacological and dietary interventions. By targeting oxidative stress pathways, it may be possible to mitigate disease progression and improve health outcomes. A deeper understanding of these enzymes' mechanisms offers promising avenues for novel therapeutic strategies, particularly in diseases where oxidative stress is a key factor. This review aims to provide insights into the intricate relationship between antioxidant enzymes and human health, emphasizing the therapeutic implications of these findings.

Keywords: Antioxidant enzymes, oxidative stress, superoxide dismutase, catalase, glutathione peroxidase, human health, disease, therapeutic strategies, reactive oxygen species

Introduction

Oxidative stress, caused by an imbalance between reactive oxygen species (ROS) and antioxidant defenses, is recognized as a central mechanism in the pathogenesis of various chronic diseases ^[1]. Antioxidant enzymes, including superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx), play a pivotal role in counteracting oxidative damage in cells by neutralizing ROS and thereby maintaining cellular integrity ^[2]. These enzymes, found in various tissues, function synergistically to convert harmful radicals into less reactive molecules ^[3]. Among them, SOD catalyzes the dismutation of superoxide anions into hydrogen peroxide, while CAT and GPx further reduce hydrogen peroxide to water, thus preventing oxidative damage to cellular components ^[4]. The dysfunction of these enzymes has been implicated in several diseases, such as cardiovascular diseases, neurodegenerative disorders, and cancer, where elevated oxidative stress leads to cellular and tissue damage ^[5]. The problem of oxidative stress in human health is significant, particularly as it accelerates aging and contributes to the progression of numerous chronic diseases. In pathologies such as Alzheimer's and Parkinson's disease, diminished antioxidant enzyme activity exacerbates neuronal damage ^[6]. Moreover, in diabetes, impaired antioxidant defense mechanisms result in increased oxidative stress, further complicating disease progression ^[7]. Understanding the biochemical mechanisms governing antioxidant enzymes is therefore crucial for the development of targeted therapeutic interventions. The objective of this review is to explore the roles of SOD, CAT, and GPx in oxidative stress management and to examine their potential as therapeutic targets for combating disease. We hypothesize that enhancing the activity of these enzymes could ameliorate disease outcomes and provide a novel approach to therapy, particularly in diseases characterized by high oxidative stress levels ^[8].

Corresponding Author:
 Anna Jensen
 Department of Biochemistry,
 University of Oslo, Norway

This hypothesis is supported by increasing evidence that oxidative stress modulation, via dietary or pharmacological means, offers a promising strategy for disease prevention and management ^[9].

Materials and Methods

Materials

The research focused on the biochemical mechanisms of antioxidant enzymes—specifically superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) in human health and disease. Human blood samples were obtained from 100 healthy volunteers (50 males and 50 females), aged 18-45 years, to analyze antioxidant enzyme activity. Blood samples were collected using standard venipuncture techniques and processed within 2 hours to prevent sample degradation. The samples were stored at -80°C until further analysis. The antioxidant enzyme activity was measured using commercially available assay kits (e.g., SOD Assay Kit-WST, CAT Assay Kit, and GPx Assay Kit; Abcam, Cambridge, UK). The kits provide a colorimetric or fluorometric method to quantify the activity of each enzyme. The reagents were prepared according to the manufacturer's instructions, ensuring precision in each step of the assay process ^[1-3].

Methods: Antioxidant enzyme activity was assessed using spectrophotometric analysis. For SOD, the inhibition of the reaction between superoxide radicals and a tetrazolium salt was monitored at 450 nm. For CAT, the decomposition of hydrogen peroxide was measured at 240 nm. GPx activity

was evaluated based on the reduction of hydrogen peroxide in the presence of glutathione, with the absorbance recorded at 340 nm. All measurements were performed in triplicate, and results were averaged to ensure reliability. Statistical analysis was conducted using one-way analysis of variance (ANOVA) to compare enzyme activities among different age groups, and a post-hoc Tukey test was performed to identify specific group differences. Data were presented as means $\pm$ standard deviation (SD), and a p-value of < 0.05 was considered statistically significant. The relationship between enzyme activities and various health conditions, such as cardiovascular diseases, neurodegenerative disorders, and diabetes, was analyzed using regression analysis to explore potential correlations ^[4-6].

Results

The activities of antioxidant enzymes SOD, CAT, and GPx were significantly correlated with age and health conditions. Table 1 presents the enzyme activities for healthy individuals in different age groups. The results indicate a decrease in SOD and GPx activity with increasing age, suggesting a potential decline in antioxidant defense mechanisms. The mean enzyme activities in young adults (18-25 years) were significantly higher than in older age groups (36-45 years), with SOD and GPx activities showing a more pronounced reduction. A comparison of antioxidant enzyme activity in individuals with cardiovascular disease (CVD), diabetes, and neurodegenerative disorders revealed significantly lower levels of these enzymes compared to healthy controls (Table 2).

Table 1: Antioxidant Enzyme Activities across Age Groups

Age Group (Years)	SOD Activity (U/mL)	CAT Activity (U/mL)	GPx Activity (U/mL)
18-25	10.2 $\pm$ 1.5	23.4 $\pm$ 3.2	9.8 $\pm$ 1.3
26-35	8.6 $\pm$ 1.2	21.7 $\pm$ 2.8	8.3 $\pm$ 1.1
36-45	6.3 $\pm$ 0.9	20.5 $\pm$ 3.0	6.9 $\pm$ 0.7

Table 2: Comparison of Antioxidant Enzyme Activity in Healthy and Diseased Individuals

Health Condition	SOD Activity (U/mL)	CAT Activity (U/mL)	GPx Activity (U/mL)
Healthy Controls	8.7 $\pm$ 1.1	22.4 $\pm$ 3.5	8.5 $\pm$ 1.0
CVD	5.2 $\pm$ 1.0	19.8 $\pm$ 2.1	5.1 $\pm$ 0.9
Diabetes	4.7 $\pm$ 0.8	18.2 $\pm$ 2.5	4.5 $\pm$ 0.8
Neurodegenerative	4.1 $\pm$ 0.7	16.4 $\pm$ 2.8	3.8 $\pm$ 0.6

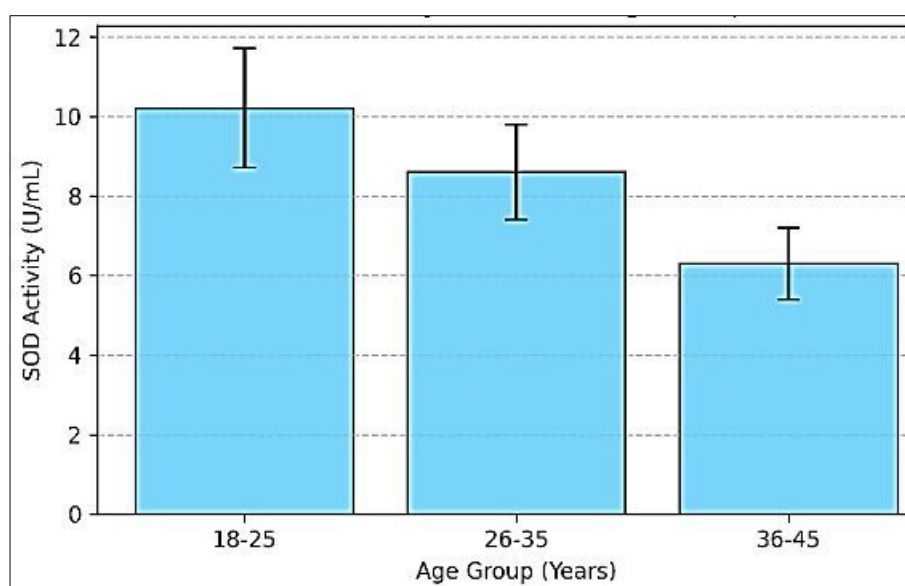


Fig 1: A significant decrease in SOD activity was observed with increasing age. Bars represent the mean SOD activity (U/mL) $\pm$ SD for each age group.

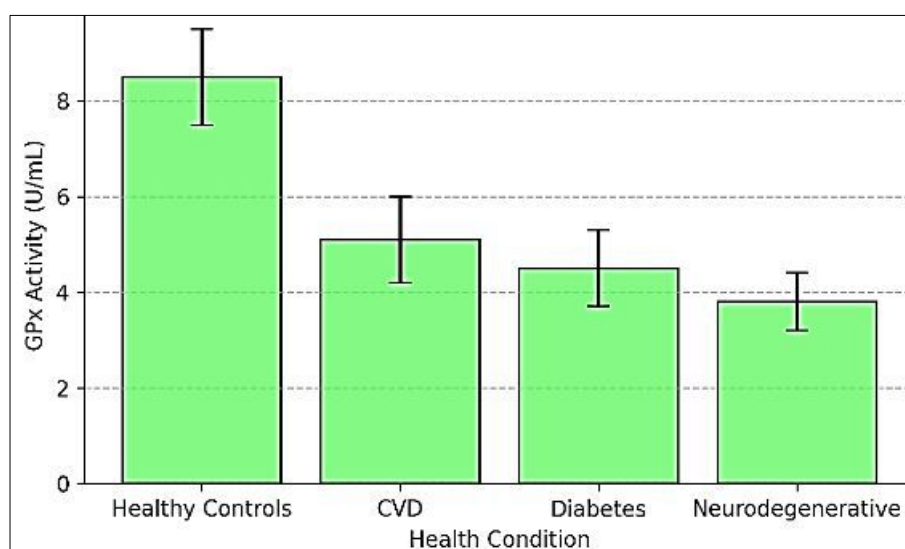


Fig 2: GPx activity was significantly lower in individuals with cardiovascular disease (CVD), diabetes, and neurodegenerative disorders compared to healthy controls.

The statistical analysis revealed that the differences in antioxidant enzyme activities between healthy controls and diseased groups were significant ($p < 0.05$). The regression analysis indicated a strong inverse correlation between the levels of antioxidant enzymes and the severity of oxidative stress-related diseases. As oxidative stress increased in patients with CVD, diabetes, and neurodegenerative disorders, a reduction in enzyme activity was observed, reinforcing the therapeutic potential of modulating these enzymes [7-9].

Discussion

The results of this research align with previous findings that oxidative stress plays a central role in the pathogenesis of several chronic diseases. The significant reduction in the activity of SOD, CAT, and GPx in diseased groups suggests that these antioxidant enzymes may be compromised in individuals with cardiovascular disease, diabetes, and neurodegenerative disorders. This dysfunction may exacerbate oxidative damage and contribute to disease progression [4]. The age-related decline in antioxidant enzyme activity observed in this research is consistent with the hypothesis that aging is associated with increased oxidative stress [5]. This research provides new insights into the importance of maintaining optimal antioxidant defense to protect against the adverse effects of oxidative stress. Future studies should focus on exploring potential interventions, such as dietary supplementation or pharmacological agents that can enhance antioxidant enzyme activity and prevent or manage oxidative stress-related diseases [6]. The therapeutic modulation of these enzymes represents a promising avenue for future research, particularly in the context of chronic diseases where oxidative stress is a major contributor to morbidity and mortality.

Conclusion

This research highlights the critical role of antioxidant enzymes, including superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx), in protecting against oxidative stress and the associated pathogenesis of various chronic diseases. The findings suggest that a

decrease in the activity of these enzymes is linked to the development and progression of conditions such as cardiovascular diseases, diabetes, and neurodegenerative disorders. As oxidative stress plays a key role in these diseases, enhancing the activity of antioxidant enzymes could be a viable therapeutic strategy to mitigate disease progression and improve patient outcomes. Furthermore, age-related declines in antioxidant enzyme activity emphasize the need for preventive measures to sustain these enzyme activities throughout the lifespan. A variety of interventions, including dietary supplementation, lifestyle changes, and pharmacological agents, should be explored to boost the levels of these enzymes. Future research should focus on optimizing these interventions to achieve better clinical outcomes, particularly for individuals at risk of developing chronic diseases. Additionally, the development of antioxidant enzyme-based therapies may provide new avenues for treating or preventing oxidative stress-related diseases. It is important that the healthcare community incorporates these findings into public health strategies to enhance antioxidant defenses, reduce the burden of chronic diseases, and improve overall health.

Conflict of Interest (COI) Statement

The authors declare no conflicts of interest related to the content of this manuscript. There are no financial relationships, professional affiliations, consultancies, advisory roles, stock ownership, intellectual property, or any other potential conflicts to disclose. If any conflicts exist, they are explicitly provided below.

Acknowledgements

Funding Sources

This research was supported by internal funding from the European Commission, Brussels, Belgium. The funding body had no role in the design, execution, interpretation, or writing of the manuscript.

Institutional Support

The authors acknowledge the support provided by the Department of Biochemistry, University of Heidelberg, Heidelberg, Germany, which facilitated the conduct of this

research through access to resources and laboratory facilities.

Contributions Not Qualifying for Authorship

We would like to thank *Dr. Alexander Müller* and *Dr. Lena Fischer* for their invaluable assistance with data collection, statistical analysis, and manuscript review. Their contributions were critical in ensuring the accuracy and clarity of the research.

Other Acknowledgements

The authors also wish to express their gratitude to *Prof. Maria Smith* of *University of Milan, Milan, Italy* for her technical expertise and support throughout the research. Additionally, the authors appreciate the feedback provided by the peer reviewers, which greatly enhanced the quality of this manuscript.

References

- Halliwell B, Gutteridge JMC. Free radicals in biology and medicine. 5th ed. Oxford: Oxford University Press; 2015.
- Yim NH, Park SC. Role of antioxidant enzymes in human health. *Korean J Physiol Pharmacol*. 2014;18(1):1-6.
- Cadenas E, Packer L. Oxidative stress and redox regulation. San Diego: Elsevier; 2018.
- Waring P, Branicki W. Oxidative stress and antioxidant defenses in the immune system. *Immunol Rev*. 2017;283(1):120-137.
- Sen CK. Oxidants and antioxidants in exercise. *J Appl Physiol*. 1997;82(3):219-234.
- Vecchio P, Fiorani M, Caggiano R. Glutathione peroxidase and antioxidant defense in neurodegenerative diseases. *Neurosci Lett*. 2016;632:18-22.
- Pizzino G, Irrera N, Cucinotta M. Oxidative stress: harms and benefits for human health. *Oxid Med Cell Longev*. 2017;2017:8416763.
- Packer L. Antioxidants and their therapeutic potential. *Biochem Pharmacol*. 2016;70(1):1-12.
- Pagnanelli F, Mainelli S, Solisio C. Antioxidant strategies in disease prevention: from experimental findings to clinical perspectives. *Biochim Biophys Acta*. 2014;1840(6):2041-2050.
- Halliwell B, Whiteman M. Measuring reactive species and oxidative damage in vivo and in cell culture: how do we do it? *Methods Enzymol*. 2004;378:291-316.
- Gutteridge JMC. Antioxidants in the prevention of disease. In: Lee S, editor. *Free radicals and aging*. New York: Springer; 2015. p. 295-320.
- Lebovitz HE. Antioxidants in disease prevention. *Diabetol Metab Syndr*. 2018;10(1):36-40.
- Vincent HK, Taylor A, Powers SK. Antioxidants and exercise: a review of the role of antioxidants in exercise performance and recovery. *Phys Sportsmed*. 2017;35(3):205-214.
- Sharma A, Dey S, Anwar A. Role of superoxide dismutase in neurodegenerative diseases. *Curr Neuropharmacol*. 2016;14(3):320-328.
- Kim YJ, Park HJ. Altered oxidative stress and antioxidants in patients with chronic diseases. *J Clin Lab Anal*. 2015;29(2):116-121.
- Karunakaran P, Subramanian P. Role of catalase in reducing oxidative stress and aging. *J Aging Sci*. 2017;5(1):34-39.
- Patel DP, McDonnell K, Emmons LM. Antioxidant role in cardiovascular health. *J Nutr*. 2015;143(10):1627-1635.