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A review on oxidative stress in small ruminants induced by helminthes

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Abstract
In this paper, the most recent findings about the oxidative stress that helminth parasites cause in small ruminants like sheep, goat are reviewed. As a by-product of regular cell metabolism, an imbalance between the production of oxidant and antioxidant components results in oxidative stress. Many of these molecules are essential components of numerous metabolic pathways. Numerous Reactive Oxygen Species (ROS) are produced by the body, but these are countered by an antioxidant defence mechanism that keeps the concentrations of these oxidising substances below tolerable limits. There are a few reasons why this is the case. Helminth infection is a contributing factor. The most widespread and economically significant infectious illness in the world, gastrointestinal helminth infections kill millions of animals each year, mostly cattle, sheep and goats. ROS causes lipid peroxidation, protein oxidation, gene expression alterations, redox status changes, certain diseases, and accelerated aging in small ruminants.

Keywords: Oxidative stress, helminth parasites, small ruminants, reactive oxygen species

1. Introduction
Oxidative stress is a matter of concern in the ruminant health, particularly after helminth infection. The immunological response may be initiated and, in most cases, increases in production of reactive oxygen species (ROS), (Aktas *et al*. 2017) [3]. Oxidative stress occurs with a disrupted capacity to limit the negative consequences of ROS production (Esmaeilnejad *et al*. 2012) [27]. Small ruminants, like sheep and goats, are especially prone to helminth infections, which may result in livestock farmers to suffer significant financial losses. Intestinal parasites particularly nematodes, are quite common. The prevalence can vary from 70% to 100% in some regions. In addition to directly harming the host's tissues, these parasitic infections set off a series of physiological and metabolic problems that may affect the animal's general health (Turrens 2004) [78]. A long-standing tradition among indigenous populations in underdeveloped countries, small ruminant farming is becoming more difficult due to a number of biotic and environmental conditions, particularly gastrointestinal parasites, which have a substantial influence on farmer incomes (Hoste *et al*. 2007) [37]. Numerous pathological alterations brought on by helminthiasis, such as swelling, nutrient malabsorption, and anemia, might eventually hinder development rates, lower reproductive performance, and make a person more vulnerable to subsequent infections (Turrens 2004) [78]. A number of environmental factors, including humidity, weather, precipitation, grazing conditions and management techniques, influence the prevalence of gastrointestinal parasites in cattle [78]. Together, these elements influence the dynamics of infectious larvae's survival, growth, and transmission, which in turn affects the total burden of parasitic diseases in livestock (Turrens 2004) [78].
The complex relationship between small ruminant reproductive efficiency and parasite infections highlights the complex effects of helminth invasions on the physiological processes of animals. Helminth infections are common and can have major impact on animal health, production and welfare in ruminants, including cattle, sheep and goats.

These parasites stimulate the recognition of the immune system of the host by certain natural immune cells, like neutrophils and macrophages, to purchase natural defense products including ROS in the host's immune defense. Usually, these parasites prefer to live in gastrointestinal tract, liver or lungs of the host, causing tissue damage and inflammation (Lushchak 2008) [50].

2. Helminth-induced oxidative stress in ruminants

In ruminant helminthic infections, significant oxidative stress occurs those results in an imbalance in the ability of the body to quench Reactive Oxygen Species (ROS) and in the generation of these species (Selkirk *et al*. 1998; Turrens 2004) [70, 78]. Adverse effects of this oxidative stress on the health and productivity of ruminants. Infection of helminths elicits an activated response by the immune cells, generating more of the ROS, activated by these cells (Hoste *et al*. 2007) [37]. The impacts of oxidative stress caused by helminth parasites in the host's body are depicted in the accompanying Figure 1.

Oxidation of cell membranes resulting in disruption of integrity and capacity of functionality. Immune responses to others infections are suppressed by chronic oxidative stress and the host is more susceptible to other infections (Lightbody *et al*. 2001) [47]. The antioxidant systems in ruminant are both enzymatic (such as glutathione peroxidase, catalase and superoxide dismutase) and non-enzymatic (such as glutathione, vitamins C and E). During helminth infections, these defence mechanisms may be overtaken to cause oxidative stress (Torres-Acosta and Hoste 2008) [37]. Treatment for anthelmintic reducing parasites burden and related oxidative stress. Antioxidants supplementation in the diet as a means to boost the host's defence mechanisms. Selective breeding of animals having higher antioxidant capacity and resistance to parasite (Golden *et al*. 2010) [31]. Investigation of new antioxidant interventions and their effectiveness in combating the effects of helminth infections. To investigate the relationship between components of production metrics and those of oxidative stress markers in order to develop early intervention diagnostic tools.

3. Oxidative stress Biomarkers in small ruminants

The unstable production of reactive oxygen species and the capacity of biological systems to detoxify these reactive intermediates or to repair the visible damage constitutes a serious challenge to the health and productivity of ruminants, so-called oxidative stress. The physiological perturbation is caused due to the inherent decomposition of oxygen during aerobic respiration and results in a long-term state in which the formation of reactive oxygen species exceeds the natural capacity of the antioxidant system (Moldogazieva *et al*. 2019) [57]. After that, this imbalance damages cells and can lead to a variety of disease problems, including ruminant reproductive, immunological and metabolic dysfunction. Overproduction of reactive oxygen species can oxidatively alter vital macromolecules, such as lipids and proteins and nucleic acids, changing their chemical structure and biological functions (Sharma *et al*. 2012) [71]. Because living things are constantly exposed to both endogenous and exogenous reactive oxidants, they must evolve strong antioxidant defense mechanisms to prevent cell damage (Alam *et al*. 2020) [4]. The incidence of oxidative stress is closely linked to lipid peroxidation, an important marker of cell damage (Maizels *et al*. 2004) [54]. The high concentrations of 4-hydroxynonenal (4-HNE) and malondialdehyde which are common lipid peroxidation byproducts, as potent indicators for diagnosis that can be utilized to assess ruminant oxidative stress (Makkar *et al*. 2007) [55]. Approach of assessing reactive oxygen species' physiological impacts accumulation and the effectiveness of antioxidant reactions is also provided by the examination of these particular oxidative stress markers, including aldehydes (Cui *et al*., 2018) [20]. To properly evaluate the effects of oxidative stress in bovine physiology, more biomarkers are available. Additionally, 8-hydroxy-2'-deoxyguanosine and protein carbonyls are used to examine the oxidative stress levels found in DNA and proteins, respectively. When developing therapies to enhance the well-being and productive efficiency of ruminants, the detection and assessment associated biomarkers is essential. The accurate measurement of these antioxidant producing markers is actually necessary for the development of specific nutritional therapies and management techniques that increase ruminants' resilience to physiological and environmental stressors (Aramouni *et al*. 2023) [6]. Understanding the mechanisms driving the disparity between the anti-oxidant and antioxidant mechanisms is essential for developing diagnostic and treatment approaches. To more accurately determine a ruminant's oxidative status, new diagnostic techniques must be developed because existing biomarkers are occasionally inadequate detectable and specific (Tsubokawa 2023) [77]. In turn, there is a growing interest in identifying and certifying new biomarkers which capture the subtlety of oxidative stress, such as genetic and epigenetic biomarkers, to give a more detailed and timely assessment of the oxidative load on an animal. In ruminants, oxidative stress has been connected to a number of pathological conditions, including as mastitis, pneumonia and metabolic disorders, which significantly impact both overall health and productivity (Celi 2010) [13]. Oxidative stress is specifically characterized by an imbalance in the availability of oxidants and antioxidants, leading to adverse effects such as membrane lipid peroxidation and protein and enzyme degradation. Reactive oxygen species are produced at particularly high metabolic rates in high-producing ruminants, which eventually reduces their physiological integrity and productive longevity (Ceciliani *et al*. 2018) [12]. The purpose of the review is to synthesize the existing information on the use of multiple diagnostic and biomarker signs of oxidative stress in ruminants and investigate its potential to identify health conditions and susceptibility to disease. It will also look at the possible uses of cutting-edge-omics technologies like proteomics and metabolomics in order to gain a greater awareness of the oxidative stress response and find new biomarkers in ruminants (Ceciliani *et al*. 2018) [12].

3.1 Lipid peroxidation products produced by oxidative stress

Oxidative stress, which causes cellular damage and a variety of illnesses, is largely caused by lipid peroxidation. Reactive oxygen species (ROS) and free radicals are responsible for the oxidative degradation of lipids, particularly polyunsaturated fatty acids (PUFAs) found in cell membranes (Jarikre *et al*. 2019) [42].

Lipid peroxidation affects various functions on cellular level. In membrane damage it alters membrane fluidity, permeability and integrity affecting cellular homeostasis (Kamel *et al*. 2018) [43]. There are three different stages to this process. Lipid peroxidation produces reactive aldehydes that can form DNA adducts, which could lead to mutations. Reactive oxygen species (ROS) are produced at a higher rate and energy synthesis is disturbed (Sheng *et al*. 2014) [72].

Furthermore, products of lipid peroxidation can activate pro-inflammatory signaling pathways. Apoptosis or necrosis may happen in cases of severe lipid peroxidation. The feedback loop that lipid peroxidation produces may increase cellular damage by either causing or resulting from oxidative stress. Moreover, the wide range of lipid peroxidation byproducts, including both simple aldehyde and polymeric compounds, is known to have a cytotoxic and genotoxic effect, and these compounds help to cause extensive tissue damage. Among them, malondialdehyde and 4-hydroxynonenal (4-HNE) are the most important ones since with their small size, they can easily diffuse through the biological membranes and serve as secondary messengers, which further damage the DNA. These reactive aldehydes, such as 4-hydroxynonenal (4-HNE) and malondialdehyde, are products of the breakdown of lipid hydroperoxides, particularly of the peroxidation of polyunsaturated fatty acids such as arachidonic acid, which is very abundant in cell membranes (Aebi 1984) [1].

3.2 Protein oxidation markers in oxidative stress

To evaluate oxidative stress and its impact on cellular function, protein oxidation markers are required. By illustrating protein oxidative damage, these indicators offer vital information on the general oxidative state of biological systems (Lubos *et al*. 2011) [49]. Protein oxidation markers, which serve as biomarkers of oxidative stress within cells, show the equilibrium between oxidant generation and antioxidant defense capabilities. Numerous degenerative conditions are associated with elevated levels of these markers. There are many uses for protein oxidation indicators, including as tracking the development of conditions associated with oxidative stress, assessing the effectiveness of antioxidant therapies, and determining oxidant intake in environmental and industrial contexts (Couto *et al*. 2016) [19]. Cellular processes may be able to restore some signs of protein oxidation. The efficacy of oxidized proteins as biomarkers may vary depending on their stability and half-life (Forman *et al*. 2009) [30]. To make cross-study comparisons easier, measurement methods must be standardized. Understanding the function of protein oxidation biomarkers in oxidative stress is necessary to clarify the mechanisms that result in cellular damage and to create targeted treatments to minimize oxidative stress related disorders (Pivoto *et al*. 2015) [62].

3.3 DNA/RNA oxidation markers in oxidative stress

Indicators that gauge the oxidation of DNA and RNA are the main tool for evaluating oxidative stress and its effects on cellular processes. These markers are useful for assessing the general oxidative state of biological systems and offer information on the degree of oxidative degradation of nucleic acids (Forman *et al*. 2009) [30]. The study of cellular aging and age-related disorders benefits greatly from the use of DNA and RNA oxidation markers. Cellular processes may be able to repair some of the byproducts created by the oxidation of DNA and RNA. Moreover, oxidized nucleic acids' half-lives and stability might differ greatly (Saleh 2008) [68]. Therefore, a thorough assessment of these particular indicators is essential to accurately assessing an organism's overall oxidative stress environment. The issue is particularly acute in small ruminants, where the deleterious effects of oxidative stress on essential biological macromolecules such as proteins, lipids, DNA, and RNA can significantly reduce animal productivity and well-being (Forman *et al*. 2009) [30]. Numerous negative consequences result from this type of cellular damage, such as a lower feed conversion ratio, compromised immunity, and higher death rates. In this sense, precise assessment of oxidative stress markers is crucial for the development of successful therapies meant to lessen such detrimental effects and enhance animal welfare in general. As a means of evaluating oxidative stress in ruminants, hemoglobin biomarkers have taken center stage, frequently linking their alterations to other conditions like metabolic or reproductive disorders (Forman *et al*. 2009) [30].

3.4 Redox status markers in oxidative stress

Oxidative stress and its effects on cellular function are evaluated using indicators of DNA/ribonucleic acid (RNA) oxidation. These indicators can show the degree of oxidative damage to nucleic acids and assess the general oxidative condition of biological systems (Selkirk *et al*. 1998) [70]. Thus, the biomarkers are essential for accurately determining the level of oxidative stress and its physiological repercussions in small ruminants. The imbalance between oxidizing agents and antioxidants that results in the overproduction of reactive oxygen species, which can damage not only nucleic acids but also proteins and lipids, is what defines oxidative stress (Rahal *et al*. 2014) [64]. When parasite infections impair the host's cells, tissues and subcellular organelles, this detrimental effect often referred to as oxidative damage occurs. Major antioxidant defenses, including as catalase and superoxide dismutase action, non-protein thiols, and glutathione concentrations of the impacted small ruminants, are typically reduced as a result of the ensuing redox homeostatic imbalance. Furthermore, helminth induced overproduction of hydrogen peroxide and hydroxyl radicals exacerbates these pro-oxidative state alterations, outperforming host detoxifying processes (Selkirk *et al*. 1998) [70].

4. Physiological and metabolic functions are impacted by oxidative stress in small ruminants

4.1 Cellular damage due to oxidative stress

It consists of the production of reactive oxygen species that flood the antioxidant protection of the host and causes significant molecular damage and malfunctioning of cells. Particularly, the overproduction of intracellular reactive oxygen species may oxidise critical biomolecules, such as proteins, lipids, and DNA, and, thus accelerating the process of cellular apoptosis and aggravating pathological states. The lack of balance, which frequently should be aggravated by environmental factors like insufficient control or nutrition, interferes with the cellular balance and disrupts energy metabolism. Such oxidative effects may ruin cell structural integrity and cause disrupted physiological processes, and its effects are of considerable importance to the functionality and health of animals, in general. Reactive oxygen species (ROS) can harm lipids, proteins, and deoxyribonucleic acid (DNA), leading to loss of cell function and even cell death, provided that there is a surplus of them. Dietary supplementation with antioxidants (e.g., vitamin E, selenium, and polyphenols). Application of better management practices to diminish environmental stressors. Nutrition optimization to counter the antioxidant defence mechanisms. Breeding programs will be implemented to choose animals with better oxidative stress resistance. It is important to understand and resolve oxidative stress induced cellular damage so that ruminants maintain ruminant health, productivity and welfare in their respective production system (Liebler at al. 1993; Perl *et al*. 2002) [46, 61]. The following figure. 2 illustrates the oxidative stress caused by helminth parasites on small ruminants' cells.

4.2 Reduced productivity capacity in small ruminants

Oxidative stress can negatively impact the growth rates, milk production, and reproductive capacities of ruminants. Free radicals generated through oxidative stress can damage the proteins, lipids, and DNA of cells (Heidarpour *et al*. 2012) [34]. Reduced development, production of milk, and reproductive capacity can result from this harm to cells. Furthermore, oxidative stress compromises egg maturation, sperm quality and embryo viability, all of which have a detrimental effect on reproductive function and ultimately lead to lower fertility and birth rates (Drackley 1999) [24]. Numerous environmental influences increase the severity of this physiological imbalance, with under optimal husbandry habits, extreme stressors and nutritional deficiencies being some of the factors that contribute to the amplification of the oxidative load placed on reproductive systems. High oxidative stress has been directly linked to reduced litter size and low weight of piglets in pregnant sows hence having a strong influence on the overall productivity of livestock. The pathophysiological mechanism is the increased generation of reactive oxygen species or a parallel loss of antioxidant capacity which forces the animal to channel metabolic resources that otherwise would be used to promote somatic growth and production into repairing the oxidative damage. As a result, effective performance is affected and economic losses are followed. These nutritional stressors, which are often met in production animals, result in a poor health condition and a significant reduction in the reproductive competence (Heidarpour *et al*. 2012) [34].

4.3 Oxidative stress compromised immune function in small ruminants

An animal's immunity may be weakened by prolonged oxidative stress, making it more vulnerable to illnesses and infections. Oxidative stress negatively impacts the immune system, making ruminants especially susceptible to disease (Makkar *et al*. 2007) [55]. Reduced production may arise from the energy needed to combat illnesses. Long-term oxidative stress also depletes an animal's organic antioxidant stores, making it harder for the living thing to protect itself from oxidative threats in the future. Because the delicate balance between anti-oxidants and antioxidants is upset, the resulting depletion in turn weakens immune function and makes tiny ruminants more susceptible to a variety of diseases. A decrease in the conversion of food ratio, weakened resistance to disease, and mortality are frequently the results of this dysregulation when additional environmental factors, such as subpar management techniques or severe stresses, exacerbate the condition. As a result, the combination of immunodeficiencies causes the cattle population's disease rate to rise and ultimately results in a marked decline in the animals' total production. Furthermore, in these compromised conditions, it is impossible to control the production of reactive oxygen species, which could exacerbate the condition by directly oxidatively stressing immune-mediated cells and signaling cascades, thereby reducing the effectiveness of the immune system's response (Hite and Cressler 2019) [36].

4.4 Oxidative stress causes tissue inflammation in small ruminants

Damage to tissue and calcification in afflicted organs can result from the inflammatory response brought on by parasites and made worse by oxidative stress. Chronic inflammation, which takes energy away from useful tasks and can result in tissue damage and decreased performance, can be brought on by oxidative stress (Martin *et al*. 2006) [56]. In addition to being crucial for the development of numerous illnesses, an excess of reactive oxygen compounds also feeds inflammatory processes by activating pro-inflammatory cytokines and immune effector cells. Fibrogenesis, a chronic inflammatory phenotype that is brought on by excessive cellular matrix molecule accumulation that ultimately leads to organ failure, can be brought on and maintained by chronic oxidative stress (Alam *et al*. 2020) [4]. Reactive oxygen species, cytokines that promote inflammation, and immune effector cells most notably macrophages interact intricately to obstruct tissue repair. In almost every organ system, especially vital organs like the respiratory system, circulatory, renal and hepatic cells, pathological deposition of their extracellular matrix elements, has unfavourable effects that can result in severe malfunction of the organs and even death. Controlling the course of cellular fibrogenesis, which is frequently linked to alteration of the metabolism and stimulation of transforming growth factor-β1 signaling pathways, requires fine-tuning oxidant and antioxidant systems. A redox-centric fibrotic condition, in which excess reactive oxygen species not only directly activates fibrogenic cells but also promotes inflammatory events and parenchymal cell apoptosis, can be brought on by disruptions in these regulatory mechanisms (Martin *et al*. 2006) [56].

4.5 Metabolic disturbances occur due to oxidative stress

Animal health and nutrition consumption may be impacted by oxidative stress's disruption of regular metabolic processes. Regular metabolic processes may be disrupted by oxidative stress, which reduces food uptake and feed efficiency. This inefficiency results in poorer milk production and slower growth rates (Dong *et al*. 2017; Finkel 2011) [22, 28]. As a result, somatic cells proliferate and milk's circulatory ketone body levels increase, indicating a breakdown in metabolic balance and the onset of inflammatory processes. Furthermore, excessive oxidative stress can result in subclinical level diseases such hepatic lipidosis or ketosis in dairy ruminants subjected to adverse consumption of energy in the context of increasing physiological demands or environmental calamities. Therefore, by decreasing the sensory characteristics of meat and raising the somatic cell count of milk, these metabolic diseases can significantly affect meat nutritional and performance metrics (Pivoto *et al*. 2015) [62]. This dysregulation of cellular and muscle activity typically happens when reactive oxygen species outnumber the antioxidant defense system, leading to ineffective energy metabolism and inappropriate nutrition distribution in the animal. When free radicals overwhelm the body's antioxidant defenses, this redox imbalance also referred to as oxidative distress causes a series of detrimental molecular events, including harm to membranes lipids, proteins and genetic material. According to Dong *et al*. (2017) [22], affected small ruminants exhibit decreased feed-conversion ratios, compromised immunity, and elevated death rates as a result of the cellular damage.

4.6 Several kinds of oxidative stress-related reproductive abnormalities in small ruminants

Helminth infections can have a severe negative impact on small ruminants' ability to reproduce due to oxidative stress. These parasite diseases increase the generation of ROS in the animal, and oxidative damage results from ROS overwhelming the animals' antioxidant defenses. This result may affect gametogenesis, fertilization, embryonic development, etc. In females there is the interference of oxidative stress with oocyte maturation, corpus luteum

function, folliculogenesis that may decrease fertility and increase embryonic loss. In males, too much ROS can harm sperm DNA, impede sperm motility, and reduce the overall chemical content of the semen in general. Oxidative stress also further impairs reproductive performance as it interferes with synthesis and control of reproductive hormones. All of which will result in reduced conception rates, increased embryonic mortality and decreased reproductive efficiency due to helminth associated oxidative stress on reproduction. The oxidative imbalance which is seen in such cases may trigger a continuum of reproductive pathologies such as metritis, retained placenta. Also, the augmented generation of reactive oxygen species and reactive nitrogen species (RNS) can destroy the crucial cellular components like proteins, lipids as well as nucleic acids and eventually disrupt the cellular balance and increase the frequency of reproductive dysfunction. This figure explains oxidative stress affects small ruminants' ability to reproduce.

4.7 Deterioration of meat and milk quality in small ruminants

The quality of meat and milk from small ruminant hosts can be degraded if subjected to oxidative stress associated with helminths. As a result of the parasite infection, a parasite produces ROS and the body is unable to neutralize the excess ROS (Alam *et al*. 2020) [4]. The oxidative damage leads to impact of tissues, such as mammary glands and muscle. Increased lipid peroxidation, protein oxidation, colour and texture changes in meat reduce shelf life and nutritional value of the meat. Oxidative stress in milk can affect milk fat stability and composition and thereby change products' flavour, processing characteristics and overall quality, similarly to oxidative stress in juice that leads to defects. The milk production can also be substantial effect on milk output and also the changed nutrient composition. The milk production may have a significant impact on the milk production and the nutrient contents altered. In addition to the milk quality, oxidative stress has a major effect on the surface of meat in small ruminants by derailing the cellular pathways that regulate energy metabolism and nutrient partitioning thus decreasing the performance and products of the animal including changes in preservative and sensory quality. This phenomenon takes place when the production of the reactive oxygen species and reactive nitrogen species overloads the animal endogenous antioxidant defense, causing an indiscriminate oxidation of biomolecules and redox signal disruptions. In small ruminant farming we need to develop the measures to reduce the impact of helminth infection on the quality of animal products utilizing these mechanisms.

5. Antioxidant defence produced in small ruminant's body due to oxidative stress

Oxidative stress occurs when the body's capacity to eliminate reactive oxygen species through antioxidant defences outstrips the generation of ROS (Murphy 2008) [59]. The antioxidant defence is required in order to prevent oxidative damage to tissues and cells (Cheeseman and Slater 1993) [14]. Enzymatic antioxidants (Eggler and Savinov 2013) [26] change superoxide radicals to hydrogen peroxide via the enzyme superoxide dismutase (SOD). A reaction that catalase is good at is the conversion of hydrogen peroxide to oxygen and water. Glutathione peroxidase (Cheeseman and Slater 1993; Perl *et al*. 2002) [14, 61] reduces hydrogen peroxide and lipid peroxides. This figure shows the body of a small ruminant produces antioxidant defense against ROS/RNS generation.

Glutathione is a reducing agent and free radical scavenging antioxidant in non-enzymatic antioxidants. Vitamin C (ascorbic acid) free radicals and regenerate vitamin E (Tebay *et al*. 2015) [74]. Vitamin E (tocopherols) prevent lipid peroxidation from damage the cell membrane (Cheeseman and Slater 1993; Wang *et al*. 2009; Baeeri *et al*. 2019) [14, 79]. Carotenoids are good free radical neutralizers and quench singlet oxygen. Ferritin sequestrates iron in order to prevent Fenton reactions; thus, antioxidant defence of metal binding proteins is of critical importance. The ferrous iron is oxidized by ceruloplasmin to its less reactive ferric form (Sabharwal and Schumacker 2014; Itsara *et al*. 2014) [66, 39]. DNA repair enzymes correct DNA damage and compartmentalization sequestration of ROS generating processes in specific cells compartment. All of these mechanisms, however, work in synergism to preserve cellular redox balance and prevent oxidative damage, and to maximally contribute to the cellular health (Maizels *et al*. 2004) [54].

6. Modulation of the immune system under oxidative stress in small ruminants

The interactions that helminths have with their ruminant hosts have a considerable impact on the host's immunological responses. These interactions frequently lead to immunomodulation, which helps the parasite stay alive and also impacts the host's health and effectiveness. This modulation may involve the diversion of host resources, particularly energy, towards immune defense, compromising vital processes such as growth and fat storage, and causing major metabolic costs to the host (Douhard *et al*. 2025) [23]. In host-parasite partnerships, this type of resource transfer is crucial since the host's nutritional state influences its capacity to mount an immune response or withstand infectious burdens (Coop & Kyriazakis 1999) [18]. This intricate link highlights the significance of food control in strengthening host defenses and increasing their resistance to helminth infection (Coop & Kyriazakis 1999) [18]. Furthermore, the idea that immunological competence has a nutritional cost and makes hosts more vulnerable during times of elevated metabolic demand is supported by the periparturient drop in ruminant immunity (Brindle *et al*. 2007) [10]. This kind of resource usage frequently results in unfavorable relationships between resistance to parasite infections and production characteristics (such as gaining weight or milk production) (Brindle *et al*. 2007) [10]. These compromises, which are more prevalent whenever energy is inadequate such as during infection related anorexia or other times of nutritional stress, hinder the capacity to effectively combat parasite burdens (Douhard *et al*. 2025) [23]. The immune system's alteration in small ruminants under oxidative stress is depicted in the accompanying Figure 5.

Chen *et al*. (2021) [10] claim that the complex adaptations of helminths that control host immunometabolism also include the active creation of chemicals derived from the parasite that have suppressive or misdirected impact on host immune responses. These compounds contribute to persistent infections without causing low-level clinical symptoms. By altering immune cell gene expression, altering host signaling networks, and warping cell metabolism, these released chemicals may foster an environment that is conducive to parasite survival and growth. The resulting result of this type of parasitic manipulation is frequently the induction of a predominantly T-helper 2 immune response, the generation of specific cytokines and antibodies which, though necessary in the clearance of certain helminth stages, can also inhibit the

important Th1-mediated immunity necessary to clear other co-infections. This complex immunomodulation highlights the vulnerability of the resource-immune connection since it is reliant on the nutritional status of the host and directly affects the susceptibility and severity of helminth infections (Bryan *et al*. 2013) [11]. It has been widely known within ecological research that the flows of energy and nutrients are central to the study of the functioning of an ecosystem and the adaptive response of food webs to changes in the environment (Bryan *et al*., 2013) [11]. This applies to host parasite interactions, where the availability of resources has a direct effect on the energetic costs of immune defense, host biomass, and parasite development in an infected host (Brindle *et al*. 2007) [10]. These complex resource interactions are important to predict how anthropogenic and other environmental resources can modify host vulnerability and disease spread among populations (Perl *et al*. 2002) [61]. Additionally, the increasing issue of anthelmintic resistance complicates control efforts and renders the current methods of control unsustainable. The production of immunomodulatory substances, which directly affect host lymphocyte populations by modifying cytokines and inhibiting effector action, is one of these complex evasion techniques (Maizels *et al*. 2004) [54]. These parasite secretions have the potential to polarize macrophages, promote regulatory T cell proliferation, and control host antigen presentation. The helminth-derived chemicals often activate host receptors that detect patterns to cause particular differences in immune cells and cytokine generation. The immune response is further skewed toward the less potent type 2 defense that fosters tolerance as a result of this long-term immunological regulation (Tsubokawa 2023) [77].

7. Role of Gut Microbiota in modulating oxidative stress

The pathophysiology of many diseases and the issue of livestock system production are both significantly influenced by oxidative stress. It is the outcome of an imbalance between an organism's antioxidant capability and the generation of reactive oxygen species (Tebay 2015) [74]. Such physiological disturbances may have a serious negative effect on the health of small ruminants, which could lead to a decrease in productivity and significant economic losses for farmer (Grotto *et al*. 2009) [32]. Therefore, a thorough understanding of the intricate relationships involving the bacteria in the gut and regulating oxidative stress is necessary for the development of efficient mitigation techniques to avoid these adverse effects in sheep and goats. A boost in oxidative stress causes lipid peroxidation, which destroys cell membranes and degrades vital antioxidant enzymes that protect cell function and structure and affects cell equilibrium (Moldogazieva *et al*. 2019) [57]. The negative effects that such interruptions can have, such as reduced productivity in feeding, decreased reproducing efficiency, and an increased susceptibility to infectious diseases, put a lot of pressure on sustainable animal husbandry (Aktas *et al*. 2017) [3]. The role of gut microbiota in reducing oxidative stress is depicted in figure.6

The host's metabolic rate, immune system growth, and overall health all depend on the gut microbiota, an eclectic assortment of microorganisms that live in the gastrointestinal tract. It may therefore affect the oxidative status of the host (Chen *et al*. 2021) [15]. This malfunction also adds to systemic inflammatory conditions by producing pro-oxidant chemicals and decreasing cellular energy generation (Birben *et al*. 2012). Therefore, the interaction between the gut microbiota's composition, mitochondrial activity, and the whole system's oxidative stress is one of the most important areas of research for improving the efficiency and well-being of small ruminants. The prospect for microbiota-focused measures to lessen oxidative stress in small-ruminant populations will be evaluated, the processes by which gut microbe dysbiosis can result in oxidative stress will be systematically addressed, and the contribution of particular microbial metabolic products to the control of host redox status will be extensively investigated (Angeles *et al*. 2020). One of the main goals will be to identify the molecular pathways that produce particular microbial species and their byproducts from metabolism (Rahal *et al*. 2014) [64]. This mechanistic understanding is necessary to develop focused dietary and probiotic therapies to increase small ruminants' tolerance to oxidative stress and enhance their general health and productivity (Sahoo *et al*. 2013) [67].

8. Implications for disease management in small ruminants

The complicated relationship among helminthes and their host nutrition is vital for growing long-term control measures, particularly in view of the increased threat of anthelmintic resistance (Molla *et al*. 2023; Coop & Kyriazakis 1999). The interaction has a direct effect on the host capacity to generate an effective immune response, and also resource allocation, which has an overall effect on productivity (Coop & Kyriazakis 1999) [18] (Brindle *et al*. 2007) [10]. Nutritional interventions may also greatly benefit the resilience of the host, the ability to remain productive despite a parasite challenge as well as resistance, the ability to restrict parasite establishment, growth and fecundity (Coop & Kyriazakis 1999) [18]. Immune reactions to parasitic infections involve a significant expenditure of energy and can sometimes require a redistribution of energy resources at the expense of other physiological functions such as growth and energy storage (Douhard *et al*. 2025) [23]. Diversion of metabolic resources has the potential to increase the loss of production hence strategic nutritional management is one of the cornerstone elements in reducing economic impact of helminthiasis. Additionally, altering the host's diet can regulate the gut microbiota, which in turn can influence the susceptibility to parasite infections and potentially lead to the development of novel disease treatment strategies (Hite and Cressler 2019) [36]. These steps are crucial for small ruminant animals since helminth parasites are endemic there and can cause high rates of illness and mortality, which can have a major negative impact on livestock output and food security. Parasitism is an essential component of small ruminant herd management due to the high rates of strongyle gastrointestinal prevalence, 70%-100% and the ability to contribute up to one-third of total productivity losses. It is well known that such a large parasitic load, especially of helminths, is a significant barrier to the productivity of small ruminants in Africa (Grotto *et al*. 2009) [32]. As a result, effective parasite control is not only a veterinary concern but also a fundamental element of sustainable livestock production, requiring a combination of techniques that consider both conventional and innovative approaches. In regions like the Nigerian savanna, where West African dwarf goats are known to be naturally immune to the impact of some helminths, it is necessary to review the current disease management paradigm in order to incorporate tactics that would strengthen host resistance and dependence on chemical interventions (Chiejina *et al*. 2010). This natural opposition highlights how genetic selection programs can increase resilience to parasitic diseases as an addition to

nutritional approaches and an alternative to over-reliance on anthelmintics. Due to the extensive resistance caused by the careless use of anthelmintics, farmers in some regions, like Niger, are adopting traditional plant medicines as alternative control (Chen *et al*. 2021) [15]. Current research aims to standardize these traditional methods and ascertain their efficacy and safety in order to identify the most effective treatment modalities. These methods typically involve ethnobotanical activities that involve the utilization of different plant families. To overcome resistance, this will entail going over the anthelmintic properties of several plant extracts and how they might be used in addition to standard treatment (Grotto *et al*. 2009) [32]. For small ruminant farmers and the livestock sector, helminth infection related oxidative stress could cause loss of high revenue. These parasite illnesses jeopardize the animals' health, and thus these adversely affect the animals' production, which in turn decreases their total economic value. It is known that helminth induced oxidative damage affects a host of physiological functions, such as those that affect reduced growth rates, poor reproduction and poor production of milk and wool. Additionally, diseased animals might need more feed to be healthy, thereby increasing cost of production (Alam *et al*. 2020) [4]. The economic impact is not limited to the simple losses, because infected animals are more prone to developing secondary infections and diseases leading to morbidity and mortality, at higher veterinary and economic costs for the producer. In addition, milk, meat, and wool from sick animals will also cost less on the market and therefore the earnings from farmers will further decrease. The tails of these elements put small ruminant farmers through these hardships thereby highlighting the importance of efficient parasite control methods in maintaining profitability of livestock enterprises.

9. Conclusion

Oxidative stress is an idea that is still being established in the field of small ruminant medicine. Although oxidative stress is linked with many diseases in ruminants, there is a dearth of understanding of the effect of oxidative stress on the health and productivity of ruminants. The pathologic change either initiated or caused by oxidative stress is not clear, for instance. If all is known about the pathology of oxidative stress in ruminants, then we will be able to use targeted antioxidant therapy.

The research of oxidative stress includes the establishment of a panel of blood biomarkers that will precisely characterize the tissue oxidative state of an animal (Lykkesfeldt and Svendsen 2007) [52]. Several ways of estimating oxidative damage have been devised. Veterinary scientists and physicians must determine validity of the biomarkers of oxidative stress that are tested in blood and serum as signal of the total oxidative stress state. According to Arguelles *et al*. (2004) [7], 1) the increased blood biomarker concentration as a function of the degree of tissue damage by a widespread rise in oxidative stress in as much as for most tissues, 2) an increase in oxidative damage produced specifically in the cardiovascular system, 3) the dysfunction is pathogenic caused by an increase in oxidative stress in a single tissue. If nutraceuticals, antioxidants and organ snatching drugs are added to the therapy plan, a better response can be expected for this kind of mange. Oxidative stress and organ function tests may be beneficial to physicians.

Abbreviations

- **DNA:** Deoxyribonucleic acid
- **GPx:** Glutathione peroxidase
- **HNE:** Hydroxynonenal
- **MDA:** Malonadialdehyde
- **PUFAs:** Polyunsaturated fatty acids
- **RNA:** Ribonucleic acid
- **RNS:** Reactive nitrogen species
- **ROS:** Reactive oxygen species
- **SOD:** Superoxide dismutase
- **NADPH:** Nicotinamide Adenine Dinucleotide Phosphate
- **IL:** Interleukins
- **TGF:** Transforming Growth Factor
- **GSH:** Glutathione
- **GSSG:** Glutathione Disulfide
- **NO:** Nitric Oxide
- **ONOO:** Peroxynitrite anion
- **OH:** Hydroxyl group
- **H_2S:** Hydrogen Sulfide
- **LH:** Luteinizing Hormone

Figure 1

Alt text: Oxidative stress's effects on the bodies of small ruminants can lead to various health issues, including tissue inflammation, apoptosis and lipid peroxidation.

Figure 2

Alt text: Cell damage happens due to oxidative stress and can lead to various health issues if not managed properly. To combat this, antioxidants play a crucial role in neutralizing free radicals and reducing the risk of cellular injury.

Figure 3

Alt text: Different types of reproductive impairments are identified in small ruminants each with unique causes and implications for herd management

Figure 4

Alt text: Antioxidant defence produced in the body due to oxidative stress can help mitigate cellular damage and promote overall health. By enhancing the body's natural defenses, individuals may experience improved resilience against various diseases and better aging processes

Figure 5

Alt text: Modulation of the immune system under oxidative stress in small ruminants has significant implications for their overall health and productivity. Understanding these mechanisms can help in developing strategies to enhance resilience and improve management practices in livestock.

Figure 6

Alt text: Role of Gut Microbiota in modulating oxidative stress has gained significant attention in recent research. Studies indicate that a balanced gut microbiome can enhance the body's antioxidant defenses, thereby reducing the detrimental effects of oxidative stress on cells and tissues

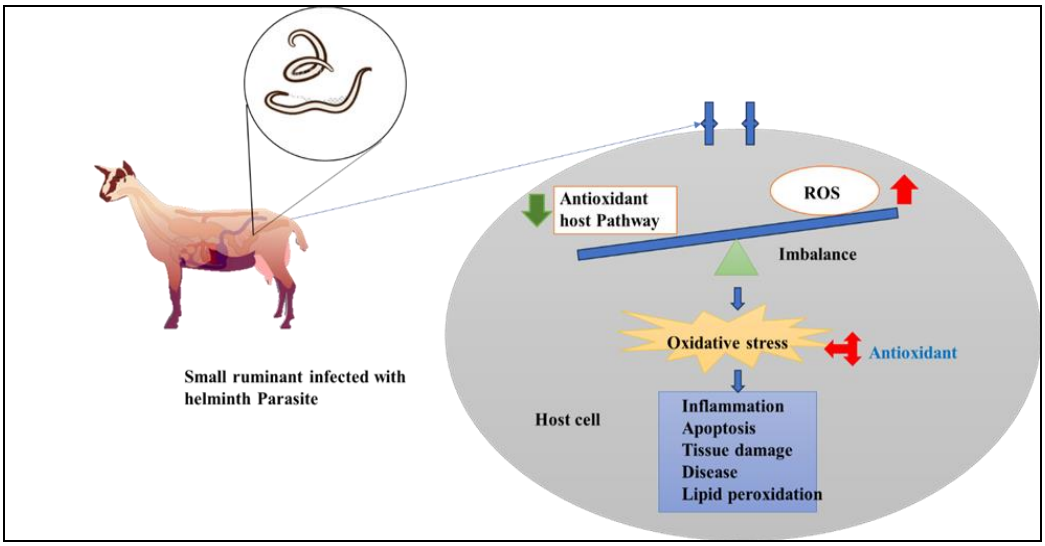

Fig 1: The impact of oxidative stress in small ruminant's bodies.

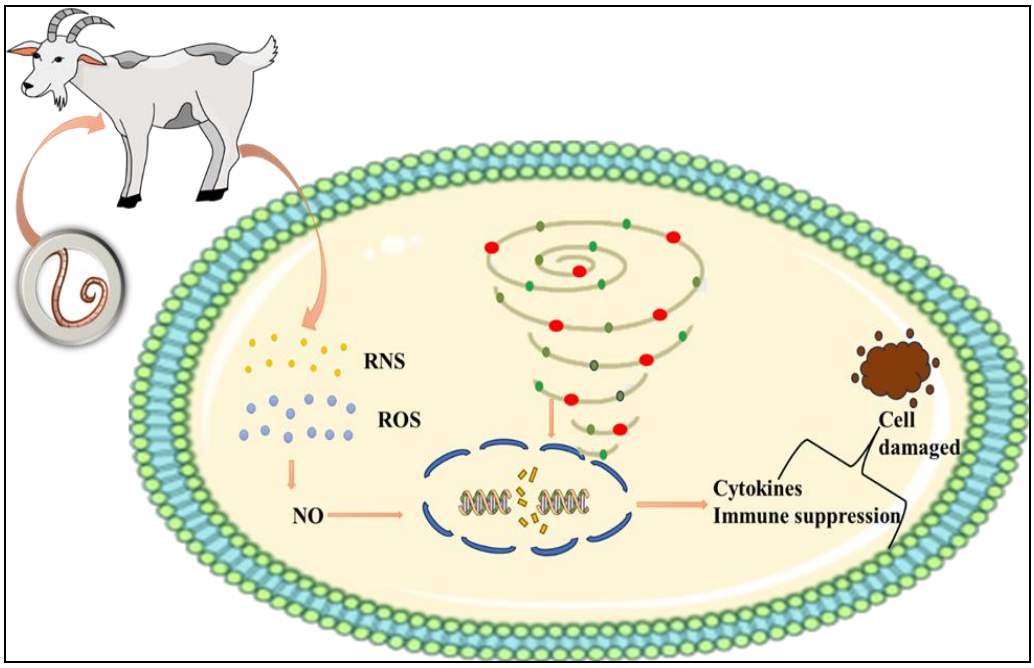

Fig 2: Helminth infections cause oxidative stress that damages cells in small ruminants.

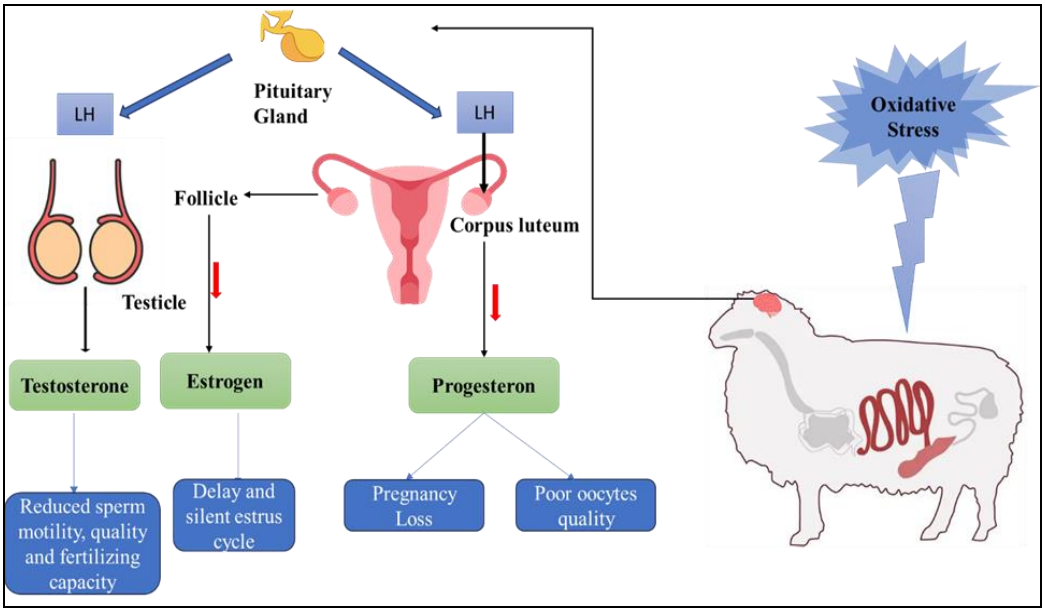

Fig 3: Fertility challenges in small ruminants caused by oxidative stress in small ruminants.

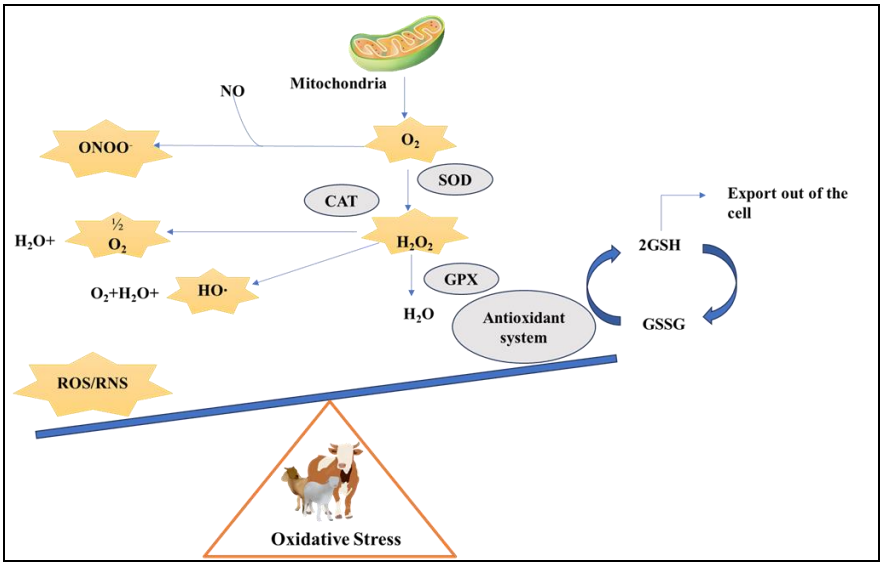

Fig 4: Small ruminants' bodies create antioxidant defense as a result of oxidative stress.

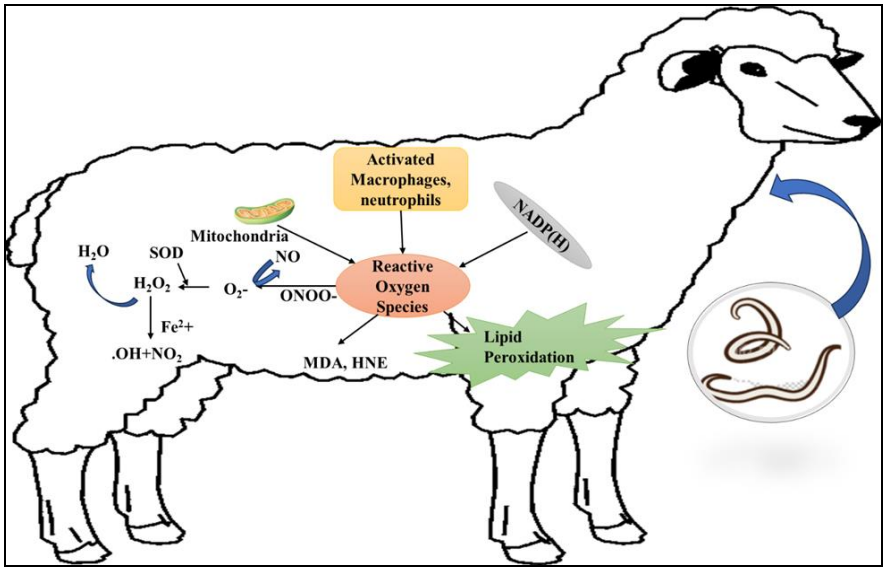

Fig 5: Immune system alteration in small ruminant's body during ROS/RNS generation.

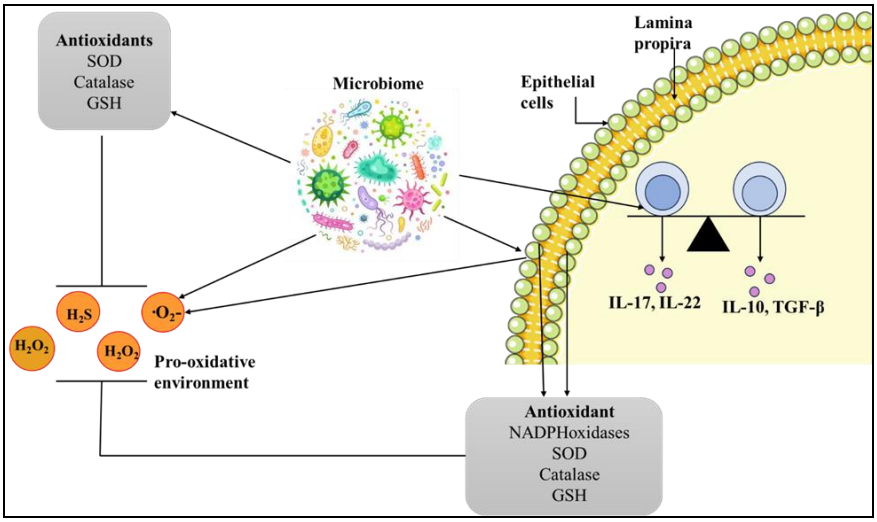

Fig 6: The gut microbiota's activity towards reducing oxidative stress.

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Compliance with ethical standards
This article does not contain any studies with human participants performed by any of the authors.

Author's contribution
MM Kumbhar conceived the study. The manuscript was prepared by PS Sethy. All authors read and approved the final manuscript.

Competing interests
The authors declare that they have no competing interests.

Data availability
The data presented in this study are available on request from the corresponding author.

Conflict of Interest: Not available

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