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Oxidative Stress in Animals and the Balance Between Reactive Oxygen Species Production and Antioxidant Systems

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Annotation: Oxidative stress which results from the imbalance between reactive oxygen species (ROS) generation and antioxidant defense is a fundamental issue influencing animal health and productivity. Excessive ROS can cause oxidations of lipids, proteins and DNA, which results in growth retardation, reproductive depression, immunosuppression of animals and low quality of animal products. In this review, the main sources of ROS will be addressed along with how they play a role in oxidative stress in biological systems and how antioxidant defenses - both enzymatic and non-enzymatic -come into play. Regular management strategies and environment are identified as effective measures to counteract oxidative stress, as well as based nutritional interventions (i.e., vitamins, trace elements and phytochemicals). Furthermore, recent techniques of biomarkers and molecular diagnosis allow the early detection, the phytoplanktic redox imbalance as well as species specific management. The balance between ROS and the antioxidant system is absolutely necessary to optimize growth, reproduction, immune status, and product quality in animals. Further studies are required for the optimization of interventions and to explore new biomarkers in order to more accurately assess oxidative stress.

Keywords: Oxidative stress, ROS,

Antioxidants, Animal health, Biomarkers.

Introduction

Oxidative stress (OS) is characterized as an imbalance between the generation of reactive oxygen species (ROS) and the capacity of antioxidant defense mechanisms to detoxify them, thereby leading to a perturbed cellular redox status (1). This phenomenon is a fundamental principle of animal physiology and has an essential function in the regulation of physiological metabolisms to maintain homeostasis, and also participates in the pathogenesis of a variety pathological diseases (2).

ROS are mainly formed in the context of aerobic metabolism, especially mitochondrial ETC activity, which results in the production of superoxide anion (O_2^-), hydrogen peroxide (H_2O_2), and hydroxyl radicals (•OH) (3). While ROS at low levels significantly function as signaling molecules in the control of gene expression, cell growth, and immune defense (4), their excessive generation overloads antioxidant activities leading to free radical-induced damage to lipids, proteins and DNA damaging ultimately impairing cellular function (5).

In farm animals, oxidative stress is frequently linked with important physiological stages (e.g., fast growth, pregnancy, lactation and heat stress) which impact productivity performance and product quality (6). It has also been shown that redox balance is an early sign of welfare (7).

In companion animals (dogs and horses), oxidative stress is also associated with the development of inflammatory and metabolic disease (8). In ruminants suffering from parasitic blood infections like theileriosis and piroplasmosis oxidative stress enhances tissue damage and decreases immune competency (9, 10).

In addition, oxidative stress regulates life history traits such as growth, reproduction and life span, indicating its evolutionary and physiological importance across species (10).

The knowledge on sources of ROS, mechanisms of oxidative damage and antioxidant defense system will be fundamental in the development of nutritional, management and therapeutic interventions to enhance their health status and productivity (6,10).

Far from being understood, the nature of oxidative stress in different animals in different physiological and environmental states is only now beginning to be revealed. In this review, the knowledge of ROS generation, oxidative damage and antioxidant defense systems is overviewed in order to focus on protective measures that mitigate oxidative stress and with it health and performance.

2. Origins and Physiological Significance of ROS Production in Animals

Reactive oxygen species (ROS) are constantly produced within animal cells as side products of normal metabolism, predominantly in mitochondrial oxidative phosphorylation (3). Although low levels of ROS are important for cellular signaling, immune defense and adaptation to stress (4), high levels should be restricted because they cause oxidative damage on lipids, protein and DNA5.

2.1. Major Sources of ROS

In animals, ROS are produced from several principal sources. Mitochondrial respiration is the major source although electron leakage from the electron transport chain will generate superoxide anions that in turn form hydrogen peroxide and hydroxyl radicals (3,4). ROS are generated by immune cells, including neutrophils and macrophages during pathogen clearance, and can also cause damage to host tissues when produced in excess (2,9). ROS production and oxidative damage in susceptible tissues are intensified by environmental stressors (heat, toxins and pollutants) (6). Lastly, physiological stress including pregnancy, lactation or rapid growth

raises metabolic ROS production and may surpass antioxidant hurdles (6,10), Table 1.

Table 1. Main Sources of ROS in Animals and Their Impacts

Source	Mechanism	Impact on Animals
Mitochondrial respiration	Electron leakage in electron transport chain	Oxidative damage in liver, muscle, and heart tissues (3,4)
Immune response	ROS produced by phagocytes	Tissue inflammation and collateral damage (2,9)
Environmental stressors	Heat, toxins, pollutants	Lipid peroxidation, reduced growth, impaired reproduction (6)
Physiological stress	High metabolic demand (pregnancy, lactation, rapid growth)	Increased ROS, decreased antioxidant defense (6,10)

2.2 Mechanisms of Oxidative Damage

In animals, excessive ROS can be deleterious for lipids, proteins and DNA in the cell which can interfere with cellular processes and increase susceptibility to various diseases and loss of productivity (5,11), Table 2. The main mechanisms include:

- **Lipid Peroxidation**: The reaction of the ROS with the polyunsaturated fatty acids in cellular membranes and production of reactive aldehydes, such as MDA or 4-HNE, has been described as an endpoint for lipid peroxidation (Fig. 3). These chemicals disrupt the cell membrane, enzyme function and can also generate other free radicals (5,12).
- **Protein Oxidation**: ROS change the side chains of amino acids and the backbones of proteins, leading to changes in enzyme activity, protein conformation and signaling paths 9). Oxidation of proteins could interfere with muscle function, metabolism, and immune response (5,13).
- **Damage from hydrogen peroxide**: Hydroxyl radicals (•OH) can induce strand breaks, base modification and cross-linking. Such damage may result in mutations, disrupted celldivision, apoptosis or cancer in a chronic oxidative stress (5,14).

Table 2. Main Mechanisms of Oxidative Damage in Animals

Target	ROS Involved	Consequences	Key Biomarkers
Lipids	O_2^-, •OH, H_2O_2	Membrane damage, lipid peroxidation	Malondialdehyde (MDA), 4-HNE (12)
Proteins	O_2^-, •OH	Enzyme inactivation, structural damage	Carbonylated proteins (13)
DNA	•OH	Mutations, strand breaks, apoptosis	8-OHdG (14)

2.3 Tissue Susceptibility and Species-Specificity

Tissues with high metabolic activity, such as liver, heart and skeletal muscle are more susceptible to ROS mediated injury (3,10). Among species, there are variations in the activity and concentration of antioxidant enzymes which contribute to resistance against oxidative stress (1,6,10). Physiological or stress conditions increase ROS generation so that it is frequently in excess of their antioxidant capacity with resulting functional consequences (6,10,11).

3. Antioxidant Defense Systems in Animals

A fine equilibrium exists in animals between the generation of reactive oxygen species (ROS), and their removal by intricate antioxidant defense mechanisms. Such machineries are required

for maintaining cellular wholeness, controlling metabolism and partially shielding tissues against oxidative damage resulting from high metabolic rates, environmental stress, or disease states (15,16). The antioxidant defense systems of animals can be divided into enzymatic and non-enzymatic ones, Table 3, which cooperate to scavenge ROS and keep redox balance (15).

Enzyme antioxidants are the first line to confront oxidative stress. Among first defenses, superoxide dismutase (SOD) is turning into the conversion of a new enemy to an old one -the transformation of superoxides anions (O_2^-) into hydrogen peroxide (H_2O_2). This reaction is important since superoxide radicals are very reactive and may start lipid peroxidation or protein oxidation if not scavenged. SOD has several isoforms in the cytosol, mitochondria and extracellular space (15) and it's activities can change depending on tissue and species which represents metabolic activity as well as a sensitivity to oxidative stress (16).

H2O2 generated by SOD is also detoxified by catalase (CAT) into water and oxygen as a final product. Catalase activity is especially abundant in metabolically active tissues like the liver and erythrocytes, and thus ensures a rapid conversion of hydrogen peroxide with no hydroxyl radical generated through the Fenton reaction (15,16,17). In addition to these enzymes, also glutathione peroxidase (GPx) reduces both hydrogen peroxide and lipid hydroperoxides with reduced glutathione (GSH) as the substrate. GPx is a selenium dependent enzyme whose activity not only indicates the nutritional level of this element in body, but also oxidative stress experienced by the animal. GPx is particularly crucial in guarding its erythrocytes, milk and reproductive tissues from oxidative insult (15,16,18).

Non-enzymic antioxidants represent a second line of defense, as they are able to scavenge directly ROS or can support enzymatic systems. Glutathione (GSH) is the main intracellular thiol, which is involved in ROS scavenging and acting as a cofactor for GPx. Vitamins are essential components for the functioning of cellular enzymatic activities; among which, vitamin E(a lipid-soluble antioxidant) blocks the nonenzymic peroxidation of lipids in cellular membranes and also works as an alkaline buffer against hydrogenperoxide and also protects against oxidative damage, whereas water-soluble vitamin C regenerates oxidized form of vitamin E and scavenges aqueous-derived ROS.16 19 Furthermore, selenium, zinc and copper as cofactor ions of some pivotal antioxidant enzymes are required for the redox balance. Selenium, for example, is a cofactor of GPx; zinc and copper are cofactors for SOD activity (16,18).

The balance between enzymatic antioxidative systems and non-enzymatic antioxidants is delicate and depends on various factors (such as species, tissue type, age, nutritional status, or environmental conditions) (1). Stress-induced animals, physiological, nutritional or environmental stress has been known to result in alteration of activity of antioxidant enzymes and levels of non-enzymatic antioxidants. It is of great importance to monitor these parameters for determining the status of oxidative stress as well as devising strategies to improve animal health and productivity (6, 10, 15–18).

Table 3. Key Antioxidants in Animals and Their Roles

Antioxidant	Function	Key Tissues	Function
SOD	Converts $O_2^- \rightarrow H_2O_2$	Cytoplasm, mitochondria	Primary defense against superoxide radicals (15,16)
Catalase	Converts $H_2O_2 \rightarrow H_2O + O_2$	Liver, erythrocytes	Prevents hydroxyl radical formation (15,17)
GPx	Reduces H_2O_2 and lipid hydroperoxides	Liver, erythrocytes, milk	Selenium-dependent, cofactor: GSH (15,18)
Glutathione (GSH)	ROS scavenger, cofactor for GPx	Intracellular	Concentration depends on nutrition and stress (15,18)
Vitamin E	Protects lipid membranes	Liver, muscle, reproductive tissues	Lipid-soluble antioxidant (16,19)

Vitamin C	ROS scavenger, regenerates vitamin E	Plasma, tissues	Water-soluble, dietary requirement varies (16)
Selenium	Cofactor for GPx	Liver, erythrocytes	Essential trace element (18)
Zinc & Copper	Cofactors for SOD	Liver, muscle	Maintain SOD activity (16,18)

4. The determinants of the redox homeostasis in animals

In animals, redox balance is a dynamic status and dependent on various factors such as physiological status, nutrition, environmental experience, age or disease. Any disturbance of this equilibrium can lead to an excess of oxidative stress that affects cell function, productivity and general wellbeing (6,10,15).

4.1. Age

Age plays a key role in the antioxidant activity of an individual. Young animals frequently possess incomplete antioxidant pathways, so they are more susceptible to oxidative stress during rapid growth and organogenesis. On the other hand, the reduced antioxidant enzyme activities including superoxide dismutase (SOD) and glutathione peroxidase (GPx) due to aging may result in attenuated ability to oppose increased ROS generation,10,15 suggesting the potential impairment of system functionality in older animals. These changes associated with age affect not only metabolic ability, but also reproductive and immune capacity.

4.2. Nutrition

Diet has a direct action on antioxidant defense by supplying substrates for enzymatic and non-enzymatic systems. Vitamin E and C, selenium or other trace element-deficient diets restrict the synthesis and activity of key antioxidant enzymes in the body as well degrade its ability to scavenge ROS. Supplementation of these nutrients has been found to increase antioxidant defense, decrease lipidperoxidation and to improve performance and reproductive responses in livestock species (16,18,19). For example, dietary supplement of selenium and vitamin E can enhance the activity of GPx and SOD (16) but decrease MDA content, an important marker for lipid peroxidation (18).

4.3. Physiological stress

Further, OD could lead to increased production of ROS due to higher metabolic requirements under physiologic stresses, like pregnancy, lactation and rapid growth. In dairy cattle, high lactation levels increase oxidative metabolism which may saturate endogenous antioxidants thereby leading to conditions such as mastitis or lowered fertility (6, 10). Pregnant ewes and sows are also exposed to higher rates of ROS generation that may influence fetal development and lower birth weights of offspring (6,10).

4.4. Environmental factors

High synthesis temperature, toxins, and pollutants can as well affect redox balance in a body. Metabolically active tissues generate ROS in response to high ambient temperature or environmental toxins and result in oxidative damage, reduced enzyme activity, growth and/or reproduction (6,10,16). These stressors show the effect of sound environmental practices in farm animal systems on antioxidant capacity.

5. Effects of Oxidative Stress on Animal Health and Production

Oxidative stress (OS) considerably affects the health and productivity of animals. If ROS production is more than the cellular antioxidant ability, then tissue and cell functions are disturbed resulting in growth retardation, reproductive failure, immune deficiency, and compromised product quality (6,10,15), Table 4. These consequences are of special interest in farm animals, since the oxidative imbalance can affect profitability by lowering meat; milk and

egg production (4, 6,10,15).

Concerning growth performance, oxidative stress can inhibit the development of muscle and metabolic efficiency. ROS and subsequent lipid peroxidation and protein oxidation modified membranes and enzymes, which resulted in lower nutrient utilization efficiency and slower BWG (5,12,13). In poultry, it was found that increased oxidative stress reduces feed conversion efficiency, although when supplemented with antioxidants such as vitamin E and selenium, redox state is improved and growth improves (16,18,19). Comparatively, in ruminants, there is a decrease in body weight gain and overall performance when oxidative damage occurs during either rapid growth or high metabolic activity (6-10).

Reproductive performance is very sensitive for oxidative stress. ROS also can cause harms to the ovarian follicles, quality of oocyte and embryo development in mammalian females. Oxidative stress in lactating cows Early lactation oxidative stress has been associated with lower conception rate and increased calving intervals (6,10,15). High maternal ROS during pregnancy in small ruminants can reduce fetal growth and birth weights, showing the importance of maintaining redox balance for successful reproduction (10,12).

The immune system is also a principal victim of oxidative stress. Although necessary for pathogen control, ROS can be harmful to immune cells in high amounts, as they result in immunoinhibition by depressing the phagocytosis and cytokine production of animals after infecting pathogens (2,5,9). In cattle, piroplasmosis-infected animals are extra susceptible to red blood cell lysis when ROS production is increased or(Tijsenet al., 1990) immune function suppressed indicating a link between infection and the generation of ROS and in this respect an adequate antioxidant capacity (5, 9, 10).

Last, oxidative stress has an impact on the quality of animal productions. Lipid peroxidation in meats causes rancidity and shortened shelf-life, while oxidative damage in milk decreases vitamin composition and modifies the functionality of proteins (4,6,16). These effects have been attenuated by supplementation with dietary antioxidants, thereby enhancing product quality and extending storage stability (4,16,18).

Table 4. Implications of Oxidative Stress on Animal Performance

Aspect	Effect of Oxidative Stress	Mitigation Strategy
Growth	Reduced weight gain, impaired muscle function	Antioxidant supplementation (vitamins E, C, selenium) (4,16,18)
Reproduction	Lower conception rates, fetal growth restriction	Nutritional antioxidants, management of lactation stress (6,10,12)
Immunity	Immune cell damage, higher susceptibility to infections	Dietary antioxidants, disease prevention strategies (2,5,9)
Product Quality	Lipid peroxidation in meat, reduced milk quality	Antioxidant-enriched feed, proper storage (4,16,18)

6. Ways to Reduce Oxidative Stress in Your Animals

OP can be efficiently controlled by the nutrition-based approach together with antioxidant supplementation or environmental/management programmes. These strategies are designed to increase the animal intrinsic antioxidant system and decrease ROS disarrangement, leading to an increase in overall health and productivity (20, 21).

The role of nutrition in reducing oxidative stress. Conventional antioxidants found in the diet, including vitamins E and C, selenium, as well as carotenoids have been shown to enhance both enzymatic and non-enzymatic antioxidant defence systems. For example, vitamin E supplementation preserves cell membranes against lipid peroxidation and selenium potentiates

the glutathione peroxidase activity, leading to the immediate reduction of H 2 O 2 in tissues (21,22). Furthermore, polyphenolic compounds in plant-derived feeds can be ROS scavengers and also regulate redox-sensitive signal pathways to contribute the antioxidant defense (20,23).

It is also a vital consideration based on environmental and management factors. Lowering the body temperature, managing stress levels, ensuring cleanhousing and good ventilation help diminish overproduction of ROS in metabolically active organs. In addition, reducing exposure to environmental toxins and pollutants such as heavy metals that generate ROS and/or deplete antioxidants is crucial for redox homeostasis (20,24).

Character of the feed can be changed not only to add antioxidant compounds, but also to enhance macronutrient balance, that affect oxidative metabolism. High PUFA diets favor growth and good product quality, but also make the fish more susceptible to lipid peroxidation. In these instances, providing antioxidants at the same time guarantees that ROS activity is kept in check and oxidative damage to lipids, proteins and nucleic acids are avoided (20,21,25).

Timing of intervention is crucial. Physiological critical periods, such as early lactation, fast growth phases and gestation, are characterised by enhanced oxidative stress. Introduction of antioxidant supplementation can avoid performance decrements and decrease the likelihood of disease during these periods (6,10,20). For instance, antioxidant (AOX)-fortified diets during early lactation improve milk quality and reproductive performance as well as decrease oxidative damage endpoints such as malondialdehyde and protein carbonyls (20–22, 25).

7. Novel Biomarkers and Innovative Tools for Assessing Oxidative Stress in Animals

Oxidative stress in animals is an objective condition; a physiological process as well as a quantifiable state, which can be assessed through different biomarkers and laboratory tests. Due to such early detection of redox balance disorders before development of overt pathology recent investigations were focused on finding sensitive and specific markers which could reflect the balance between ROS generating system(s) and antioxidant defensive barrier (22,26,27).

The lipid peroxidation product malondialdehyde (MDA) is one of the most widely studied markers and is considered as a biomarker that characterises damage to cellular membranes by ROS. Elevated MDA has also been documented in other species due to physiological stress, infection or environmental insult, which reflects continual oxidative damage (22,28, 29). Likewise, the protein carbonyls are used as indicators of protein oxidation, thus giving an indication of enzymatic inactivation and structural protein damage resulting from oxidative stress (23,28).

Enzyme activities (SOD, CAT and GPx) are also often measured as indicators of antioxidant capacity. The activities of these enzymes represent the initial strategies for coping with ROS and could reflect both de novo adaptation to stress and the impairment of antioxidant defense systems in response to long-term oxidant challenge (28,29).

Apart from the classical markers, advanced molecular techniques have evolved. One example is 8-hydroxy-2′-deoxyguanosine (8-OHdG) that is a stable marker of oxidative DNA damage and therefore provides an indicator of the genomic integrity under ROS attack (29). Other approaches are assays for total antioxidant capacity (TAC) that consider the contribution of both enzymatic and non-enzymatic defenses and can provide an overview of the animal’s overall redox status (30,31,32).

New analytical methods like HPLC, spectrophotometry, and ELISA based assays allow determination of these important biomarkers in blood, milk or tissue, as well as other biological matrices. With these methods, oxidative stress can be studied in real time, the effectiveness of dietary or pharmacological interventions can be assessed and species-specific management strategies can be created (22, 33, 34).

New biomarkers (eg, redox-sensitive transcription factors and markers of mitochondrial

function) that offer more detailed information about oxidative processes at the cellular level have been further investigated in recent studies. It emphasizes the complex balance between ROS production, antioxidant defense systems and cellular signaling pathways, if possible leading to early interventions and enhanced animal welfare (30,35,36).

Conclusions

Oxidative damage causes capital loss in animal health and productivity due to lipid, protein and DNA damage. This could result in poor growth, low reproductive performance, decreased immune status and lower quality of animal products. Good management of oxidative stress is based on nutrition, antioxidants and good environmental and management practices. The monitoring of the antioxidant status and oxidative markers is useful for diagnosis at an early stage and providing targeted treatment.

Progress in the field of biomarker research, and molecular diagnostic tools provide opportunities to refine assessments on oxidative stress and to apply targeted interventions aimed at enhancing animal welfare conditions and favouring optimal production regarding growth rate, reproductive traits, disease resistance mechanisms and product quality. The ability to maintain an appropriate balance of reactive oxygen species and antioxidant defenses is vital for the health and well-being of animals, and further investigation is needed to improve existing approaches as well as identifying novel strategies for intercepting oxidative damage.

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