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REVIEW ARTICLE
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A Review of Oxidative-Stress Biomarkers in Dromedary Camels for Prediction of Survival and Mortality

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ABSTRACT

Dromedary camels (*Camelus dromedarius*) are uniquely adapted to survive in extreme arid environments, yet remain vulnerable to various physiological and environmental stressors such as heat, dehydration, transport, and infectious diseases. These stressors disrupt cellular homeostasis and trigger oxidative stress—a pathological state resulting from an imbalance between reactive oxygen species and antioxidant defenses. Oxidative stress leads to molecular damage, affecting lipids, proteins, and DNA, which contributes to impaired physiological function and an increased mortality risk. In recent years, oxidative stress biomarkers such as malondialdehyde (MDA), superoxide dismutase, glutathione peroxidase, total antioxidant capacity, and protein carbonyls have gained attention for their potential utility in monitoring health status and predicting survival outcomes in camels. This review synthesizes current knowledge on oxidative stress mechanisms in camels and evaluates the prognostic significance of key oxidative biomarkers across a range of pathological conditions, including parasitic infections, metabolic disorders, gastrointestinal and urinary obstructions, and transport-related stress. Studies consistently report elevated MDA and depleted antioxidant enzymes in non-surviving camels, suggesting that these markers may be potential early indicators of critical illness. However, considerable variability in biomarker profiles exists due to differences in disease stage, sampling methods, and environmental influences. Despite these challenges, oxidative stress biomarkers show promise as non-invasive, practical tools for early detection of physiological compromise and mortality risk. Integration of oxidative profiling into herd health monitoring, along with the development of point-of-care diagnostic tools, could enhance camel healthcare, particularly in remote and resource-limited settings. This review highlights the need for standardized protocols, longitudinal studies, and advanced biosensing technologies to fully realize the diagnostic and prognostic potential of oxidative stress biomarkers in dromedary camel medicine.

Keywords: Camels, Biomarkers, Camel health management, Oxidative stress, Survival prediction

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INTRODUCTION

Dromedary camels (*Camelus dromedarius*) are uniquely adapted to thrive in harsh arid and semi-arid environments, where few other livestock species can survive (Masebo et al., 2023). These animals are not only integral to the socio-economic fabric of many desert and pastoral communities but also provide critical resources, including meat, milk, transportation, and cultural identity (Faye, 2015). Their physiological and anatomical adaptations—such as water conservation mechanisms, tolerance to extreme temperatures, and efficient metabolism—enable them to maintain functionality under environmental stressors that are otherwise debilitating to other species (Tharwat et al., 2013; El-Hady et al., 2023).

Despite their exceptional adaptability, camels remain susceptible to a range of environmental and physiological stressors (Hoter et al., 2019). Major challenges include prolonged dehydration, nutritional deficiencies, extreme heat, parasitic and infectious diseases, and long-distance transport under suboptimal conditions (Ali et al., 2023). These stressors can disrupt internal homeostasis, weaken immune responses, and increase the risk of morbidity and mortality (Hoter et al., 2019). Furthermore, the escalating

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impacts of climate change and desertification are amplifying these pressures, underscoring the urgent need for sensitive, early diagnostic tools capable of detecting health deterioration in camels before the onset of overt clinical symptoms (Ashour et al., 2024).

Oxidative stress arises from an imbalance between reactive oxygen species (ROS) production and the biological system's antioxidant defenses (Pooja et al., 2025). Excessive ROS can cause significant damage to cellular components, including lipids, proteins, and DNA, thereby impairing normal physiological function and promoting cell death (Afzal et al., 2023). In animals, oxidative stress is closely associated with various pathological conditions such as inflammation, infection, and metabolic dysfunction (Blanca et al., 2024). The study of oxidative stress is therefore essential for understanding how organisms respond to environmental and physiological challenges at the molecular level.

The evaluation of oxidative stress biomarkers—such as malondialdehyde (MDA), superoxide dismutase (SOD), glutathione peroxidase (GPx), and total antioxidant capacity (TAC)—represents a promising strategy for monitoring health status and predicting clinical outcomes in animals subjected to stress (Tufarelli et al., 2023). In dromedary camels, fluctuations in these biomarkers have been documented during episodes of dehydration, heat stress, and disease, supporting their utility as non-invasive indicators of physiological compromise (Tharwat & El-Deeb, 2021; Almundarij & Tharwat, 2023). Given that camel mortality can often occur abruptly. In the absence of clear preclinical signs, the capacity to monitor oxidative stress levels may significantly enhance early detection and intervention efforts, ultimately improving survival rates (Tharwat, 2023).

This review aims to synthesize current knowledge on oxidative stress biomarkers in dromedary camels and evaluate their potential role as predictors of survival and mortality. Emphasis is placed on the biological significance of key biomarkers, their responsiveness to various stressors, and the feasibility of integrating such measures into camel health management programs. By bridging the gap between oxidative stress research and practical applications in camel health monitoring, this review seeks to inform future research and contribute to the development of early diagnostic tools for this vital species.

Oxidative Stress: Biological Basis and Mechanisms

Definition and Generation of Reactive Oxygen Species

Oxidative stress arises from an imbalance between the production of ROS and the capacity of biological systems to detoxify these reactive intermediates or repair the resulting damage (Pizzino et al., 2017). ROS are chemically reactive molecules derived from oxygen, including free radicals such as superoxide anion, hydroxyl radical, and non-radical species such as hydrogen peroxide (Sies, 2017). These species are generated endogenously during mitochondrial oxidative phosphorylation, enzymatic reactions involving xanthine oxidase or NADPH oxidase, and through the activity of cytochrome P450 enzymes (Holmström & Finkel, 2014). Exogenous sources such as ionizing radiation, environmental pollutants, and toxins can also contribute to ROS generation (Birben et al., 2012).

Physiological Versus Pathological Roles of ROS

At physiological levels, reactive oxygen species (ROS) play vital roles in cellular signaling, immune defense, and the maintenance of homeostasis. Low concentrations of ROS are involved in regulating key cellular processes such as proliferation, apoptosis, and gene expression, primarily through the activation of redox-sensitive transcription factors (Schieber and Chandel, 2014). Additionally, phagocytic immune cells generate ROS during oxidative bursts as a defense mechanism against invading pathogens (Nathan & Cunningham-Bussel, 2013). However, when ROS production surpasses the capacity of the antioxidant defense system, oxidative stress arises, resulting in cellular damage and functional impairment. Chronic oxidative stress is implicated in the development and progression of numerous diseases, including cancer, neurodegenerative disorders, cardiovascular conditions, and inflammatory pathologies (Forman & Zhang, 2021). In animals—including camels—oxidative stress is increasingly recognized as a critical factor affecting resilience and survival, particularly under conditions of heat stress, dehydration, and infectious disease (Tharwat & El-Deeb, 2021).

Antioxidant Defense Systems

To mitigate the harmful effects of ROS, organisms have evolved complex antioxidant defense systems, encompassing both enzymatic and non-enzymatic components.

Enzymatic Antioxidants

The primary enzymatic antioxidants include superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx). SOD catalyzes the dismutation of superoxide anion into hydrogen peroxide, which is subsequently converted into water and oxygen by CAT, primarily in peroxisomes (Wang et al., 2018). GPx, a selenium-containing enzyme, reduces hydrogen peroxide and lipid peroxides using glutathione (GSH) as a substrate, thereby preventing lipid and protein oxidation (Brigelius-Flohé & Maiorino, 2013). These enzymes act synergistically to maintain redox homeostasis under both normal and stressed physiological conditions.

Non-Enzymatic Antioxidants

Non-enzymatic antioxidants include small molecules such as glutathione, vitamins C (ascorbic acid) and E (α-tocopherol), carotenoids, and flavonoids (Rudenko et al., 2023). Glutathione serves as a major intracellular antioxidant, directly scavenging ROS and functioning as a cofactor for GPx and glutathione reductase (GR) (Lubos et al., 2011). Vitamin C, a water-soluble antioxidant, neutralizes free radicals in the aqueous compartments of cells and regenerates vitamin E from its oxidized form (Traber and Stevens, 2011). Vitamin E, a lipid-soluble antioxidant, protects cell membranes from lipid

peroxidation by interrupting the chain reaction of lipid radical formation (Traber and Atkinson, 2007). Together, these antioxidants form a network that stabilizes cellular redox status and prevents oxidative damage.

Mechanisms of Oxidative Damage

Excessive ROS can inflict damage on lipids, proteins, and nucleic acids, leading to impaired cellular function and viability.

Lipid Peroxidation

Lipid peroxidation refers to the oxidative degradation of polyunsaturated fatty acids within cellular membranes, primarily initiated by ROS such as hydroxyl radicals (Su et al., 2019). This process results in the formation of reactive aldehydes, including malondialdehyde (MDA) and 4-hydroxynonenal, which compromise membrane integrity and form covalent adducts with proteins and nucleic acids, thereby amplifying cellular damage (Ayala et al., 2014). In dromedary camels, elevated MDA levels have been correlated with thermal stress and systemic inflammation, underscoring its value as a biomarker of oxidative injury and physiological stress (Tharwat and El-Deeb, 2021).

Protein Oxidation

ROS can modify protein structure and function by oxidizing amino acid side chains, forming carbonyl derivatives, disulfide bonds, or aggregating cross-linked proteins (Feng et al., 2022). Such modifications may result in enzyme inactivation, loss of receptor function, or increased protein degradation (Dalle-Donne et al., 2003). Protein oxidation is particularly detrimental in tissues with high metabolic rates or prolonged exposure to oxidative environments (Shields et al., 2021).

DNA Damage

ROS can induce a variety of DNA lesions, including strand breaks, base modifications, and the formation of 8-hydroxy-2′-deoxyguanosine (8-OHdG), a well-established marker of oxidative DNA damage (Valavanidis et al., 2009). Oxidative DNA damage can lead to mutations, genomic instability, and apoptosis, contributing to disease development and decreased survival (Chandimali et al., 2025).

Fig. 1 summarizes the major mechanisms through which ROS induce cellular damage. It outlines the specific macromolecular targets—lipids, proteins, and DNA—and the molecular events triggered by oxidative stress, such as lipid peroxidation, protein carbonylation and DNA strand breaks (Valavanidis et al., 2009; Su et al., 2019; Feng et al., 2022). The downstream consequences of these processes include membrane disruption, enzymatic inactivation, genomic instability, and ultimately, loss of cellular function and viability (Tharwat and El-Deeb, 2021).

Oxidative Stress in Dromedary Camels

Unique Physiological Adaptations of Camels to Oxidative Environments

Dromedary camels have evolved exceptional physiological mechanisms to thrive in harsh desert environments characterized by high temperatures, dehydration, and UV exposure (Hoter et al., 2019). These adaptations include controlled hyperthermia and water conservation, reducing evaporative cooling needs during extreme heat stress (Ali et al., 2023; Tharwat et al., 2023). Additional adaptations also include elevated baseline levels of antioxidant enzymes—such as SOD, CAT, and GPx—to combat lipid peroxidation and maintain cellular integrity (Ahmadpour and Zarrin, 2025; Tharwat and Al-El-Deeb, 2021). The liver plays a central role in modulating antioxidant defense during oxidative challenges, as demonstrated by coordinated upregulated mRNA expression of CAT and GPx during the peripartum period (Ali et al., 2023).

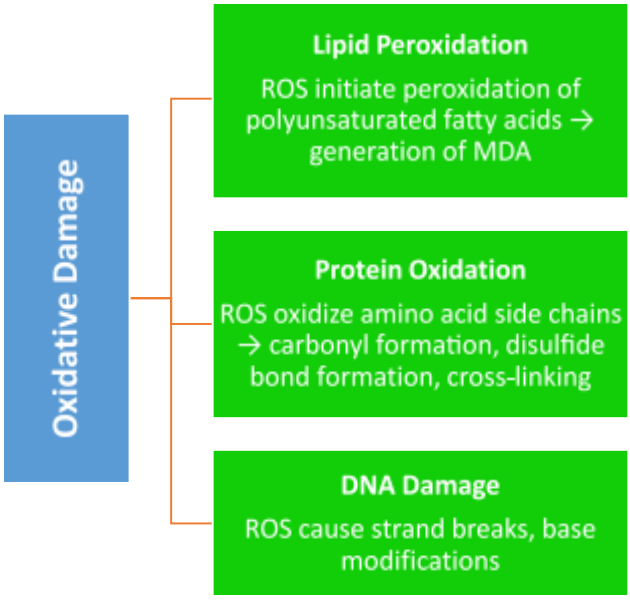

Fig. 1: Mechanistic pathways of oxidative damage induced by reactive oxygen species (ROS) and their implications in camels. MDA; malondialdehyde.

Baseline Oxidative and Antioxidant Profiles in Healthy Camels

In clinically healthy dromedary camels, plasma and serum analyses consistently reveal robust activity of key antioxidant enzymes such as GPx, SOD, and CAT, which collectively play a crucial role in maintaining oxidative balance (Tharwat and El-Deeb, 2021). The sustained activity of these enzymes indicates an efficient enzymatic defense system that effectively scavenges ROS, thereby preventing cellular damage and preserving physiological homeostasis (Pooja et al., 2025). For instance, SOD catalyzes the dismutation of superoxide radicals into hydrogen peroxide, which is subsequently broken down by CAT and GPx into harmless water molecules, minimizing oxidative injury (Tharwat and El-Deeb, 2021). This coordinated enzymatic interplay underscores the camel's evolved adaptive mechanisms to counter oxidative challenges, particularly given its harsh environmental exposure. Furthermore, camel blood analyses show stable concentrations of GSH, a vital non-enzymatic antioxidant, which reflects solid intracellular antioxidant reserves (Tharwat and El Sobayil, 2025). GSH serves multiple protective roles, including direct scavenging of free radicals and serving as a substrate for GPx activity. The preservation of GSH levels

suggests a well-maintained redox status and highlights the camel's capacity to regenerate and recycle antioxidants efficiently under physiological conditions. This resilience in antioxidant defense is likely essential for camels to cope with frequent oxidative stressors such as dehydration, heat exposure and variable nutrition, which are common in their native arid environments (Pooja et al., 2025).

Factors Influencing Oxidative Status in Camels

Elderly and overweight camel's exhibit decreased SOD and GSH activity and lower CAT levels, suggesting heightened vulnerability to oxidative imbalance (Almundarij, 2024). Pregnancy and lactation are also associated with elevated MDA and cortisol levels, and a protective compensatory increase in enzymatic antioxidants (GPx, CAT, GSH) (Abo El-Maaty et al., 2019). Seasonality (heat, humidity, forage scarcity) alters antioxidant-oxidant balance, weakening enzymatic defenses during the hot season (Tharwat and Al-Sobayil, 2025). Acute dehydration initially triggers oxidative stress and elevated MDA, but enzymatic antioxidants rebound upon rehydration (Ali et al., 2023). Transport and infections (e.g., trypanosomiasis, urinary tract obstruction) elevate oxidative stress biomarkers (MDA, cortisol) and suppress antioxidant enzymes, underscoring the camel's physiological vulnerability under pathological stress (Tharwat and Al-Sobayil, 2025).

Biomarkers of Oxidative Stress in Camels

A comprehensive range of molecular and enzymatic biomarkers serves as indicators of oxidative stress in dromedary camels across various physiological and pathological conditions. These biomarkers encompass not only the direct products of oxidative damage but also the dynamic status of the antioxidant defense system (Tharwat and Al-Sobayil, 2025). For instance, lipid peroxidation markers such as MDA reflect the degree of oxidative damage to cell membranes, which can compromise cellular integrity and function. Similarly, protein carbonyl content serves as a reliable indicator of oxidative modification to proteins, which can impair enzymatic activity and structural stability (Mohideen et al., 2023). DNA oxidation products, including 8-OHdG, provide insight into genotoxic stress that may predispose cells to mutations and impaired replication (Blanca et al., 2024). In parallel, enzymatic antioxidants such as GPx, SOD and CAT are frequently assessed to gauge the organism's capacity to counteract ROS (Pooja et al., 2025). Changes in these enzyme activities can reveal compensatory upregulation or depletion of the antioxidant system depending on the severity and duration of oxidative insults. The interplay between these markers offers a holistic understanding of oxidative stress status, enabling differentiation between adaptive responses during physiological stress—such as exercise or dehydration—and maladaptive responses observed in pathological states like infections or metabolic disorders (Tharwat and Al-Sobayil, 2025). Such biomarker profiling is critical in elucidating the camel's unique oxidative stress responses, which likely contribute to its resilience in extreme environments. By systematically assessing both oxidative damage and antioxidant defense, researchers can better understand the molecular mechanisms underpinning health and disease in camels, guiding improved management and therapeutic strategies (Tharwat and Al-Sobayil, 2025).

Lipid Peroxidation Products

Markers such as MDA and thiobarbituric acid reactive substances (TBARS) reflect the peroxidation of cell membrane lipids (Mohideen et al., 2023). Infested camels demonstrate significantly elevated erythrocytic MDA levels compared with healthy controls, highlighting oxidative damage to erythrocyte membranes (Saleh et al., 2011). Camels with hepatic echinococcosis also show increased serum MDA concurrent with altered hepatic function (Heidarpour et al., 2012).

Protein Oxidation Markers

Oxidative modification of proteins is detectable via protein carbonyls and advanced oxidation protein products (AOPPs) (Gryszczyńska et al., 2017). Saleh and colleagues reported elevated protein carbonyl concentrations in camels with sarcoptic mange, correlating with infestation severity, and reduced total protein and albumin (Saleh et al., 2011).

DNA Oxidation Markers

8-OHdG is widely recognized as a marker of oxidative DNA damage (Valavanidis et al., 2009). Although studies are limited in camels, urinary 8-OHdG increases under xenobiotic-induced oxidative stress in other animals (Blanca et al., 2024). This suggests its potential applicability in camels under heat or disease stress.

Enzymatic Antioxidants

Key antioxidant enzymes include SOD, CAT, and GPx (Tharwat, 2023). In mange-infested camels, SOD and CAT activities initially increase in mild cases but decline in severe infestation stages, while GPx follows a similar downward trend as stress intensifies (Saleh et al., 2011). Around parturition, dromedary camels show significant fluctuations in hepatic CAT, SOD, and GPx, reflecting dynamic shifts in oxidative defenses during the transition period (Ahmadpour and Zarrin, 2025).

Non-enzymatic Antioxidants

Reduced GSH and vitamins E and C serve as vital non-enzymatic antioxidants (Rudenko et al., 2023). Infested camels exhibit lower levels of serum ascorbate and GSH, particularly in moderate and severe disease stages (Saleh et al., 2011). Vitamin E status has also been reported to decline during dehydration and stress in pregnant or lactating camels (El-Deeb et al., 2022).

Total Antioxidant Capacity (TAC)

Measures such as total antioxidant capacity (TAC), total oxidant status (TOS) and oxidative stress index (OSI) (OSI = TOS/TAC) offer integrated assessments of oxidative balance (Admasu et al., 2022). Camels with echinococcosis exhibit reduced TAC alongside elevated MDA and lowered

GPx activity (Heidarpour et al., 2012). Similarly, racing-related lameness in camels is associated with lowered TAC and altered oxidative stress profiles (Faye and Bengoumi, 2018). During dehydration, camels show modest changes in TAC, indicating robust rehydration-associated recovery of antioxidant reserves (Ali et al., 2023; Tharwat and Al-Sobayil, 2025).

Fig. 2 illustrates the major categories of oxidative stress biomarkers in camels, grouped into oxidative damage markers, antioxidant defense markers, and integrated oxidative stress indices. Oxidative damage is reflected through lipid peroxidation products such as MDA and TBARS, protein oxidation markers including protein carbonyls and AOPPs, and DNA oxidation markers like 8-OHdG. Antioxidant defenses are divided into enzymatic antioxidants—such as SOD, CAT and GPx—and non-enzymatic antioxidants like GSH, vitamin C, and vitamin E. Integrated indices, including TAC, TOS and the OSI, provide an overall measure of oxidative balance.

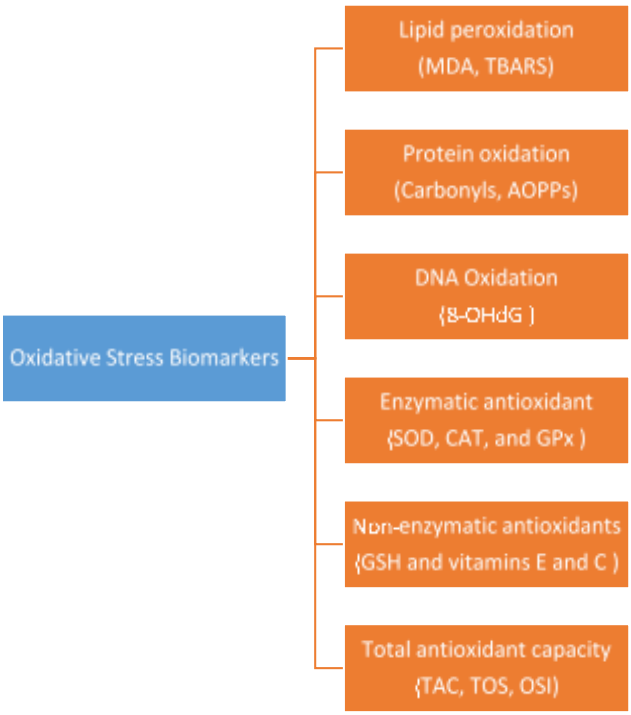

Fig. 2: Major categories of oxidative stress biomarkers in camels, grouped into oxidative damage markers, antioxidant defense markers, and integrated oxidative stress indices. Oxidative damage is reflected through lipid peroxidation products such as malondialdehyde (MDA) and thiobarbituric acid reactive substances (TBARS), protein oxidation markers including protein carbonyls and advanced oxidation protein products (AOPPs), and DNA oxidation markers like 8-hydroxy-2'-deoxyguanosine (8-OHdG). Antioxidant defenses are divided into enzymatic antioxidants—such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx)—and non-enzymatic antioxidants like reduced glutathione (GSH), vitamin C, and vitamin E. Integrated indices, including total antioxidant capacity (TAC), total oxidant status (TOS), and the oxidative stress index (OSI), provide an overall measure of oxidative balance.

Role of Oxidative Stress in Camel Diseases and Stress Conditions

Oxidative Stress in Parasitic and Bacterial infections

Camel trypanosomiasis (caused by *Trypanosoma evansi*) provokes pronounced oxidative damage, evidenced by elevated MDA levels and depleted antioxidant defense such as SOD, CAT, GSH, and albumin (Saleh et al., 2009; El-Bahr and El-Deeb, 2016) (Fig. 3). Hemoglobin oxidation, reflected by increased methemoglobin, contributes to anemia (Saleh et al., 2009). Furthermore, El-Deeb and Elmoslemany (2015) demonstrated that cardiac markers (cTnI, CK-MB) and serum MDA were significantly elevated in infected camels, while SOD activity dropped—changes which normalized in successfully treated animals, indicating predictive utility for prognosis. Given camel analog data from paratuberculosis, one anticipates similar antioxidant depletion and lipid peroxidation accompanying viral pathogenesis (El-Deeb et al., 2014) (Fig. 4). In camels affected by sarcoptic mange, MDA levels remain comparable to healthy controls in mild cases but show significant elevation in moderate and severe infections. Compared to healthy camels, SOD and catalase activities are significantly increased in mild cases but markedly reduced in moderate and severe stages. A similar pattern is observed for GSH levels, which are elevated in mild cases and significantly decreased in more advanced stages of the disease (Saleh et al., 2011) (Fig. 5).

Metabolic and Urinary and Gastrointestinal Disorders

While direct data in camels remain scarce, metabolic stress (e.g., ketosis, fatty liver) and reproductive disorders often involve oxidative imbalance (Tharwat and Al-Sobayil, 2025). In dromedaries, dehydration-induced anabolic shifts include modulation of fat metabolism and stress hormone pathways (Ali et al., 2023), often accompanied by elevated MDA and compensatory increases in antioxidant vitamins. In camels with gastrointestinal and urinary obstructions, MDA levels did not differ significantly between the two groups. However, GSH levels were significantly lower in camels with intestinal obstruction compared to the control group. Similarly, SOD levels were also significantly reduced in camels with intestinal obstruction relative to healthy controls. In contrast, no significant differences in GSH and SOD levels were observed between camels with urinary obstruction and the control group. Additionally, CAT levels showed no significant variation among the control and both diseased groups (Almundarij and Tharwat, 2023) (Fig. 6).

Environmental and Physiological Stressors

Transport and Handling Stress

Transport stress in camels, similar to other livestock species, triggers a cascade of physiological and biochemical responses characterized by elevated circulating catecholamines and cortisol, key hormones involved in the stress response (El Khasmi et al., 2015). These hormonal surges not only prepare the animal for acute stress by mobilizing energy reserves but also inadvertently increase the production of ROS, contributing to oxidative stress. This is evidenced by the documented increase in MDA levels, a reliable biomarker of lipid peroxidation, which reflects enhanced oxidative damage to cellular membranes during transport. Concurrently, a reduction in the overall antioxidant capacity suggests that the camel's endogenous defense mechanisms are overwhelmed or depleted under prolonged or intense

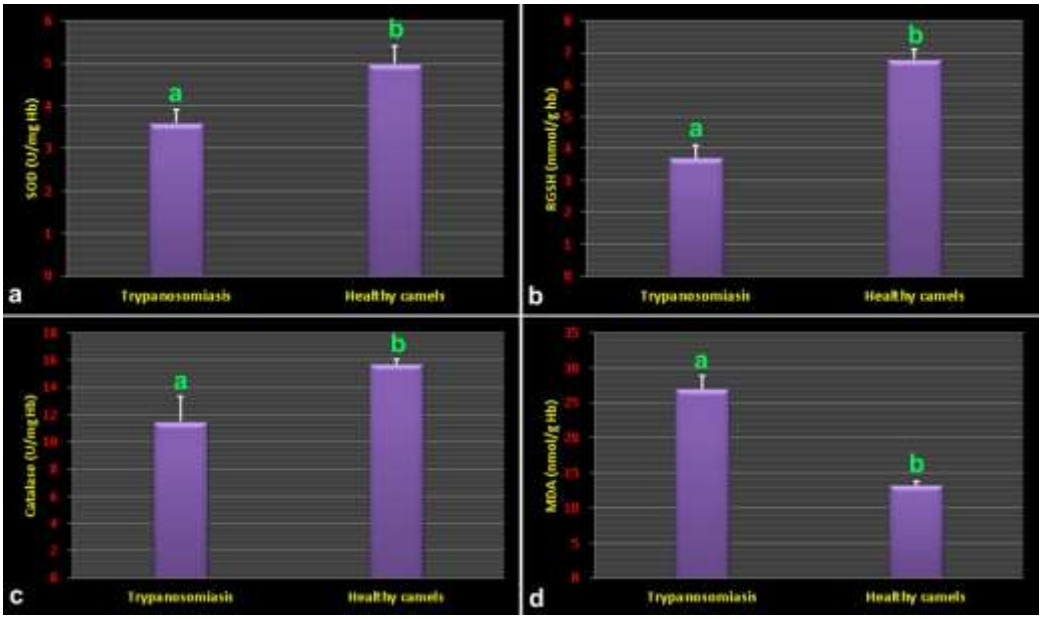

Fig. 3: Mean values of the oxidative stress biomarkers superoxide dismutase (SOD) (**a**), reduced glutathione (RGSH) (**b**), catalase (**c**) and malondialdhyde (MDA) (**d**) in camels with trypanosomiasis compared to healthy camels (Modified from El-Bahr and El-Deeb, 2016).

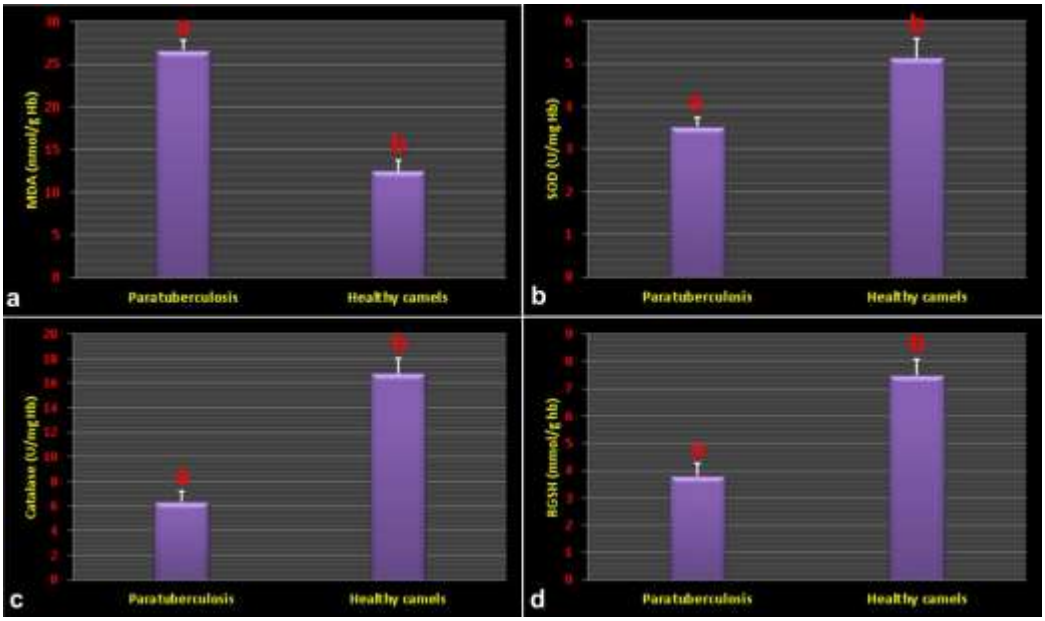

Fig. 4: Mean values of the oxidative stress biomarkers superoxide dismutase (SOD) (a), catalase (b), malondialdhyde (MDA) (c) and reduced glutathione (RGSH) (d) in camels with paratuberculosis compared to healthy animals (Modified from El-Deeb et al., 2014).

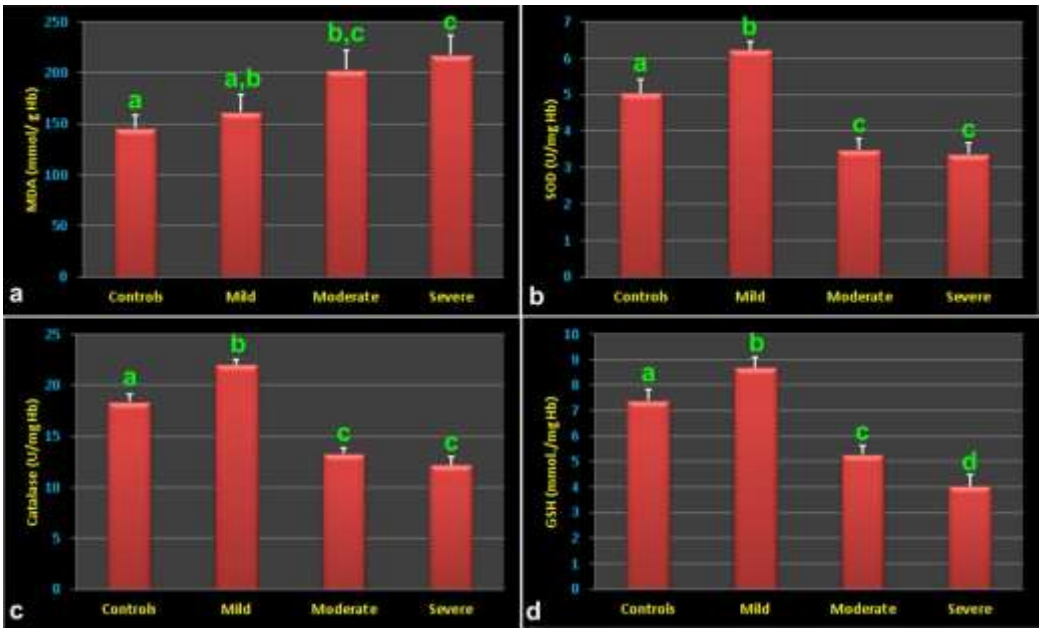

Fig. 5: Mean values of the oxidative stress biomarkers malondialdhyde (MDA) (**a**), superoxide dismutase (SOD) (**b**), catalase (**c**) and glutathione (GSH) (**d**), and in camels with sarcoptic mange compared to healthy camels. [a,b,c,d]Differ significantly at $P \leq 0.05$ (Modified from Saleh et al., 2011).

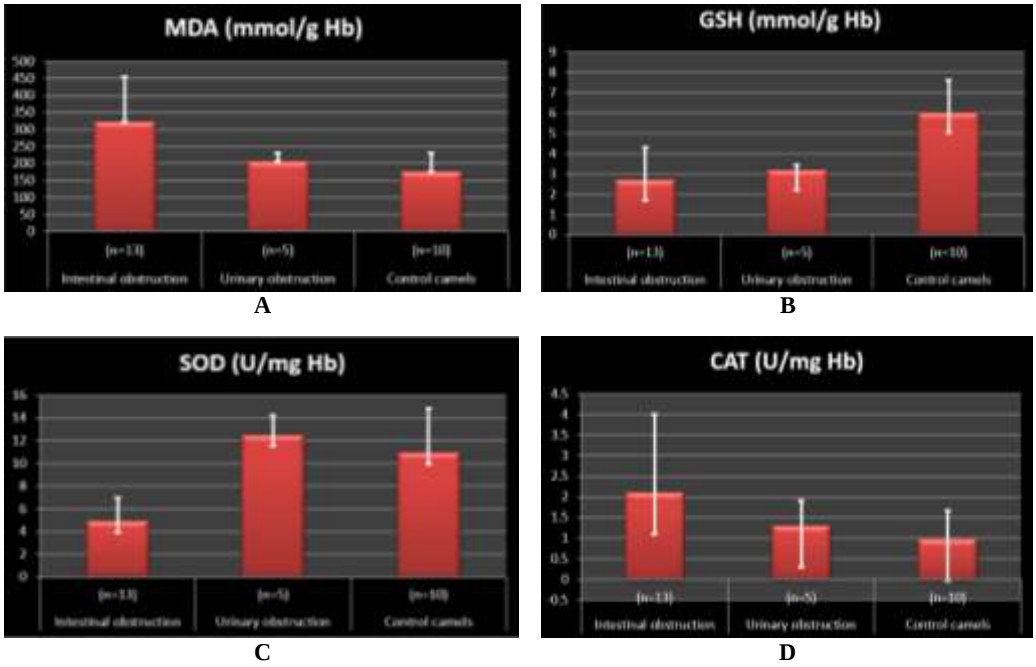

Fig. 6: Means±SD of the stress biomarker malondialdehyde (MDA, P=0.32), glutathione (GSH, P=0.008), superoxide dismutase (SOD, P=0.0001), and catalase (CAT, P=0.45) in camels with obstructions of the gastrointestinal and urinary tracts compared to controls (Modified from Almundarij and Tharwat, 2023).

stress conditions. Moreover, the observed rise in cardiac-specific biomarkers such as CK-MB and troponin I in transported camels points to possible myocardial injury mediated by oxidative stress (El-Deeb and Elmoslemany, 2015). These enzymes, normally confined within cardiac muscle cells, are released into circulation when oxidative damage compromises cellular membranes, indicating early signs of cardiac tissue stress or injury. This highlights a critical link between transport-induced physiological stress and oxidative damage at the organ level, underscoring the vulnerability of cardiac tissue to stress-related ROS accumulation. Such findings emphasize the need for improved handling and transport protocols to mitigate oxidative damage and protect animal welfare, which could ultimately enhance camel health and productivity in husbandry systems (Tharwat and Al-Sobayil, 2025).

Heat Stress and Dehydration

Prolonged water deprivation induces oxidative stress across kidney, stomach, and gastric mucosa, marked by elevated MDA, GSH, and CAT, while SOD becomes depleted (Ali et al., 2023). Dehydration-induced oxidative signals are accompanied by inflammatory markers (IL-1β, IL-18), apoptosis, and tissue vacuolization, which are largely reversible upon rehydration (Ali et al., 2019).

Oxidative Patterns in Acute vs. Chronic Disease Conditions

Acute infections or stressors in dromedary camels typically precipitate rapid and pronounced surges in ROS, MDA, and pro-inflammatory cytokines, reflecting an immediate oxidative and inflammatory response aimed at combating the insult (Tharwat and Al-Sobayil, 2025). This acute phase is often characterized by a transient decline in the activity of key antioxidant enzymes such as SOD and CAT, likely due to their consumption in neutralizing the sudden increase in ROS (Ali et al., 2019). The imbalance created during this acute oxidative burst can lead to cellular damage if not promptly controlled, highlighting the delicate interplay between pro-oxidant forces and antioxidant defenses.

In contrast, chronic conditions such as parasitic infestations or prolonged dehydration induce a more sustained oxidative challenge. These states are marked by persistent elevations in lipid peroxidation markers and inflammatory mediators, indicative of ongoing cellular and tissue stress. Interestingly, camels appear to mount a compensatory response in these chronic scenarios by upregulating specific antioxidants like GR and maintaining elevated levels of GSH, which work to buffer the prolonged oxidative load (Afzal et al., 2023). This adaptive mechanism suggests a degree of plasticity in the camel's antioxidant system, allowing it to adjust to long-term oxidative pressures. Recovery dynamics following oxidative insults also differ significantly between acute and chronic stress. Acute oxidative stress often resolves swiftly with appropriate treatment—such as antimicrobial therapy or rehydration—allowing antioxidant enzyme activities and redox balance to normalize (Ali et al., 2019). However, chronic oxidative damage tends to persist, necessitating longer recovery periods and potentially targeted therapeutic interventions aimed at restoring redox homeostasis and mitigating long-term tissue damage (Tharwat and Al-Sobayil, 2025). This distinction underscores the importance of early detection and management of oxidative stress to prevent progression to chronic pathology and optimize health outcomes in camels.

Predictive Value of Oxidative Biomarkers for Survival and Mortality

Differences in Oxidative Profiles between Survivors and Non-Survivors

Oxidative stress biomarkers exhibit significant differences between surviving and non-surviving dromedary camels during critical illness or systemic inflammatory conditions (Tharwat, 2023). Survivors typically maintain a more balanced redox status,

characterized by lower levels of ROS and lipid peroxidation products such as MDA, alongside higher concentrations of endogenous antioxidants including SOD, CAT, and reduced GSH (Tharwat and Al-Sobayil, 2025). In contrast, non-survivors tend to show severe oxidative derangements with diminished antioxidant reserves, suggesting a failure in redox homeostasis and a predisposition to oxidative tissue injury. These findings underscore the prognostic implications of OS biomarkers in camel medicine (Faye and Bengoumi, 2018).

Prognostic Biomarkers Correlated with Poor Outcomes

Among various oxidative markers, MDA, nitric oxide (NO), and protein carbonyls have been most consistently associated with poor clinical outcomes (Tharwat and Al-Sobayil, 2025). Elevated MDA levels, indicative of enhanced lipid peroxidation, are commonly observed in non-survivors and are reflective of widespread cellular damage (Saleh et al., 2009). Similarly, increased NO levels can reflect not only oxidative imbalance but also the presence of systemic inflammation, particularly in septic or dehydrated camels (Ali et al., 2023). Depletion of antioxidant enzymes such as SOD and GPx has also been inversely correlated with survival, indicating their protective role against oxidative organ dysfunction (Tharwat and Al-Sobayil, 2025).

Limitations and Variability among Current Studies

Despite encouraging findings, several limitations hinder the widespread application of oxidative stress biomarkers in camel medicine (Tharwat, 2023). Notable inter-study variability exists regarding assay techniques, sample collection timing, and population characteristics, making cross-study comparisons challenging. Additionally, the absence of large-scale, longitudinal studies restricts the generalizability and clinical relevance of current data (Tharwat and Al-Sobayil, 2025). Environmental influences—such as ambient temperature, hydration status, and nutrition—may further confound biomarker levels, underscoring the need for controlled investigations to enhance their diagnostic and prognostic reliability (Tharwat and El-Deeb, 2021). Until these limitations are systematically addressed, oxidative stress biomarkers should be interpreted with caution and always considered within the broader clinical and environmental context.

Future Directions and Research Gaps

While current findings highlight the prognostic relevance of oxidative-stress biomarkers in dromedary camels, significant opportunities remain for advancement. Future research should prioritize the discovery and characterization of novel oxidative biomarkers, such as advanced oxidation protein products and 8-isoprostane, which may offer improved specificity and sensitivity in clinical contexts (Faye and Bengoumi, 2018). Integrating oxidative biomarkers with multi-omics platforms—including proteomics, transcriptomics, and metabolomics—could enhance our understanding of the complex biological networks underlying disease progression and survival outcomes in camels (Tharwat and Al-Sobayil, 2025). In addition, the development of point-of-care testing devices and portable biosensors suitable for use in remote desert settings is essential to translate laboratory findings into practical tools for field veterinarians (Tharwat, 2023). Finally, incorporating oxidative-stress profiling into breeding programs and herd health-monitoring systems may provide new avenues for selecting animals with superior resilience to oxidative challenges, thereby improving overall productivity and disease resistance (Tharwat and El-Deeb, 2021). Addressing these research gaps will not only refine our prognostic capabilities but also contribute to sustainable camel health management in arid regions.

Conclusion

Dromedary camels exhibit exceptional physiological adaptations that allow them to endure extreme environmental conditions; however, they remain vulnerable to various oxidative stressors, including dehydration, heat, transportation, and infectious diseases. Biomarkers of oxidative stress—such as MDA, SOD, GPx, and TAC—have shown significant promise as indicators of physiological distress, disease severity, and prognostic outcomes in camels. Emerging evidence links elevated oxidative damage with increased mortality risk, with non-surviving camels often displaying profound disruptions in redox homeostasis and severely diminished antioxidant defenses. These findings highlight the potential of oxidative biomarkers not only as diagnostic indicators but also as early predictors of clinical outcomes. Nevertheless, substantial variability across studies and the absence of standardized methodologies currently limit their clinical applicability. Future research should prioritize the refinement of biomarker assays, establish validated prognostic thresholds across diverse camel populations, and integrate oxidative stress profiling into comprehensive health-monitoring frameworks. The advancement of portable diagnostic tools and field-deployable biosensors will also be essential for translating these insights into practical, real-world applications, particularly in desert and pastoral environments. Ultimately, oxidative stress biomarkers represent a promising frontier in camel health management, offering new avenues for early intervention, improved survival prediction, and enhanced resilience to environmental and pathological challenges.

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