Diyala Journal of Medicine

ORIGINAL RESEARCH
Published: 25 October 2025
DOI: 10.26505/djm.v29i1.1494

Changes in the Levels of Methionine Adenosyltransferase, Methionine Sulfoxide Reductase A, and Thioredoxin are Associated with Oxidative Stress in Patients with Hyperthyroidism

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Received: 22 May 2025
Accepted: 01 August 2025
Published: 25 October 2025

Abstract

Background: Hyperthyroidism is associated with increased oxidative stress and alterations in enzymatic and non-enzymatic antioxidant systems. Methionine adenosyltransferase (MAT) plays a key role in cellular metabolism and may be involved in redox homeostasis in thyroid disorders.

Objectives: This study aimed to evaluate the levels of MAT and investigate its association with selected enzymatic and non-enzymatic oxidative stress markers in patients with hyperthyroidism.

Patients and Methods: A total of 90 blood serum samples were collected from patients with hyperthyroidism and compared with 50 healthy controls. Enzymatic markers measured included methionine sulfoxide reductase A (MsrA), thioredoxin (Trx), catalase (Cat), myeloperoxidase (MPO), lactoperoxidase (LP), xanthine oxidase (XO), glutathione S-transferase (GST), and senescence marker protein-30 (SMP-30). Non-enzymatic markers included glutathione (GSH), uric acid (UA), albumin (Alb), malondialdehyde (MDA), and peroxynitrite ($ONOO^-$).

Results: Compared to healthy controls, patients with hyperthyroidism showed a significant increase in MAT, Cat, XO, GST, Trx, and LP levels, while SMP-30 was significantly decreased. Among non-enzymatic parameters, MDA and $ONOO^-$ were significantly elevated, and albumin levels were decreased. No significant changes were found in the remaining markers. MAT showed a direct correlation with SMP-30, MPO, MsrA, UA, $ONOO^-$, and Alb, and an inverse relationship with Cat, XO, and GSH. No correlation was observed between MAT and GST, LP, or Trx.

Conclusion: The findings suggest a strong association between MAT activity and oxidative stress in patients with hyperthyroidism. The observed changes point to metabolic imbalances and compromised antioxidant defense mechanisms in these patients.

Keywords: Hyperthyroidism, Enzyme, Methionine adenosyltransferase, Methionine sulfoxide reductase A, Thioredoxin.

Introduction

Hyperthyroidism is a condition that occurs when the thyroid gland secretes excess hormones (1). leading to accelerated metabolism pathways (2). Signs of this disorder may include heartbeats, struggles with heat sensitivity, feelings of unease, and unintended loss of weight (3). Hyperthyroidism

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can manifest in subtle forms. Clear hyperthyroidism is distinguished by levels of thyroid stimulation hormone (often abbreviated as TSH) paired with heightened levels of triiodothyronine (known as T3) or increased levels of thyroxine (referred to as T4). If T3 and T4 levels are elevated while TSH is diminished and T4 levels remain normal, the condition is termed "T3". Subclinical hyperthyroidism is when the TSH levels are low. Both T3 and T4 levels are within the normal range, which can lead to significant long-term complications in both overt and subclinical cases of hyperthyroidism (4).

Hyperthyroidism impacts around 2.5% adults, affecting more women than men at a rate of 2% for women and 0.5% globally. If left untreated. It can lead to heart irregularities. Bone fragility and metabolism issues that result in weight loss Graves' disease, the cause of hyperthyroidism, is an autoimmune condition that appears more frequently in women (5). Research indicates that genetic factors play a role in determining the likelihood of developing Graves' disease accounting for approximately 60 to 80 percent of the risk involved (6). Toxic nodular goiter, the second most common cause, affects 1.5-18 cases per 100,000 people worldwide annually. This disease is characterized by thyroid nodules releasing excess thyroid hormone and is more common in areas with iodine deficiency (5). Excessive exposure to iodine can also lead to hyperthyroidism due to failure of normal homeostasis mechanisms. Iodine-induced hyperthyroidism occurs more commonly in areas that have historically suffered from iodine deficiency (7). Hashimoto's thyroiditis can also cause hyperthyroidism, although Hashimoto's thyroiditis is the most common cause of hypothyroidism (8).

Methionine adenosyltransferases (MATs; EC 2.5.1.6) are very important enzymes for living cells. There are three isomers of the enzyme:

MAT I, II, and III (9). MAT is the only enzyme that produces S-adenosylmethionine (SAM) from adenosine triphosphate (ATP) and methionine (10). SAM is synthesized in the liver and plays a crucial role as an essential donor of the methyl group required for many biological functions (11). It methylates DNA, RNA, and proteins, which is necessary for maintaining genomic stability, regulating gene expression, and maintaining cellular homeostasis (12). Additionally, it regulates cellular processes whose dysregulation may contribute to pathological conditions (13). Low levels of SAM affect lipid metabolism, contributing to the development of fatty liver disease, injury, and even cancer. SAM deficiency may lead to increased fat accumulation in the liver, contributing to the development of fatty liver disease (12). SAM is used to treat liver dysfunction (14), and also in the treatment of depression (11) as it is necessary for the production of neurotransmitters that affect mental health (15, 16).

Furthermore, SAM plays a role in reducing increased homocysteine levels. After demethylation of SAM, it is converted to S-adenosylhomocysteine (SAH), which is then hydrolyzed to homocysteine (Hcy)(17). Homocysteine can enter the transsulfate pathway to promote glutathione synthesis, or it can be converted back to methionine and then to SAM (17, 18). Elevated homocysteine levels are primarily related to their association with endothelial dysfunction and atherosclerosis(19). In addition to elevated SAM levels, homocysteine levels can also be elevated for several other reasons, including genetic factors, deficiencies of B vitamins and folic acid, aging, certain medications, and pathological conditions (19, 20).

A 2020 study showed that changes in the activity of the MAT1A and MAT2A enzymes affect methionine metabolism in chronic liver disease, contributing to the development of cirrhosis and liver cancer (17).

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A 2023 study on mice also found that MAT2A increases with age. Increased MAT2A activity in the skeletal muscles of elderly mice led to impaired metabolism, contributing to muscle weakness. However, inhibiting MAT2A has been shown to improve muscle strength (21).

Previous studies have primarily focused on general oxidative markers in thyroid disorders, with limited evaluation of specific enzymatic systems such as MAT, MsrA, and Trx. However, none have systematically analyzed the interaction between these enzymatic antioxidants and non-enzymatic parameters in the context of hyperthyroidism. Given the lack of studies on the MAT and its relationship with enzymatic and non-enzymatic variables in patients with hyperthyroidism, as well as to understand the biological changes associated with these conditions and their relationship to oxidative balance and metabolism, this study was conducted to evaluate this enzyme and important measured variables, especially methionine sulfoxide reductase A and thioredoxin.

Patients and Methods

Study design and blood sample collection:

Blood samples were collected from individuals with hyperthyroidism and healthy individuals at Al-Salam Teaching Hospital in Mosul, under the supervision of specialist physicians, between November 2024 and the end of February 2025(There was enough time due to the increasing number of patients in Mosul). After completing a questionnaire, blood was drawn from a vein, and serum was separated. The serum was not hemolyzed to ensure clarity. The sample was then divided into three parts and stored frozen at -20°C to measure the variables selected in the study: The study protocol was approved by the Institutional Ethics Committee of the University of Mosul, Iraq (Approval No. DMED-2024-015). All participants gave informed consent before enrollment.

The age range of the hyperthyroid patients was 25–60 years, and samples were collected between 8:00 and 11:00 am to minimize diurnal variations in hormonal and oxidative stress markers.

Among the 90 hyperthyroid patients, 68 were female and 22 were male, reflecting the higher prevalence in women.

Biochemical assays:

Methods used to measure enzymatic and non-enzymatic variables:

Standard kits from the French company BioLabo were used to measure albumin and uric acid concentrations. Manual methods were also used to measure the levels of enzymatic and non-enzymatic parameters, as follows:

MAT activity: The activity of the MAT was assessed by catalyzing the conversion of methionine to SAM in the presence of ATP, which releases the resulting phosphate group, which can be detected by the Malachite green reaction (22).

Methionine sulfoxide reductase A activity: The activity of the methionine sulfoxide reductase A (MsrA) was assessed by catalyzing the reduction of methionine sulfoxide to methionine in the presence of dimethyl sulfoxide as a substrate and dithiothreitol (23).

Glutathione S-transferase activity: The activity of glutathione S-transferase (GST) was estimated according to the method used by researchers Habig *et al*. (1974) (24). GST enzyme catalyzes the binding of compounds containing electrophilic groups, especially aromatic rings such as 1-chloro-2,4-dinitrobenzene, with the thiol group (-SH) of glutathione. Then, the absorbance intensity of the resulting solution is measured using a spectrophotometer.

Trx activity: The researcher Holmgren (1979) (25) used the method to measure the activity of Trx in the sample. The measurement process involves using

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Trx in the sample to reduce the disulfide bonds of the insulin hormone with dithiothreitol, resulting in a cloudy white color, the absorption intensity of which is measured spectroscopically.

Lactoperoxidase activity: The activity of lactoperoxidase (LP) was determined according to the method used by Tayefi-Nasrabadi *et al*. (2011) (26), as LP oxidizes the substrate pyrogallo to purpurogallin in the presence of hydrogen peroxide.

Gluconolactonase as SMP-30 Activity: SMP-30 activity was determined by measuring its gluconolactonase activity using the colorimetric method (27). It hydrolyzes the substrate D-gluconolactone, resulting in ring opening and the formation of acidity, which reduces the absorption of the added p-nitrophenol.

Catalase activity: The activity of the catalase (Cat) was estimated according to the method of Boriskin *et al*. (2019) (28), which is based on the oxidation of 4-ammonium molybdenum by hydrogen peroxide remaining from the enzymatic reaction of the Cat, producing a colored substance whose absorption intensity can be measured spectrophotometrically.

Myeloperoxidase activity: Myeloperoxidase's (MPO) function was determined using the technique outlined in the study by Kumar *et al*. (2002) (29), which involves the enzyme oxidizing orthodiensidine with hydrogen peroxide to generate a colored compound that can be analyzed using a spectrophotometer.

Xanthine oxidase activity: The activity of xanthine oxidase was assessed following the technique outlined by Ackermann and Brill in 1974 (30), to measure the production of acid.

Albumin concentration: The concentration of albumin was determined using the Bromocresol method with a kit from BioLabo, a company based in France.

Glutathione concentration: To determine serum glutathione concentration, a modified version of the method developed by Sedlak and Lindsay in 1968 (31), which utilizes Ellman's reagent, was employed.

Uric acid concentration: The level of acid was measured using a BioLab kit that relies on an enzymatic approach. The enzyme uricase converts uric acid into allantoin and hydrogen peroxide.

Malondialdehyde concentration: Malondialdehyde concentration was determined using a modified method from Guidet and Shah (1989) (32), which is based on the reaction of malondialdehyde with thiobarbituric acid (TBA).

Peroxynitrite concentration: Peroxynitrite was determined using a modified method from Vanuffelen *et al*. (1998) (33), which oxidizes phenol to nitrophenol.

Statistical Analysis

SPSS 21 was used to determine the mean, standard deviation (SD), and correlation. The t-test was chosen to compare each pair of variables and determine the significance of the difference indicated by the p-value. A significant difference occurs when the p-value is ≤ 0.05, while a non-significant difference occurs when the p-value is >0.05 (34).

Results

Study of MAT enzyme and other enzyme variables in hyperthyroid patients: The enzymatic profile in hyperthyroid patients shows statistically significant alterations in several oxidative stress-related enzymes when compared to the control group (Table 1). Specifically, levels of methionine adenosyltransferase ($p=0.031$), Thyroidoxin ($p=0.031$), Lactoperoxidase ($p=0.017$), Catalase ($p=0.047$), Xanthine oxidase ($p=0.0001$), and Glutathione S-transferase ($p=0.002$) were significantly elevated in hyperthyroid patients, suggesting an upregulation of antioxidant and redox-modulating enzymes in response to increased metabolic activity and oxidative stress associated with hyperthyroidism.

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Interestingly, senescence marker protein-30 was significantly lower in hyperthyroid patients (p=0.0001), possibly indicating altered cellular aging processes or stress responses. No significant differences were observed in Methionine sulfoxide reductase A (p=0.382) and Myeloperoxidase (p=0.966), suggesting these enzymes may not be directly influenced by the hyperthyroid state.

The findings highlight an enhanced oxidative stress environment in hyperthyroidism, triggering compensatory changes in enzymatic antioxidants (Table 1).

Table 1. Enzymatic results in hyperthyroidism patients compared with the control group.

Measured enzymes (U/L)	Control group		Hyperthyroid patients group		Probability Value
	Average	Standard deviation	Average	Standard deviation	
Methionine adenosyltransferase	50.04	3.17	60.99	5.58	0.031*
Methionine sulfoxide reductase A	353.12	12.2	378.85	12.55	0.382
Thyroidoxin	6.12	0.708	8.88	1.01	0.031*
Lactoperoxidase	40.81	1.82	50.06	2.76	0.017*
Catalase	69.15	4.32	81.99	5.38	0.047*
Xanthine oxidase	447.06	10.19	605.12	32.33	0.0001*
Glutathione S-transferase	94.66	8.65	189.1	21.15	0.002*
Myeloperoxidase	55.98	2.77	57.3	5.92	0.966
Aging marker protein-30	0.807	0.029	0.39	0.055	0.0001*
***Significant at (p≤0.05)**					

Study of MAT and other non-enzymatic variables in hyperthyroid patients: Table 2 shows statistically significant differences in oxidative stress markers between hyperthyroid patients and the control group (P ≤ 0.05). Glutathione and albumin levels were significantly decreased in hyperthyroid patients, indicating a compromised antioxidant defense system. In contrast, oxidative stress markers—peroxynitrite and malondialdehyde—were elevated considerably, reflecting increased lipid peroxidation and oxidative damage (Table 2).

Table 2. Levels of antioxidants and oxidants in hyperthyroid patients compared with the control group.

Measured variables	Control group		Hyperthyroid patients group		Probability Values
	Average	Standard deviation	Average	Standard deviation	
Glutathione (µmol/L)	10.66	0.289	6.87	0.263	0.031*
Albumin (g/100ml)	51.25	0.54	38.18	0.893	0.0001*
Uric acid (µmol/L)	63.06	2.15	51.94	2.87	0.048*
Peroxynitrite (µmol/L)	33.44	2.34	46.52	2.95	0.032*
Malondialdehyde (µmol/L)	11.91	1.63	19.89	1.79	0.031*
***Significant at (p≤0.05)**					

Correlation between MAT and measured variables in hyperthyroid patients: Table 3 presents the correlation coefficients (R values) and significance levels (p-values) between methionine adenosyltransferase (MAT) and various biochemical variables. Several statistically significant correlations (p≤ 0.05) were observed, indicating potential relationships between MAT activity and oxidative stress-related biomarkers in hyperthyroid patients.

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A strong positive correlation was found between MAT and albumin (R = 0.888, p = 0.0001), suggesting a close association between MAT and plasma protein synthesis or antioxidant capacity. Similarly, MAT showed a significant positive correlation with methionine sulfoxide reductase A (R = 0.638, p = 0.0001) and myeloperoxidase (R = 0.705, p = 0.001), indicating potential co-regulation or shared pathways in redox balance.

Significant negative correlations were observed with catalase (R = –0.538, p = 0.005), xanthine oxidase (R = –0.673, p = 0.0001), and glutathione (R = –0.758, p = 0.0001), reflecting an inverse relationship between MAT and these oxidative stress markers. These findings suggest that increased MAT activity may be associated with decreased levels or activity of certain antioxidant enzymes, potentially due to feedback mechanisms or shifts in redox homeostasis.

Additionally, uric acid exhibited a moderate positive correlation with MAT (R = 0.487, p = 0.010), which may reflect its role as a secondary antioxidant in compensating for oxidative stress.

In contrast, lactoperoxidase, glutathione S-transferase, peroxynitrite, malondialdehyde, and thioredoxin did not show statistically significant correlations with MAT (p > 0.05), suggesting limited or variable associations in the hyperthyroid context.

Overall, the results indicate that MAT activity is significantly correlated with key oxidative stress and antioxidant parameters, highlighting its potential role in redox regulation during hyperthyroidism.

Table 3. Correlations of the measured biochemical variables with methionine adenosyl transferase.

Measured variables	R value	p value
Lactoperoxidase	-0.338	0.085
Catalase	-0.538	0.005*
Xanthine oxidase	-0.673	0.0001*
Glutathione S-transferase	0.135	0.501
Glutathione	-0.758	0.0001*
Albumin	0.888	0.0001*
Uric acid	0.487	0.010*
Methionine sulfoxide reductase A	0.638	0.0001*
Peroxynitrite	0.031	0.877
Malondialdehyde	-0.003	0.989
Myeloperoxidase	0.705	0.001*
Aging marker protein	0.765	0.0001*
Thioredoxin	-0.334	0.15

Discussion

The current findings demonstrate significant enzymatic alterations in hyperthyroid patients, reflecting increased oxidative stress and compensatory antioxidant responses. Methionine adenosyltransferase, thioredoxin, lactoperoxidase, catalase, xanthine oxidase, and glutathione S-transferase levels were significantly elevated in the hyperthyroid group compared to controls. These enzymes have functions in maintaining redox balance and defending against oxidative harm. This aligns with the heightened metabolic state linked with a thyroid (35, 36). An increase in xanthine oxidase activity could lead to the production of reactive oxygen species (ROS), intensifying oxidative stress (37). Likewise. In the same vein as well as similarly, the surge in thioredoxin and glutathiophoresis S transferase levels indicates a compensatory boost in cellular antioxidant mechanisms aimed at reducing

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oxidative harm (36). The notable rise in catalase and lactoperoxidase levels might also help counteract hydrogen peroxide production— a reactive oxygen species generated when metabolic activity is heightened (38).

It is quite intriguing that the noticeable reduction in SMP-30 among individuals with hyperthyroidism could point towards a cellular aging process or hampered stress response. This is because SMP- 30 typically diminishes in conditions. On the other hand, there were no significant variances noted in the levels of methionine sulfoxide reductase A and myeloperoxidase in hyperthyroid patients (39). This might indicate regulation of enzymes in the hyperthyroid state due to potential tissue-specific expression or differing regulatory pathways (40). These results back up the theory that an overactive thyroid leads to stress and prompts specific enzyme reactions to maintain the redox balance.

In individuals with a thyroid gland (hyperthyroidism), the rise in MAT activity is a typical reaction to the body's increased metabolic rate and the greater demand for methylation reactions essential for biosynthesis that rely upon SAM. A compound derived from MAT itself. This phenomenon is directly influenced by the activation of genes prompted by thyroid hormones. Thyroid hormones, recognized for their metabolic effects, are heightened. The hormones trigger thyroid hormone receptors (TRs), which attach to gene promoters, like the MAT enzyme promoter, to produce the MAT enzyme. The activation of these genes results in MAT gene activity. As a result of the heightened metabolic functions during hyperthyroidism, there is a demand for S-adenosylmethionine (SAM), a crucial compound in cell methylation processes. This increased need for SAM is essential for DNA adjustments (DNA methylation), controlling gene expression patterns, and creating phospholipids and polyamines vital for cell expansion and development (41).

The notable rise in catalase (CAT) action among individuals with hyperthyroidism when compared to those who are in good health is linked to a rise, in oxidative stress. This increase is believed to stem from the overproduction of thyroid hormones (T3 and T4) (42). These hormones are known to boost metabolism and oxygen intake levels. Consequently, this leads to the generation of levels of reactive oxygen species (ROS) (43). The body responds by triggering its defense mechanisms, which involve boosting the activity of the catalase enzyme. This enzyme helps break down hydrogen peroxide into oxygen and water, mitigating the impact of oxidative stress (OS), minimizing oxidative damage, and upholding the balance within cells (44).

Supporting this interpretation, a recent study in mice with hyperthyroidism demonstrated increased activity of antioxidant enzymes, such as catalase, in pancreatic tissue (45, 46).

Elevated XO is a biomarker of exacerbated OS associated with hyperthyroidism (47). The significant increase in the activity of this enzyme in patients may be attributed to increased OS resulting from hypermetabolic syndrome associated with increased secretion of thyroid hormones, which leads to increased production of various ROS such as hydrogen peroxide and anion superoxide radical formed by XO (2, 48) through the conversion of xanthine to uric acid, with hydrogen peroxide being produced as a by-product (49). In cases of hyperthyroidism, an increase in the activity of XO is observed, which exacerbates OS, leading to cell and tissue damage (50). A study by Kihara *et al*. (51) on the effect of some compounds on the XO showed that inhibiting XO using allopurinol reduces ROS levels in animal models of hyperthyroidism. Studies have shown that the effectiveness of XO increases in cases of hyperthyroidism, which enhances OS.

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The significant increase in thioredoxin (Trx) in hyperthyroid patients is attributed to the increased OS resulting from the increased production of thyroid hormones (52). This condition leads to the stimulation of gene and protein expression of thioredoxin as a defines mechanism against oxidative damage resulting from hyperthyroidism, which improves the ability of cells to resist free radicals and protects tissues from damage resulting from OS by reducing hydrogen peroxide to water and oxygen by Trx, improving the redox balance and maintaining the redox balance (53). A study by Kihara *et al*. (2005) (54) showed that serum Trx levels were significantly elevated in Graves' disease patients compared to healthy controls, regardless of thyroid function status, suggesting a role for thioredoxin in responding to OS and regulating thyroid hormone production. A recent study conducted on animal models showed that hyperthyroidism led to increased gene expression of thioredoxins (TXN1 and TXN2) and thioredoxin reductase 1 (TXNRD1) in the liver, suggesting a compensatory response to OS. Furthermore, treating these models with an antioxidant decreased this expression, demonstrating the role of antioxidant therapies in reducing oxidative damage (55).

The significant increase in lactoperoxidase (LP) levels in hyperthyroid patients may be attributed to elevated levels of T3 and T4 hormones, which stimulate the body's metabolism. This acceleration of metabolism leads to increased production of oxidizing compounds such as hydrogen peroxide, causing OS in cells (2, 56, 57).To reduce harmful hydrogen peroxide, the body stimulates the production of lactoperoxidase (LP). LPO contributes to the consumption of hydrogen peroxide, forming thiocyanate ($OSCN^-$), a compound with antibacterial properties that reduce oxidative damage to cells (58). GST is significantly elevated in hyperthyroid patients as a result of increased OS resulting from accelerated metabolic processes (2). GST acts as an antioxidant enzyme that contributes to detoxification and neutralization of free radicals by binding to the glutathione molecule, which explains its high level as a cellular defines response (59).

A study by Baek *et al*. (2021) (51) showed that SMP-30 is one of the proteins directly linked to aging indicators and OS. Due to severe OS resulting from metabolic hyperactivity in hyperthyroid patients (2, 37), SMP-30 becomes deficient due to cell damage and increased enzyme consumption in attempts to combat oxidative damage. Consequently, the efficiency of SMP-30 expression decreases, leading to lower levels in the blood. This accelerates cellular aging, which impairs the cells' ability to regenerate and perform their functions properly (60).

Recent studies indicate that hyperthyroid patients have significantly higher levels of malondialdehyde (MDA), a biomarker of lipid peroxidation and OS (61), compared to healthy individuals. This increase is attributed to the heightened production of ROS due to increased metabolic activity and high oxygen consumption associated with hyperthyroidism (2). Especially superoxide anion radical and nitric oxide, which rapidly react to form peroxynitrite (ONOO-) (62), a potent oxidizing molecule that can damage proteins, lipids, and DNA (54). Oxidants interact with lipids in cell membranes, leading to their decomposition and the formation of MDA upon lipid damage. Studies indicate that this elevation in MDA is associated with a decreased ability of the body to resist OS, leading to tissue damage and worsening clinical symptoms in hyperthyroid patients (61).

Low glutathione levels in hyperthyroid patients are an essential indicator of increased OS in the body. Evidence suggests that hyperthyroidism leads to an increase in the production of ROS, which depletes glutathione stores and leads to decreased levels. This decrease in glutathione may contribute to the worsening of oxidative damage associated with

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hyperthyroidism (63). A study published in 2022 analyzed the relationship between hyperthyroidism and OS. The results showed a positive correlation between thyroid hormone levels and OS markers such as malondialdehyde (MDA) and glutathione (GSH). This suggests that hyperthyroidism may lead to increased OS, reflected in decreased GSH levels (64). Low albumin levels in hyperthyroid patients are caused by excessive secretion of thyroid hormones, which accelerates metabolism (2) and increases the breakdown (consumption) of proteins in the body, including albumin (58). Albumin acts as an essential antioxidant in plasma, and continuous exposure to OS leads to the degradation and oxidative modification of albumin, reducing its functional levels and consequently causing a deficiency in active albumin in the body (59). In hyperthyroidism, metabolism accelerates, increasing uric acid production due to the accelerated purine metabolism. However, these hormones also improve kidney function by increasing glomerular filtration rate (GFR) and renal plasma flow, enhancing the kidneys' ability to excrete uric acid. Thus, despite increased uric acid production, increased renal excretion leads to lower blood levels. Supporting this interpretation, a recent study showed that patients with hyperthyroidism had significantly lower uric acid levels, suggesting that increased GFR plays a significant role in this decrease (64). When comparing hyperthyroid patients with each other, a direct correlation was observed between MAT and methionine sulfoxide reductase A, senescence aging marker-30, myeloperoxidase, uric acid, and albumin (Table 3), and an inverse correlation with catalase, xanthine oxidase, and glutathione: The correlation analysis reveals that methionine adenosyltransferase (MAT) is significantly associated with several oxidative stress markers in hyperthyroid patients. The strong positive correlations with albumin, methionine sulfoxide reductase A, and myeloperoxidase suggest a possible link between MAT activity and antioxidant defense mechanisms. Conversely, the negative correlations with catalase, xanthine oxidase, and glutathione indicate a potential compensatory or regulatory relationship in.

Conclusion

Our findings demonstrate that AI can effectively automate blood cell classification, reducing the subjectivity of manual microscopy. All three models-wavelet scattering with SVM, a custom CNN, and ResNet-achieved high accuracy (>95%), with the wavelet-SVM combination performing best (~98.9%). However, a limitation of this study is the genetic properties of our dataset, which differs from others and may impact model generalizability. Future research should expand datasets and incorporate genetic variability to strengthen clinical applicability. Despite this, our work confirms that AI-driven frameworks are promising tools for enhancing hematological diagnostics

Source of funding: No source of funding.
Ethical clearance: The Research Ethics Committee of Department of Chemistry, College of Sciences, Mosul University approved this study, and in accordance with the ethical guidelines of the Declaration of Ethical Committee of the College (2024-015). Written consent was obtained from all patients before inclusion.
Conflict of interest: None.
Use of Generative Artificial Intelligence (AI): The authors state that they did not use any generative AI tools for creating or editing the language of the manuscript.
Acknowledgments: We sincerely thank the physicians and laboratory staff of Al-Salam Teaching Hospital in Mosul and the staff and management of the Chemistry Department at the College of Science, University of Mosul, for their invaluable support and contributions, which were instrumental in the successful completion of this

ORIGINAL RESEARCH
Published: 25 October 2025
DOI: 10.26505/djm.v29i1.1494

study.

References

1. Chaker L, Cooper DS, 1. Chaker L, Cooper DS, Walsh JP, Peeters RP. The Hyperthyroidism. Lancet. 2024 Feb 24;403(10428):768-80. https://doi.org/10.5772/intechopen.1007146
2. Sultana R, Shahin AD, Jawadul HM. Measurement of oxidative stress and total antioxidant capacity in hyperthyroid patients following treatment with carbimazole and antioxidant. Heliyon. 2022 Jan 1;8(1). https://doi.org/10.1016/j.heliyon.2021.e08651
3. Battikh E, Mostafa S, Alfar H, Obaid K, Ahmed AI, Sawaf B, Al-Mohanadi D. Hyperthyroidism-Induced Nondiabetic Ketoacidosis: A Rare Case Report. Clinical Case Reports. 2025 Feb;13(2):e9698. https://doi.org/10.1002/ccr3.9698
4. Mathew P, Kaur J, Rawla P, Fortes K. Hyperthyroidism (Nursing). DOI: https://doi.org/10.1001/jama.2023.190.
5. Lee SY, Pearce EN. Hyperthyroidism: a review. Jama. 2023 Oct 17;330(15):1472-83. https://doi.org/10.1001/jama.2023.19052
6. Grixti L, Lane LC, Pearce SH. The genetics of Graves' disease. Reviews in endocrine and metabolic disorders. 2024 Feb;25(1):203-14. https://doi.org/10.1007/s11154-023-09848-8
7. Braverman KD, Pearce EN. Iodine and Hyperthyroidism: A Double-Edged Sword. Endocrine Practice. 2025 Mar 1;31(3):390-5. https://doi.org/10.1016/j.eprac.2024.10.014
8. Fariduddin MM, Singh G. Thyroiditis. InStatPearls 2023 Aug 8. https://www.ncbi.nlm.nih.gov/books/NBK555975/
9. Gou L, Liu D, Fan TP, Deng H, Cai Y. Efficient spermidine production using a multi-enzyme cascade system utilizing methionine adenosyltransferase from Lactobacillus fermentum with Reduced Product Inhibition and Acidic pH Preference. Journal of Biotechnology. 2025 Mar 1;399:141-52. https://doi.org/10.1016/j.jbiotec.2025.01.016
10. Zhu X, Zhang T, Tang C, Wang Z, Guo L, Wang P, Zhang S, Wu J. Methionine adenosyltransferase MAT3 positively regulates pear pollen tube growth, possibly through interaction with pectin lyase-like protein PLL1. Physiologia Plantarum. 2025 Jan;177(1):e70122. https://doi.org/10.1111/ppl.70122
11. Limveeraprajak N, Nakhawatchana S, Visukamol A, Siripakkaphant C, Suttajit S, Srisurapanont M. Efficacy and acceptability of S-adenosyl-L-methionine (SAMe) for depressed patients: A systematic review and meta-analysis. Progress in Neuro-Psychopharmacology and Biological Psychiatry. 2024 Jun 8;132:110985. https://doi.org/10.1016/j.pnpbp.2024.110985
12. Fernández-Ramos D, Lopitz-Otsoa F, Lu SC, Mato JM. S-adenosylmethionine: a multifaceted regulator in cancer pathogenesis and therapy. Cancers. 2025 Feb 5;17(3):535. https://doi.org/10.3390/cancers17030535
13. Xing Z, Tu BP. Mechanisms and rationales of SAM homeostasis. Trends in biochemical sciences. 2025 Mar 1;50(3):242-54. https://doi.org/10.1016/j.tibs.2024.12.009
14. Baden KER, McClain H, Craig E, Gibson N, Draime JA, Chen AMH. S-Adenosylmethionine (SAMe) for liver health: A systematic review. Nutrients. 2024;16(21):3668. https://doi.org/10.3390/nu16213668
15. Abdulhameed O, Al-Helaly L. Methionine Sulfoxide Reductase A And Neurotransmission Enzymes In Autism Spectrum Disorder And Dystocia Related Autistics. Georgian medical news. 2024 May(350):36-41.
16. Al-Taee KM, Al-Helaly LA. Hydrogen Sulfide and Cystathionine γ–Lyase with Oxidants and Antioxidants Levels for Patients with Epilepsy Diseases. Pharmacognosy Journal. 2024;16(2). https://doi.org/10.5530/pj.2024.16.48
17. Bravo AC, Aguilera MN, Marziali NR, Moritz L, Wingert V, Klotz K, Schumann A, Grünert SC, Spiekerkoetter U, Berger U, Lederer AK. Analysis of S-adenosylmethionine and S-adenosylhomocysteine: method optimisation and profiling in healthy adults upon short-term dietary intervention. Metabolites. 2022 Apr 20;12(5):373. https://doi.org/10.3390/metabo12050373
18. Li Z, Wang F, Liang B, Su Y, Sun S, Xia S, Shao J, Zhang Z, Hong M, Zhang F, Zheng S. Methionine metabolism in chronic liver diseases: an update on

ORIGINAL RESEARCH
Published: 25 October 2025
DOI: 10.26505/djm.v29i1.1494

molecular mechanism and therapeutic implication. Signal transduction and targeted therapy. 2020 Dec 4;5(1):280.https://doi.org/10.1038/s41392-020-00349-7

19. Yuan D, Chu J, Lin H, Zhu G, Qian J, Yu Y, Yao T, Ping F, Chen F, Liu X. Mechanism of homocysteine-mediated endothelial injury and its consequences for atherosclerosis. Frontiers in cardiovascular medicine. 2023 Jan16;9:1109445. https://doi.org/10.3389/fcvm.2022.1109445

20. Hameed OM, Al-Helaly LA. Evaluation the level of total fucose and some enzymes in the blood of patients with neurological diseases. Egyptian Journal of Chemistry. 2021 Oct1;64(10):5613-8. https://doi.org/10.21608/ejchem.2021.76538.3745

21. Yin C, Zheng T, Chang X. Biosynthesis of S-Adenosylmethionine by magnetically immobilized Escherichia coli cells highly expressing a methionine adenosyltransferase variant. Molecules. 2017 Aug 18;22(8):1365. https://doi.org/10.3390/molecules22081365

22. Rajabian N, Ikhapoh I, Shahini S, Choudhury D, Thiyagarajan R, Shahini A, Kulczyk J, Breed K, Saha S, Mohamed MA, Udin SB. Methionine adenosyltransferase2A inhibition restores metabolism to improve regenerative capacity and strength of aged skeletal muscle. Nature Communications. 2023 Feb 16;14(1):886. https://doi.org/10.1038/s41467-023-36483-3

23. Wu PF, Zhang Z, Guan XL, Li YL, Zeng JH, Zhang JJ, Long LH, Hu ZL, Wang F, Chen JG. A specific and rapid colorimetric method to monitor the activity of methionine sulfoxide reductase A. Enzyme and Microbial Technology. 2013 Dec 10;53(6-7):391-7. https://doi.org/10.1016/j.enzmictec.2013.08.005

24. Habig WH, Pabst MJ, Jakoby WB. Glutathione S-transferases: the first enzymatic step in mercapturic acid formation. Journal of biological Chemistry. 1974 Nov 25;249(22):7130-9. https://doi.org/10.1016/S0021-9258(19)42083-8

25. Holmgren A. Thioredoxin catalyzes the reduction of insulin disulfides by dithiothreitol and dihydrolipoamide. Journal of Biological Chemistry. 1979 Oct 10;254(19):9627-32. https://doi.org/10.1016/S0021-9258(19)83562-7

26. Tayefi-Nasrabadi H, Hoseinpour-fayzi MA, Mohasseli M. Effect of heat treatment on lactoperoxidase activity in camel milk: A comparison with bovine lactoperoxidase. Small Ruminant Research. 2011 Aug 1;99(2-3):187-90. https://doi.org/10.1016/j.smallrumres.2011.04.007

27. Hucho F, Wallenfels K. Glucono-δ-lactonase from Escherichia coli. Biochimica et Biophysica Acta (BBA)-Enzymology. 1972 Jul 13;276(1):176-9. https://doi.org/10.1016/00052744(72)90018-6

28. Boriskin P, Deviatkin A, Nikitin A, Pavlova O, Toropovskiy A. Relationship of catalase activity distribution in serum and tissues of small experimental animals. InIOP Conference Series: Earth and Environmental Science 2019 Dec 1 (Vol. 403, No. 1, p. 012113). IOP Publishing. https://doi.org/10.1088/17551315/403/1/012113

29. Kumar P, Pai K, Pandey HP, Sundar S. NADH-oxidase, NADPH-oxidase and myeloperoxidase activity of visceral leishmaniasis patients. Journal of medical microbiology. 2002 Oct;51(10):832-6. https://doi.org/10.1099/0022-1317-51-10-832

30. Bergmeyer HU, editor. Methods of enzymatic analysis. Elsevier; 2012 Dec 2.

31. Sedlak J, Lindsay RH. Estimation of total, protein-bound, and nonprotein sulfhydryl groups in tissue with Ellman's reagent. Analytical biochemistry. 1968 Jan 1;25:192-205.

32. Guidet BE, Shah SV. Enhanced in vivo H2O2 generation by rat kidney in glycerol-induced renal failure. American Journal of Physiology-Renal Physiology. 1989 Sep 1;257(3):F440-5. https://doi.org/10.1152/ajprenal.1989.257.3.F440

33. VanUFFELEN EB, Van der ZEE J, de KOSTER MB, VanSTEVENINCK J, ELFERINK GJ. Intracellular but not extracellular conversion of nitroxyl anion into nitric oxide leads to stimulation of human neutrophil migration. Biochemical Journal. 1998 Mar 1;330(2):719-22. https://doi.org/10.1042/bj3300719

34. Morgan GA, Barrett KC, Leech NL, Gloeckner GW. IBM SPSS for introductory statistics: Use and interpretation. Routledge; 2019 Jul 15. https://doi.org/10.4324/9780429287657

ORIGINAL RESEARCH
Published: 25 October 2025
DOI: 10.26505/djm.v29i1.1494

35. Chattopadhyay S, Sahoo DK, Subudhi U, Chainy GB. Differential expression profiles of antioxidant enzymes and glutathione redox status in hyperthyroid rats: a temporal analysis. Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology. 2007 Sep 1;146(3):383-91. https://doi.org/10.1016/j.cbpc.2007.04.010
36. Kochman J, Jakubczyk K, Bargiel P, Janda-Milczarek K. The influence of oxidative stress on thyroid diseases. Antioxidants. 2021 Sep10;10(9):1442. https://doi.org/10.3390/antiox10091442
37. Bortolotti M, Polito L, Battelli MG, Bolognesi A. Xanthine oxidoreductase: One enzyme for multiple physiological tasks. Redox biology. 2021 May 1;41:101882. https://doi.org/10.1016/j.redox.2021.101882
38. Carmo de Carvalho e Martins MD, da Silva Santos Oliveira AS, da Silva LA, Primo MG, de Carvalho Lira VB. Biological indicators of oxidative stress [malondialdehyde, catalase, glutathione peroxidase, and superoxide dismutase] and their application in nutrition. InBiomarkers in nutrition 2022 Oct 15 (pp. 833-856). Cham: Springer International Publishing. https://doi.org/10.1007/978-3-031-07389-2
39. Pereira FC. Bioactive Effects of Selected Marine-derived Compounds on Breast Cancer Cell Lines (Master's thesis, Universidade do Porto (Portugal)).
40. Younes SH, Al-Helaly LA. Glutaredoxin-1 and other antioxidant enzymes in two subtypes (B-cell and T-cell) of acute lymphoblastic leukemia patients. South Eastern European Journal of Public Health.2024;11:10–7. https://doi.org/10.70135/seejph.vi.1105
41. Wu SM, Huang YH, Lu YH, Chien LF, Yeh CT, Tsai MM, Liao CH, Chen WJ, Liao CJ, Cheng WL, Lin KH. Thyroid hormone receptor-mediated regulation of the methionine adenosyltransferase 1 gene is associated with cell invasion in hepatoma cell lines. Cellular and molecular life sciences. 2010Jun;67(11):1831-43. https://doi.org/10.1007/s00018-010-0281-2
42. Festus O, Dic-Ijiewere E, Ailemen F, Imafidon N, Ebo J, Nwankwo C, Iweka F. Oxidative stress pattern of patients with abnormal thyroid function in Southern Nigeria. Journal of Research in Applied and Basic Medical Sciences. 2025 Jan 10;11(1):85-97. http://dx.doi.org/10.61186/rabms.11.1.85
43. Bushra SK, Mahmood T. Study of oxidative stress and inflammatory status in thyroid dysfunction: A systematic Review. International Journal of Drug Delivery Technology.2024;14(1):516-22. https://doi.org/10.25258/ijddt.14.1.71
44. Wang Y, Ding L, Feng J, Lin Z, Yao H, You X, Zhang X, Sun W, Liu Y, Wang P. Mesoporous cerium oxide nanoenzyme for Efficacious impeding tumor and metastasis via Conferring resistance to anoikis. Biomaterials. 2025 Mar 1;314:122876. 2025;314:122876. https://doi.org/10.1016/j.biomaterials.2024.122876
45. AL-Hamdani IH, Al-Helaly LA. PEROXIREDOXIN 3 AND OXIDATIVE STRESS IN RECURRENT ABORTION PATIENTS. Military Medical Science Letters/Vojenské zdravotnické Listy. 2023 Jan 1;92(1). https://doi.org/10.31482/mmsl.2022.034
46. Mahmood ES, Al-Helaly LA. Biochemical and Histological Study of Aminoacylase-1 Purified from Amniotic Fluid in Rats with Oxidative Stress Induced by Lead Acetate. Baghdad Science Journal. 2021 Jul 1;18(3). http://dx.doi.org/10.21123/bsj.2021.18.3.0583
47. Alfathi M, Alabdaly Y, Al-Hayyali F. Ameliorative Effect of Spirulina against gentamicin toxicity in liver and kidney tissues of male rat. Egyptian Journal of Histology. 2023 Dec 1;46(4):1666-75. https://doi.org/10.21608/ejh.2022.155247.1750
48. Badawi LF. Isolation and characterization of xanthine oxidase from tissues of benign and malignant colon tumors patients. Raf J Sci. 2012;23(4):70–82. https://search.emarefa.net/detail/BIM-322203
49. Pannala VR, Dash RK. Mechanistic characterization of the thioredoxin system in the removal of hydrogen peroxide. Biophysical Journal. 2015 Jan 27;108(2):610a-1a. . https://doi.org/10.1016/j.bpj.2014.11.3323 .
50. Kihara M, Kontani K, Yamauchi A, Miyauchi A, Nakamura H, Yodoi J, Yokomise H. Expression of thioredoxin in patients with Graves' disease. International journal of molecular medicine. 2005 May1;15(5):7959. https://doi.org/10.3892/ijmm.15.5.795

ORIGINAL RESEARCH
Published: 25 October 2025
DOI: 10.26505/djm.v29i1.1494

51. Najafi Z, Chamani E, Zarban A, Rezaei Z, Sharifzadeh G. The molecular evaluation of thioredoxin (TXN1 & TXN2), thioredoxin reductase 1 (TXNRd1), and oxidative stress markers in a binary rat model of hypo-and hyperthyroidism after treatment with gallic acid. Drug and Chemical Toxicology. 2023 Nov2;46(6):1108-15. https://doi.org/10.1080/01480545.2022.2131812
52. Alabdaly YZ. Effect of diclofenac on the pharmacokinetics of ciprofloxacin in quail. Iraqi Journal of Veterinary Sciences. 2021;35(4):777–81. https://doi.org/10.33899/ijvs.2021.128440.1576
53. Lu P, Huang H, Liu J, Cao Y, Hua Liu S, Yin J. Small Molecule Fluorescent Probes for Glutathione S-Transferase. ChemBioChem. 2025 May 5;26(9):e202400994. https://doi.org/10.1002/cbic.202400994
54. Hamdon AA, Al-Helaly LA. Biochemical and histopathological study of thioredoxin reductase isolation from blood serum in normal and oxidative stress-exposed rats. Iraqi Journal of Veterinary Sciences. 2019 Sep 1;33(2):115-24. https://doi.org/10.33899/ijvs.2019.163243
55. Abdulhameed OQ, Al-Helaly LA. Enzymatic Study of Methionine Sulfoxide Reductase A, some other Enzymes Associated with Neural Pathways and Oxidative Stress in Down Syndrome Patients in Mosul City. https://doi.org/10.33899/rjs.2024.185386
56. Cordiano R, Di Gioacchino M, Mangifesta R, Panzera C, Gangemi S, Minciullo PL. Malondialdehyde as a potential oxidative stress marker for allergy-oriented diseases: an update. Molecules. 2023 Aug 9;28(16):5979. https://doi.org/10.3390/molecules28165979
57. Jomova K, Alomar SY, Nepovimova E, Kuca K, Valko M. Heavy metals: toxicity and human health effects. Archives of toxicology. 2025 Jan;99(1):153-209. https://doi.org/10.1007/s00204-024-03903-2
58. Chandimali N, Bak SG, Park EH, Lim HJ, Won YS, Kim EK, Park SI, Lee SJ. Free radicals and their impact on health and antioxidant defenses: A review. Cell death discovery. 2025 Jan 24;11(1):19. https://doi.org/10.1038/s41420-024-02278-8
59. Olia BH, Khadem Ansari MH, Rasmi Y, Hasanzadeh-Moghadam M. Evaluation of malondialdehyde levels and total antioxidant capacity in patients with hyperthyroidism. Journal of Research in Applied and Basic Medical Sciences. 2019 May 10;5(2):121-7. http://ijrabms.umsu.ac.ir/article-1-86-en.html
60. Macvanin MT, Gluvic Z, Zafirovic S, Gao X, Essack M, Isenovic ER. The protective role of nutritional antioxidants against oxidative stress in thyroid disorders. Frontiers in endocrinology. 2023 Jan 4;13:1092837. https://doi.org/10.3389/fendo.2022.1092837
61. Gurina TS, Mohiuddin SS. Biochemistry, Protein Catabolism. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025 Jan–.
62. Roche M, Rondeau P, Singh NR, Tarnus E, Bourdon E. The antioxidant properties of serum albumin. FEBS letters. 2008 Jun 11;582(13):1783-7. https://doi.org/10.1016/j.febslet.2008.04.057
63. Xing Y, Yang L, Liu J, Ma H. The association with subclinical thyroid dysfunction and uric acid. International journal of endocrinology. 2021;2021(1):9720618. https://doi.org/10.1155/2021/9720618
64. Xing Y, Yang L, Liu J, Ma H. The association with subclinical thyroid dysfunction and uric acid. Int J Endocrinol. 2021;2021:9720618. DOI: https://doi.org/10.1155/2021/9720618.

Diyala Journal of Medicine

ORIGINAL RESEARCH
Published: 25 October 2025
DOI: 10.26505/djm.v29i1.1494

التغيرات في مستويات ميثيونين أدينوسيل ترانسفيريز، وميثيونين سولفوكساید ريدوكتيز A ، وثايوريدوكسين وعلاقتها بالإجهاد التأكسدي لدى مرضى فرط نشاط الغدة الدرقية

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الملخص

الخلفية: يرتبط فرط نشاط الغدة الدرقية بزيادة الإجهاد التأكسدي وحدوث اضطرابات في أنظمة مضادات الأكسدة الإنزيمية وغير الإنزيمية. يُعد إنزيم ميثيونين أدينوسيل ترانسفيريز (**MAT**) يلعب دورا حيويًا في عمليات الأيض الخلوي، وفي الحفاظ على التوازن الاكسدة والاختزال في اضطرابات الدرقية.

الأهداف: هدفت هذه الدراسة إلى تقييم مستويات **MAT** واستقصاء علاقته مع مجموعة من المؤشرات الإنزيمية وغير الإنزيمية للإجهاد التأكسدي لدى مرضى فرط نشاط الغدة الدرقية.

المرضى والطرق: تم جمع ٩٠ عينة مصل دم من مرضى مصابين بفرط نشاط الغدة الدرقية، ومقارنتها بـ ٥٠ عينة من أصحاء كمجموعة ضابطة. شملت المؤشرات الإنزيمية المقاسة: الميثيونين سلفوكساید ردكتيز A **(MsrA)**، الثايوريدوكسين **(Trx)**، الكتاليز **(Cat)**، المايلوبيروكسيديز **(MPO)**، اللاكتوبيروكسيداز **(LP)**، الزانثين أوكسيديز **(XO)**، الكلوتاثايون إس-ترانسفيريز **(GST)**، وبروتين علامة الشيخوخة-٣٠ **(SMP-30)**. أما المؤشرات غير الإنزيمية فقد تضمنت: الكلوتاثايون **(GSH)**، الحامض اليوريك **(UA)**، الألبومين **(Alb)**، المالونداياالديهايد **(MDA)**، وبيروكسينيترايت **(ONOO-)**.

النتائج: أظهرت نتائج المرضى ارتفاعًا معنويًا في مستويات MAT، وCat، وXO، وGST، وTrx، وLP، مقارنةً بالأصحاء، بينما سُجّل انخفاض معنوي في SMP-30. كما أظهرت المؤشرات غير الإنزيمية ارتفاعًا في MDA وONOO-، وانخفاضًا في مستوى الألبومين. لم تُسجّل تغيّرات معنوية في باقي المؤشرات. وقد أظهر MAT علاقة ارتباط مباشرة مع SMP-30، وMPO، وMsrA، وUA، وONOO-، وAlb، وعلاقة عكسية مع Cat، وXO، وGSH، بينما لم تُلاحظ علاقة ارتباطية بين MAT و GST، LP، أو Trx..

الاستنتاج: تشير النتائج إلى وجود ارتباط قوي بين فعالية MAT والإجهاد التأكسدي لدى مرضى فرط نشاط الغدة الدرقية. وتشير التغيرات الملحوظة إلى اختلالات أيضية وضعف آليات الدفاع المضادة للأكسدة لدى المرضى.

الكلمات المفتاحية: فرط نشاط الغدة الدرقية، إنزيم، ميثيونين أدينوسيل ترانسفيريز، ميثيونين سلفوكساید ريدكتيز **A** ، ثايوريدوكسين.

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تاريخ الاستلام: ٢٢ أيار ٢٠٢٥

تاريخ القبول: ١ أب ٢٠٢٥

تاريخ النشر: ٢٥ تشرين الاول ٢٠٢٥

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