e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2025; 17(8); 1108-1111

Original Research Article

Alterations in Antioxidant Enzyme Activity across Age Groups: A Biochemical Evaluation

Atul Manohar Shinde

Assistant Professor, Department of Biochemistry, Parbhani Medical College Parbhani, RP hospital and research institute

Received: 11-07-2025 Revised: 16-07-2025 Accepted: -24-7-2025
Corresponding author: Dr. Atul Manohar Shinde
Conflict of interest: Nil

Abstract

Background: Aging is linked to elevated oxidative stress, which arises from an imbalance between the production of reactive oxygen species (ROS) and the body's antioxidant defense mechanisms. Key antioxidant enzymes, including superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx), are crucial for preserving redox balance. However, their activity varies across age groups, contributing to age-related functional decline.

Material and Methods: A cross-sectional observational study was conducted on 180 healthy individuals stratified into three age groups: Group I (18–30 years), Group II (31–50 years), and Group III (51–70 years), with 60 participants in each. Fasting venous blood samples were analyzed for erythrocyte SOD, CAT, and GPx activities using standard spectrophotometric methods. Inclusion criteria comprised apparently healthy individuals without chronic illness, smoking, or alcohol consumption. Subjects with diabetes, cardiovascular disease, or antioxidant supplementation were excluded. Statistical analysis was performed using ANOVA with $p < 0.05$ considered significant.

Results: A significant decline in antioxidant enzyme activity was observed with advancing age. Mean SOD activity was highest in Group I and lowest in Group III ($p < 0.001$). CAT activity showed a gradual reduction from Group I to Group III ($p < 0.01$). GPx activity followed a similar decreasing trend ($p < 0.05$).

Conclusion: The study demonstrates a significant age-associated decline in major antioxidant enzymes, indicating a progressive reduction in endogenous defense against oxidative stress. These findings highlight the importance of dietary and lifestyle interventions to enhance antioxidant capacity in older populations.

Keywords: Aging, Oxidative stress, Antioxidant enzymes, Superoxide dismutase, Catalase and Glutathione peroxidase.

Introduction

Aging is a complex biological process characterized by progressive functional decline, increased vulnerability to diseases, and eventual mortality. Among several theories proposed, the free radical theory of aging, first introduced by Harman in 1956, has gained significant attention [1]. It postulates that the accumulation of reactive oxygen species (ROS) over time leads to oxidative damage of biomolecules, contributing to aging and age-related diseases.

ROS are continuously generated as by-products of cellular metabolism, particularly within the mitochondria. To counteract oxidative stress, cells possess an intricate antioxidant defense system comprising enzymatic and non-enzymatic components [2]. Among the enzymatic antioxidants, superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) represent the primary line of defense. SOD catalyzes the dismutation of superoxide radicals into hydrogen peroxide, which is subsequently degraded by CAT and GPx [3]. With advancing age, several studies have reported alterations in antioxidant enzyme activity, leading to increased oxidative damage and cellular dysfunction [4,5]. Reduced activity of these enzymes has been implicated in the pathophysiology of neurodegenerative disorders, cardiovascular diseases, diabetes, and cancer [6,7].

Thus, evaluating age-associated changes in antioxidant enzyme activity is crucial for understanding the biochemical underpinnings of aging and for designing potential preventive strategies. Previous investigations have yielded conflicting results, with some studies reporting a decline in enzymatic activity with age, while others observed compensatory increases in certain

enzymes [8,9]. This discrepancy may be attributed to differences in study populations, methodologies, and environmental factors. Therefore, further evaluation in diverse populations is warranted.

The present study aimed to evaluate alterations in antioxidant enzyme activities across different age groups in an apparently healthy Indian population. By examining the activities of SOD, CAT, and GPx in young, middle-aged, and older adults, this study sought to provide insights into the biochemical basis of age-related oxidative stress.

Aim and Objectives

Aim: To evaluate alterations in antioxidant enzyme activity across different age groups.

Objectives:

1. To measure the activities of superoxide dismutase, catalase, and glutathione peroxidase in healthy individuals of varying age groups.
2. To compare age-associated variations in these enzyme activities statistically.

Materials and Methods

This cross-sectional observational study was conducted in the Department of Biochemistry of a tertiary care medical institution. A total of 180 healthy volunteers aged 18–70 years were recruited after obtaining informed consent. The participants were divided into three groups: Group I (18–30 years), Group II (31–50 years), and Group III (51–70 years), with 60 subjects in each.

Inclusion criteria: Apparently healthy individuals with no history of chronic illness, non-smokers, non-alcoholic, and not on antioxidant supplements.

Exclusion criteria: Patients with diabetes mellitus, cardiovascular disease, chronic infections, liver or renal dysfunction, malignancy, or those on regular medications affecting oxidative metabolism.

Fasting venous blood samples were collected. Plasma and erythrocyte lysates were separated for biochemical analysis. SOD activity was measured by the inhibition of pyrogallol autoxidation, CAT activity by hydrogen peroxide decomposition, and GPx activity using the NADPH oxidation method. All assays were performed spectrophotometrically under standardized conditions.Data were analyzed using SPSS v25.0. Results were expressed as mean ± SD. One-way ANOVA followed by post hoc Tukey's test was applied to compare enzyme activities among groups.

A p-value <0.05 was considered statistically significant.

Results

Table 1: Mean Superoxide Dismutase (SOD) Activity across Age Groups

Age Group	n	SOD Activity (U/mg Hb, Mean ± SD)	p-value
Group I (18–30 yrs)	60	12.5 ± 1.8	<0.01
Group II (31–50 yrs)	60	10.9 ± 1.6	
Group III (51–70 yrs)	60	8.7 ± 1.5	

A significant decline in SOD activity with increasing age.

Table 2: Mean Catalase (CAT) Activity across Age Groups

Age Group	n	CAT Activity (kU/L, Mean ± SD)	p-value
Group I (18–30 yrs)	60	85.2 ± 10.5	<0.01
Group II (31–50 yrs)	60	77.6 ± 9.8	
Group III (51–70 yrs)	60	69.3 ± 8.7	

CAT activity shows a gradual decrease with age.

Table 3: Mean Glutathione Peroxidase (GPx) Activity across Age Groups

Age Group	n	GPx Activity (U/g Hb, Mean ± SD)	p-value
Group I (18–30 yrs)	60	45.8 ± 6.3	<0.05
Group II (31–50 yrs)	60	41.7 ± 5.9	
Group III (51–70 yrs)	60	37.2 ± 5.4	

Interpretation: GPx activity also declines progressively with age.

Discussion

The present study demonstrated a significant age-associated decline in antioxidant enzyme activities, including SOD, CAT, and GPx, among apparently healthy individuals. These findings are consistent with the free radical theory of aging, which suggests that oxidative stress increases with advancing age due to diminished antioxidant defenses [1]. SOD is considered the first line of enzymatic defense against superoxide radicals. Our study observed a marked reduction in SOD activity from young adults to elderly individuals, aligning with previous reports [10,11]. This decline may be attributed to cumulative oxidative damage to mitochondrial proteins and decreased gene expression of antioxidant enzymes in aging tissues [12]. Reduced SOD activity has been associated

with increased susceptibility to neurodegenerative conditions such as Alzheimer's and Parkinson's disease [13].

Catalase, located primarily in peroxisomes, decomposes hydrogen peroxide into water and oxygen, thereby preventing hydroxyl radical formation. Our results revealed a gradual decline in CAT activity across age groups, corroborating earlier findings [14]. Reduced catalase levels may exacerbate oxidative damage to DNA and lipids, predisposing to age-related pathologies including atherosclerosis and metabolic disorders [15].

GPx, a selenium-dependent enzyme, plays a crucial role in detoxifying hydrogen peroxide and lipid peroxides. The significant reduction in GPx activity observed in older individuals in this study is consistent with reports by others [16,17]. Lower GPx activity may result from decreased selenium bioavailability with age or oxidative inactivation of the enzyme [18]. Impaired GPx function has been linked to cardiovascular morbidity and cancer progression [19].

Interestingly, some studies have reported compensatory increases in certain antioxidant enzymes with age, possibly reflecting adaptive mechanisms against heightened oxidative stress [20]. However, in our population, the overall trend was a uniform decline across all three enzymes, suggesting an inadequate adaptive response. This could be influenced by genetic factors, dietary patterns, or environmental exposures unique to the studied population.

The implications of our findings are clinically relevant. Reduced antioxidant enzyme activity may partly explain the higher incidence of chronic, degenerative diseases in elderly populations. Dietary interventions rich in antioxidants, physical activity, and supplementation with micronutrients such as selenium and vitamins C and E may help strengthen endogenous defense systems [21,22]. Furthermore, pharmacological agents aimed at upregulating endogenous antioxidants or mimicking their activity are being explored as potential anti-aging strategies [23].

Limitations: Being a cross-sectional study, causal relationships cannot be inferred. Lifestyle factors and dietary intake were not quantitatively assessed. Moreover, only erythrocyte antioxidant enzyme activity was measured; tissue-specific changes may vary.

Future directions: Longitudinal studies with larger cohorts, incorporating non-enzymatic antioxidants and oxidative stress biomarkers, are warranted to provide a comprehensive understanding of redox alterations in aging.

In conclusion, the present study reinforces the role of antioxidant enzyme decline as a biochemical hallmark of aging and highlights the potential of antioxidant-based interventions in promoting healthy aging.

Conclusion

This study evaluated alterations in antioxidant enzyme activities across different age groups and demonstrated a significant age-associated decline in SOD, CAT, and GPx activities. The progressive reduction in these enzymes underscores a weakening endogenous antioxidant defense with aging, leading to increased oxidative stress and susceptibility to chronic diseases. These findings support the free radical theory of aging and emphasize the importance of early lifestyle modifications to maintain redox balance.

Nutritional strategies rich in natural antioxidants, regular exercise, and avoidance of oxidative stressors such as smoking and excessive alcohol may play crucial roles in preserving antioxidant capacity in older individuals. From a clinical perspective, monitoring antioxidant enzyme levels could serve as a valuable biomarker for assessing aging-related oxidative stress and guiding preventive strategies.

Future research should focus on longitudinal assessments and the interplay of genetic and environmental factors influencing antioxidant defense. In addition, exploring therapeutic interventions to restore antioxidant capacity may offer new avenues in geriatric healthcare and longevity research.

Thus, enhancing antioxidant defenses represents a promising strategy to mitigate the deleterious effects of aging and promote healthy aging trajectories.

References

1. Harman D. Aging: a theory based on free radical and radiation chemistry. J Gerontol. 1956 Jul; 11(3):298–300. PMID: 13332224
2. Liguori I, Russo G, Curcio F, Bulli G, Aran L, Della-Morte D, et al. Oxidative stress, aging, and diseases. Clin Interv Aging. 2018; 13:757–72. PMID: 29731617
3. Fridovich I. Superoxide dismutases. An adaptation to a paramagnetic gas. J Biol Chem. 1989 Nov 25; 264(14):7761–4. PMID: 2542245
4. Sohal RS, Orr WC. The redox stress hypothesis of aging. Free Radic Biol Med. 2012 Dec; 52(3):539–55. PMID: 22080087
5. Pérez VI, Bokov A, Van Remmen H, Mele J, Ran Q, Ikeno Y, et al. Is the oxidative stress th eory of aging dead? Biochim Biophys Acta. 20 09 Oct; 1790(10):1005–14. PMID: 1952401 6

6. Uttara B, Singh AV, Zamboni P, Mahajan RT. Oxidative stress and neurodegenerative diseases. Int J Neurosci. 2009; 19(1):37–49. PMID: 19125387
7. Madamanchi NR, Runge MS. Mitochondrial dysfunction in atherosclerosis. Circ Res. 2007 Nov; 100(4):460–73. PMID: 17332437
8. Mecocci P, Fano G, Fulle S, MacGarvey U, Shinobu L, Polidori MC, et al. Age-dependent increases in oxidative damage to DNA, lipids, and proteins in human skeletal muscle. Free Radic Biol Med. 1999 Jan; 26(3-4):303–8. PMID: 9895220
9. Pawelec G, Goldeck D, Derhovanessian E. Inflammation, ageing and chronic disease. Curr Opin Immunol. 2014 Aug; 29:23–8. PMID: 24934645
10. Choi J, Levey AI, Weintraub ST, Rees HD, Gearing M, Chin LS, Li L. Oxidative modifications and down-regulation of ubiquitin carboxyl-terminal hydrolase L1 associated with idiopathic Parkinson's and Alzheimer's diseases. J Biol Chem. 2004 Mar 26; 279(13):13256-64. doi: 10.1074/jbc. M314124200. Epub 2004 Jan 13. PMID: 14722078.
11. Rao AV, Balachandran B. Role of oxidative stress and antioxidants in neurodegenerative diseases. Nutr Neurosci. 2002; 5(5):291–309. PMID: 12495588
12. Finkel T, Holbrook NJ. Oxidants, oxidative stress and the biology of ageing. Nature. 2000 Nov; 408(6809):239–47. PMID: 11089981
13. Andersen JK. Oxidative stress in neurodegeneration: cause or consequence? Nat Med. 2004 Jul; 10 Suppl: S18–25. PMID: 15298006
14. Gaweł S, Wardas M, Niedworok E, Wardas P. Malondialdehyde (MDA) as a lipid peroxidation marker in neurological disorders. Acta Neurobiol Exp. 2004; 64(4):213–9. PMID: 15794493
15. Valko M, Leibfritz D, Moncol J, Cronin MTD, Mazur M, Telser J. Free radicals and antioxidants in normal physiological functions and human disease. Int J Biochem Cell Biol. 2007 Jan; 39(1):44–84. PMID: 16978905
16. Brigelius-Flohé R, Maiorino M. Glutathione peroxidases. Biochim Biophys Acta. 2013 May; 1830(5):3289–303. PMID: 23201771
17. Rayman MP. Selenium and human health. Lancet. 2012 Mar; 379(9822):1256–68. PMID: 22381456
18. Tapiero H, Townsend DM, Tew KD. The antioxidant role of selenium and seleno-compounds. Biomed Pharmacother. 2003 Nov; 57(3-4):134–44. PMID: 12818475
19. Lubos E, Loscalzo J, Handy DE. Glutathione peroxidase-1 in health and disease: from molecular mechanisms to therapeutic opportunities. Antioxid Redox Signal. 2011; 15(7):1957–97. PMID: 21087145
20. Dröge W. Free radicals in the physiological control of cell function. Physiol Rev. 2002 Jan; 82(1):47–95. PMID: 11773609
21. Halliwell B, Gutteridge JMC. Free Radicals in Biology and Medicine. 5th ed. Oxford: Oxford University Press; 2015. PMID: 25651114
22. Liochev SI. Reactive oxygen species and the free radical theory of aging. Free Radic Biol Med. 2013; 60:1–4. PMID: 23434764
23. McCord JM, Edeas MA. SOD, oxidative stress and human pathologies: a brief history and a future vision. Biomed Pharmacother. 2005 Nov; 59(4):139–42. PMID: 16275486.