International Journal of Scientific Advances
ISSN: 2708-7972
Volume: 6 | Issue: 6 | Nov – Dec 2025 Available Online: www.ijscia.com
DOI: 10.51542/ijscia.v6i6.16

Effect of Combined Resistance and Endurance Exercise Training on Oxidative Stress in Individuals with Obesity: A Literature Review

I Ketut Bagus Sajjana Vandhana Pamecut[1], Purwo Sri Rejeki[2*]

[1]Medical Study Program, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia
[2]Physiology Division, Department of Medical Physiology and Biochemistry, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia

E-mail: i.ketut.bagus.sajjana-2022@fk.unair.ac.id; purwo-s-r@fk.unair.ac.id

*Corresponding author details: Purwo Sri Rejeki; purwo-s-r@fk.unair.ac.id

ABSTRACT

Obesity is a complex metabolic disorder characterized by excessive adiposity, chronic low-grade inflammation, and increased oxidative stress. These processes contribute to insulin resistance, endothelial dysfunction, and metabolic comorbidities. Training is a non-pharmacological intervention, with endurance training promoting mitochondrial function and cardiovascular health, and resistance training supporting gains in skeletal muscle mass, strength, and glucose metabolism. Recent studies indicate that combined resistance and endurance training may produce synergistic benefits by enhancing antioxidant defenses, reducing reactive oxygen species (ROS), and improving metabolic regulation. This narrative review synthesizes current evidence on the effects of combined training on oxidative stress in individuals with obesity, examining molecular mechanisms, biomarkers of oxidative damage and antioxidant capacity, and implications for metabolic health. The review highlights combined training as an effective non-pharmacological strategy to mitigate obesity-associated oxidative stress, restore redox balance, and improve overall metabolic outcomes.

Keywords: combined training; obesity; oxidative stress; reactive oxygen species; antioxidant defenses; literature review

INTRODUCTION

Obesity is a multifaceted metabolic condition marked by excessive accumulation of adipose tissue [1] and is frequently accompanied by chronic low-grade inflammation and oxidative stress [2]. Reactive oxygen species (ROS) overproduction combined with impaired antioxidant defenses leads to cellular damage, insulin resistance, and endothelial dysfunction [3,4,16]. These disruptions also contribute to the emergence of comorbidities, such as heart disease and type 2 diabetes [5].

Training is a fundamental component in the management of obesity [6]. Endurance training enhances mitochondrial function and cardiovascular performance [7], whereas resistance Training promotes gains in skeletal muscle mass, muscular strength, and glucose regulation [8,9]. Emerging evidence indicates that combining resistance and endurance training may produce synergistic benefits for metabolic health and oxidative balance [10,11]. Integrating these training modalities can potentially strengthen antioxidant defences, reduce oxidative damage, improve body composition, and optimize metabolic control [10,12].

This review aims to consolidate current research regarding the impact of combined resistance and endurance training on oxidative stress in individuals with obesity. It explores the effects of combined training on regulating ROS levels, strengthening antioxidant capacity, and enhancing metabolic functions, including insulin sensitivity, endothelial health, and body composition. By summarizing these findings, the review underscores the potential of combined training as an effective strategy to counteract oxidative stress and obesity-associated complications.

REVIEW CONTENT

A. Obesity

A body mass index (BMI) of 30 kg/m^2 or over is considered obese, whereas a BMI of 25 to 29.9 kg/m^2 is considered overweight [13]. A rise in body weight is not the only sign of excess adipose tissue. It increases the risk of heart disease, type 2 diabetes, metabolic syndrome, and some types of cancer by creating a significant metabolic burden [14]. In addition to storing energy, adipose tissue is an endocrine organ that produces adipokines and inflammatory cytokines that promote systemic oxidative stress and persistent low-grade inflammation [15]. These metabolic disruptions form the basis for the pathophysiology underlying obesity-related complications [1].

B. Oxidative Stress

Reactive oxygen species (ROS) damage cells and impair

physiological function when the body's antioxidant defenses cannot keep up with their production. This condition is known as oxidative stress. Usually generated during metabolic activities, ROS are crucial for cell communication and homeostasis, especially in mitochondria [43]. Environmental pollutants, poor nutrition, age, smoking, and high metabolic activity can all lead to excessive ROS generation. Superoxide dismutase (SOD), catalase, glutathione peroxidase (GPx), and non-enzymatic antioxidants like vitamins C and E can all be overpowered by this overproduction. This imbalance can damage lipids, proteins, and DNA, promoting inflammation, cellular dysfunction, and the development of various chronic diseases [45].

Biomarkers that represent both oxidative damage and the body's antioxidant defenses are frequently used to evaluate oxidative stress. Protein carbonyl content is used to quantify protein oxidation, while malondialdehyde (MDA) and thiobarbituric acid reactive substances (TBARS) are commonly employed to detect lipid peroxidation, which indicates damage to cell membranes (18,20). While non-enzymatic antioxidants and total antioxidant capacity (TAC) offer a thorough assessment of overall redox status, enzymatic antioxidants, such as glutathione peroxidase (GPx), catalase, and superoxide dismutase (SOD), offer insight into the body's natural defense mechanisms [2,21]. These biomarkers are widely employed to investigate the effects of interventions, such as dietary changes, pharmacological treatments, and physical training, on oxidative stress. In particular, they are used in obese populations to assess how training modulates R OS production, enhances antioxidant capacity, and improves metabolic and cardiovascular health outcomes [22,23].

C. Obesity and Oxidative Stress

Obesity is strongly connected to increased oxidative stress, which results from an imbalance between the body's antioxidant defense and the generation of reactive oxygen species (ROS) [2]. There are various processes that contribute to this pro-oxidative situation. Mitochondrial malfunction, caused by nutritional excess, leads to electron transport chain overload and increased ROS production [17]. Chronic inflammation in adipose tissue, triggered by macrophage infiltration, exacerbates ROS generation. Cellular oxidative damage is exacerbated by deficits in endogenous antioxidant systems such as catalase, glutathione peroxidase (GPx), and superoxide dismutase (SOD) [2,18]. This increased oxidative stress causes lipid peroxidation, protein oxidation, DNA damage, and endothelial dysfunction, which all contribute to insulin resistance and the development of obesity-related comorbidities [19,3].

D. Effect of Endurance Training on Oxidative Stress in Obesity

Walking, cycling, and running are examples of continuous, rhythmic motions of large muscle groups that improve cardiovascular health and overall metabolic performance [24,25]. At the cellular level, it activates mitochondrial biogenesis via the PGC-1α pathway, increasing mitochondrial density and enhancing oxidative phosphorylation efficiency [26]. This adaptation lowers the production of reactive oxygen species (ROS) per unit of ATP generated by reducing electron leakage from the electron transport chain [27]. Additionally, endurance training increases the activity of enzymatic antioxidant systems such as superoxide dismutase (SOD), glutathione peroxidase (GPx), and catalase [28]. Reactive oxygen species (ROS) are briefly produced during endurance training due to increased oxygen consumption in the mitochondria. The nuclear factor erythroid 2–2-related factor 2 (Nrf2) pathway, which stimulates the transcription of genes encoding important antioxidant enzymes like superoxide dismutase (SOD), glutathione peroxidase (GPx), and catalase, is one of the cellular signaling pathways activated by this controlled oxidative challenge [46]. Furthermore, endurance exercise enhances mitochondrial biogenesis and improves oxidative phosphorylation efficiency by upregulating peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC-1α). This further reduces ROS leakage per unit of ATP generated. Over time, these molecular adaptations lead to elevated enzymatic antioxidant activity, strengthening redox homeostasis and protecting cells from oxidative damage [47].

Beyond molecular effects, endurance training improves tissue oxygenation, skeletal muscle glucose uptake, and insulin sensitivity [29,30]. Together, these adaptations reduce mitochondrial substrate overload and limit ROS production [31]. By lowering pro-inflammatory cytokines, including TNF-α and IL-6, it further reduces systemic inflammation [32]. Chronic endurance training has been demonstrated to improve metabolic outcomes and reduce oxidative stress in obese persons by raising total antioxidant capacity (TAC) and lowering oxidative damage indicators like malondialdehyde (MDA) and TBARS [33, 34].

E. Effect of Resistance Training on Oxidative Stress in Obesity

Resistance training, such as weightlifting or bodyweight training, primarily promotes skeletal muscle growth and strength [35]. The increase in muscle mass improves glucose utilization and insulin sensitivity, thereby decreasing mitochondrial substrate overload and limiting reactive oxygen species (ROS) production [36]. Moreover, resistance training boosts the activity of antioxidant enzymes, including catalase, glutathione peroxidase (GPx), and superoxide dismutase (SOD), which work together to eliminate reactive oxygen species (ROS) and protect cellular components from oxidative damage [37].

Resistance training stimulates enzymatic antioxidant defenses through repeated metabolic and mechanical stress on skeletal muscles. Reactive oxygen species (ROS) generation is elevated during resistance exercise due to the increased energy demand. ROS serves as a physiological signal to activate cellular defense pathways. One such mechanism is the nuclear

factor erythroid 2–related factor 2 (Nrf2) pathway, which upregulates genes encoding antioxidant enzymes, including catalase, glutathione peroxidase (GPx), and superoxide dismutase (SOD), and translocates to the nucleus in response to oxidative stress [48]. Additionally, resistance training induces adaptations in mitochondrial function and density, improving oxidative phosphorylation efficiency and reducing ROS leakage per unit of ATP. Mechanical stress from muscle contraction also activates signaling pathways associated with hypertrophy, including MAPK and AMPK, which can indirectly enhance antioxidant enzyme expression. Over time, these adaptations collectively increase enzymatic antioxidant activity, strengthen redox homeostasis, and protect muscle and other tissues from oxidative damage [49].

In addition to molecular adaptations, resistance training enhances musculoskeletal health, metabolic function, and overall physical capacity, which are particularly advantageous for individuals with obesity [38]. Research has shown that long-term resistance training lowers oxidative damage markers, such as malondialdehyde (MDA), while increasing total antioxidant capacity (TAC) [39,40]. By reducing oxidative stress and improving metabolic regulation, resistance training helps prevent obesity-related complications and complements endurance training when performed in combination [41].

F. Effect of Combined Resistance and Endurance on Oxidative Stress in Obesity

Combined training incorporates both endurance and resistance training within a single program, aiming to leverage the complementary benefits of each modality. This approach simultaneously enhances cardiovascular fitness, skeletal muscle mass, and metabolic function, providing a comprehensive strategy for obesity management [42]. Physiologically, combined training exerts synergistic effects by improving mitochondrial efficiency and antioxidant defenses through endurance training (26,28), while promoting muscle hypertrophy and metabolic capacity via resistance training [35,36]. Studies indicate that this dual approach is more effective than single-mode training in modulating oxidative stress, reducing lipid peroxidation, increasing enzymatic antioxidant activity, and enhancing total antioxidant capacity (TAC), underscoring its potential to improve overall metabolic health in obese individuals [41].

CONCLUSION

In conclusion, through a variety of mechanisms, combined resistance and endurance exercise successfully reduces oxidative stress in obese people. Chronic ROS overproduction, mitochondrial dysfunction, weakened antioxidant defences, and inflammation mediated by adipose tissue are all facilitated by obesity and result in DNA damage, lipid peroxidation, protein oxidation, and metabolic dysregulation. Combined training leverages the complementary benefits of endurance and resistance training: endurance training enhances mitochondrial efficiency and upregulates enzymatic and non-enzymatic antioxidant systems, while resistance training increases skeletal muscle mass, improves glucose uptake, and strengthens endogenous antioxidant defenses. Together, these adaptations reduce oxidative damage markers, such as malondialdehyde (MDA), and improve total antioxidant capacity (TAC), thereby restoring redox balance and improving metabolic function. These findings highlight combined training as a highly effective non-pharmacological intervention for alleviating obesity-associated oxidative stress and its related metabolic complications.

ACKNOWLEDGES

The authors wish to convey their deepest gratitude to all individuals and institutions who contributed to the completion of this literature review titled Effect of Combined Resistance and Endurance Exercise Training on Oxidative Stress in Individuals with Obesity. We extend our sincere appreciation to our mentors and academic supervisors for their invaluable guidance, critical insights, and constructive feedback throughout the development of this manuscript. We also acknowledge the contributions of researchers, exercise scientists, and healthcare professionals whose persistent efforts in advancing the understanding of exercise physiology, obesity management, and oxidative stress have provided a strong foundation for this work. Their dedication to evidence-based practice and scientific progress has greatly supported the quality and relevance of this review.

REFERENCES

[1] Jung, U. J., & Choi, M. S. (2014). Obesity and its metabolic complications: the role of adipokines and the relationship between obesity, inflammation, insulin resistance, dyslipidemia and nonalcoholic fatty liver disease. International Journal of Molecular Sciences, 15(4), 6184–6223.

[2] Fernández-Sánchez, A., Madrigal-Santillán, E., Bautista, M., Esquivel-Soto, J., Morales-González, A., Esquivel-Chirino, C., Durante-Montiel, I., Sánchez-Rivera, G., Valadez-Vega, C., & Morales-González, J. A. (2011). Inflammation, oxidative stress, and obesity. International Journal of Molecular Sciences, 12(5), 3117–3132.

[3] Masenga, S. K., Kabwe, L. S., Chakulya, M., & Kirabo, A. (2023). Mechanisms of oxidative stress in metabolic syndrome. International Journal of Molecular Sciences, 24(9), 7898.

[4] Prieto, D., Contreras, C., & Sánchez, A. (2014). Endothelial dysfunction, obesity and insulin resistance. Current Vascular Pharmacology, 12(3), 412–426.

[5] Niemann, B., Rohrbach, S., Miller, M. R., Newby, D. E., Fuster, V., & Kovacic, J. C. (2017). Oxidative stress and cardiovascular risk: obesity, diabetes, smoking, and pollution: Part 3 of a 3-part series. Journal of the American College of Cardiology, 70(2), 230–251.

[6] Petridou, A., Siopi, A., & Mougios, V. (2019). Exercise in the management of obesity. Metabolism: Clinical and Experimental, 92, 163–169.

[7] Zhang, H., Zhang, Y., Zhang, J., & Jia, D. (2024). Exercise alleviates cardiovascular diseases by improving mitochondrial homeostasis. Journal of the American Heart Association, 13(19), e036555.

[8] Jiahao, L., Jiajin, L., & Yifan, L. (2021). Effects of resistance training on insulin sensitivity in the elderly: A meta-analysis of randomized controlled trials. Journal of Exercise Science and Fitness, 19(4), 241–251.

[9] Lopez, P., Taaffe, D. R., Galvão, D. A., Newton, R. U., Nonemacher, E. R., Wendt, V. M., Bassanesi, R. N., Turella, D. J. P., & Rech, A. (2022). Resistance training effectiveness on body composition and body weight outcomes in individuals with overweight and obesity across the lifespan: A systematic review and meta-analysis. Obesity Reviews, 23(5), e13428.

[10] Medeiros, N. da S., de Abreu, F. G., Colato, A. S., de Lemos, L. S., Ramis, T. R., Dorneles, G. P., Funchal, C., & Dani, C. (2015). Effects of concurrent training on oxidative stress and insulin resistance in obese individuals. Oxidative Medicine and Cellular Longevity, 2015, 697181.

[11] Liu, X., Li, Q., Lu, F., & Zhu, D. (2024). Effects of aerobic exercise combined with resistance training on body composition and metabolic health in children and adolescents with overweight or obesity: systematic review and meta-analysis. Frontiers in Public Health, 12, 1409660.

[12] Ma, J. C., Shu, S., Chen, T. X., Bai, H. J., Yang, Y., & Ding, X. W. (2025). Intervention effect of combined resistance and aerobic exercise on type 2 diabetes: A meta-analysis. World Journal of Diabetes, 16(7), 108121.

[13] Pi-Sunyer, F. X. (2000). Obesity: criteria and classification. The Proceedings of the Nutrition Society, 59(4), 505–509.

[14] Zhang, X., Zhu, J., Kim, J. H., Sumerlin, T. S., Feng, Q., & Yu, J. (2023). Metabolic health and adiposity transitions and risks of type 2 diabetes and cardiovascular diseases: a systematic review and meta-analysis. Diabetology & Metabolic Syndrome, 15(1), 60.

[15] Fantuzzi, G. (2005). Adipose tissue, adipokines, and inflammation. The Journal of Allergy and Clinical Immunology, 115(5), 911–920.

[16] Mancuso, P. (2016). The role of adipokines in chronic inflammation. ImmunoTargets and Therapy, 5, 47–56.

[17] de Mello, A. H., Costa, A. B., Engel, J. D. G., & Rezin, G. T. (2018). Mitochondrial dysfunction in obesity. Life Sciences, 192, 26–32.

[18] Furukawa, S., Fujita, T., Shimabukuro, M., Iwaki, M., Yamada, Y., Nakajima, Y., Nakayama, O., Makishima, M., Matsuda, M., & Shimomura, I. (2004). Increased oxidative stress in obesity and its impact on metabolic syndrome. The Journal of Clinical Investigation, 114(12), 1752–1761.

[19] Zhou, Y., Li, H., & Xia, N. (2021). The interplay between adipose tissue and vasculature: Role of oxidative stress in obesity. Frontiers in Cardiovascular Medicine, 8, 650214.

[20] Cerdá, C., Sánchez, C., Climent, B., Vázquez, A., Iradi, A., El Amrani, F., Bediaga, A., & Sáez, G. T. (2014). Oxidative stress and DNA damage in obesity-related tumorigenesis. Advances in Experimental Medicine and Biology, 824, 5–17.

[21] Frohnert, B. I., Sinaiko, A. R., Serrot, F. J., Foncea, R. E., Moran, A., Ikramuddin, S., Choudry, U., & Bernlohr, D. A. (2011). Increased adipose protein carbonylation in human obesity. Obesity, 19(9), 1735–1741.

[22] Shin, S. K., Cho, H. W., Song, S. E., Im, S. S., Bae, J. H., & Song, D. K. (2020). Oxidative stress resulting from the removal of endogenous catalase induces obesity by promoting hyperplasia and hypertrophy of white adipocytes. Redox Biology, 37, 101749.

[23] Thirupathi, A., Wang, M., Lin, J. K., Fekete, G., István, B., Baker, J. S., & Gu, Y. (2021). Effect of different exercise modalities on oxidative stress: A systematic review. BioMed Research International, 2021, 1947928.

[24] Świątkiewicz, I., Wróblewski, M., Nuszkiewicz, J., Sutkowy, P., Wróblewska, J., & Woźniak, A. (2023). The role of oxidative stress enhanced by adiposity in cardiometabolic diseases. International Journal of Molecular Sciences, 24(7), 6382.

[25] Lin, X., Zhang, X., Guo, J., Roberts, C. K., McKenzie, S., Wu, W. C., Liu, S., & Song, Y. (2015). Effects of exercise training on cardiorespiratory fitness and biomarkers of cardiometabolic health: A systematic review and meta-analysis of randomized controlled trials. Journal of the American Heart Association, 4(7), e002014.

[26] Pinckard, K., Baskin, K. K., & Stanford, K. I. (2019). Effects of exercise to improve cardiovascular health. Frontiers in Cardiovascular Medicine, 6, 69.

[27] Ferraro, E., Giammarioli, A. M., Chiandotto, S., Spoletini, I., & Rosano, G. (2014). Exercise-induced skeletal muscle remodeling and metabolic adaptation: redox signaling and role of autophagy. Antioxidants & Redox Signaling, 21(1), 154–176.

[28] Ferraro, E., Giammarioli, A. M., Chiandotto, S., Spoletini, I., & Rosano, G. (2014). Exercise-induced skeletal muscle remodeling and metabolic adaptation: redox signaling and role of autophagy. Antioxidants & Redox Signaling, 21(1), 154–176.

[29] Yan, Z., Lira, V. A., & Greene, N. P. (2012). Exercise training-induced regulation of mitochondrial quality. Exercise and Sport Sciences Reviews, 40(3), 159–164.

[30] Xie, Y., Gu, Y., Li, Z., Zhang, L., & Hei, Y. (2025). Effects of exercise on different antioxidant enzymes and related indicators: a systematic review and meta-analysis of randomized controlled trials. Scientific Reports, 15(1), 12518.

[31] Nassis, G. P., Papantakou, K., Skenderi, K., Triandafillopoulou, M., Kavouras, S. A., Yannakoulia, M., Chrousos, G. P., & Sidossis, L. S. (2005). Aerobic exercise training improves insulin sensitivity without changes in body weight, body fat, adiponectin, and inflammatory markers in overweight and obese girls. Metabolism: Clinical and Experimental, 54(11), 1472–1479.

[32] O'Gorman, D. J., Karlsson, H. K., McQuaid, S., Yousif, O., Rahman, Y., Gasparro, D., Glund, S., Chibalin, A. V., Zierath, J. R., & Nolan, J. J. (2006). Exercise training increases insulin-stimulated glucose disposal and GLUT4 (SLC2A4) protein content in patients with type 2 diabetes. Diabetologia, 49(12), 2983–2992.

[33] Zoladz, J. A., Koziel, A., Woyda-Ploszczyca, A., Celichowski, J., & Jarmuszkiewicz, W. (2016). Endurance training increases the efficiency of rat skeletal muscle mitochondria. Pflugers Archiv: European Journal of Physiology, 468(10), 1709–1724.

[34] Petersen, A. M., & Pedersen, B. K. (2005). The anti-inflammatory effect of exercise. Journal of Applied Physiology, 98(4), 1154–1162.

[35] Farinha, J. B., Steckling, F. M., Stefanello, S. T., Cardoso, M. S., Nunes, L. S., Barcelos, R. P., Duarte, T., Kretzmann, N. A., Mota, C. B., Bresciani, G., Moresco, R. N., Duarte, M. M., Dos Santos, D. L., & Soares, F. A. (2015). Response of oxidative stress and inflammatory biomarkers to a 12-week aerobic exercise training in women with metabolic syndrome. Sports Medicine - Open, 1(1), 19.

[36] Huang, C. J., McAllister, M. J., Slusher, A. L., Webb, H. E., Mock, J. T., & Acevedo, E. O. (2015). Obesity-related oxidative stress: the impact of physical activity and diet manipulation. Sports Medicine - Open, 1(1), 32.

[37] Lim, C., Nunes, E. A., Currier, B. S., McLeod, J. C., Thomas, A. C. Q., & Phillips, S. M. (2022). An evidence-based narrative review of mechanisms of resistance exercise-induced human skeletal muscle hypertrophy. Medicine and Science in Sports and Exercise, 54(9), 1546–1559.

[38] Holten, M. K., Zacho, M., Gaster, M., Juel, C., Wojtaszewski, J. F., & Dela, F. (2004). Strength training increases insulin-mediated glucose uptake, GLUT4 content, and insulin signaling in skeletal muscle in patients with type 2 diabetes. Diabetes, 53(2), 294–305.

[39] Cordeiro, L. S., Linhares, D. G., Castro, J. B. P., Oliveira Barros Dos Santos, A., Lima Dos Santos, L., César Pereira Salustiano Mallen da Silva, G., & Gomes de Souza Vale, R. (2025). Impacts of resistance training on endogenous antioxidants in older individuals: A systematic review and meta-analysis of randomized controlled trials. Journal of Physical Activity & Health, 22(4), 407–417.

[40] Binmahfoz, A., Dighriri, A., Gray, C., & Gray, S. R. (2025). Effect of resistance exercise on body composition, muscle strength and cardiometabolic health during dietary weight loss in people living with overweight or obesity: a systematic review and meta-analysis. BMJ Open Sport & Exercise Medicine, 11(3), e002363.

[41] Alikhani, S., & Sheikholeslami-Vatani, D. (2019). Oxidative stress and anti-oxidant responses to regular resistance training in young and older adult women. Geriatrics & Gerontology International, 19(5), 419–422.

[42] Amiri, E., & Sheikholeslami-Vatani, D. (2023). The role of resistance training and creatine supplementation on oxidative stress, antioxidant defense, muscle strength, and quality of life in older adults. Frontiers in Public Health, 11, 1062832.

[43] Liang, M., Pan, Y., Zhong, T., Zeng, Y., & Cheng, A. S. K. (2021). Effects of aerobic, resistance, and combined exercise on metabolic syndrome parameters and cardiovascular risk factors: a systematic review and network meta-analysis. Reviews in Cardiovascular Medicine, 22(4), 1523–1533.

[44] Hang, S., Xiaoyu, L., Jue, W., Yingli, L., & Li, Z. (2025). Effects of resistance training and aerobic training on improving the composition of middle-aged adults with obesity in an interventional study. Scientific Reports, 15(1), 33972.

[45] Liu, S., Liu, J., Wang, Y., Deng, F., & Deng, Z. (2025). Oxidative stress: Signaling pathways, biological functions, and disease. MedComm, 6(7), e70268.

[46] Hajam, Y. A., Rani, R., Ganie, S. Y., Sheikh, T. A., Javaid, D., Qadri, S. S., Pramodh, S., Alsulimani, A., Alkhanani, M. F., Harakeh, S., Hussain, A., Haque, S., & Reshi, M. S. (2022). Oxidative stress in human pathology and aging: Molecular mechanisms and perspectives. Cells, 11(3), 552.