



Oxidant and Antioxidants in Poultry and Mammalian Animals

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Review

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ABSTRACT

Oksidatif stres, reaktif oksijen türlerinin (ROT) üretimi ile koruyucu mekanizmalar tarafından ortadan kaldırılması arasında kronik hastalıklara yol açabilen bir dengesizlik olarak görülmektedir. Bu mekanizmadaki dengesizlik, DNA, proteinler ve lipidler gibi hücrel moleküllerin zarar görmesine neden olabilir. Oksidatif stres veteriner hekimlikte araştırmaların aktif bir sahasıdır ve sepsis, mastitis, asidozis, ketozis, enteritis, pnömoni, kanser, solunum ve eklem hastalıklarının dahil olduğu pek çok organ hastalıklarında olguya dahil olmaktadır. Bu derlemede çok sayıda etken tarafından meydana getirilen oksidatif stresin, kanatlı ve memeli hayvanlarda söz konusu olumsuz etkilerin güncel literatürlerin ışığı altında oksidatif stres ve antioksidan enzim düzeyleri karşılaştırılarak değerlendirildi.

Keywords: Mammalian, Poultry, Oxidative stress, Antioxidant enzymes.

Kanatlı ve Memeli Hayvanlarda Oksidan ve Antioksidanlar

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Öz

Oxidative stress is viewed as an imbalance between the production of reactive oxygen species (ROS) and their elimination by protective mechanisms, which can lead to chronic diseases. Imbalance in this protective mechanism can lead to the damage of cellular molecules such as DNA, proteins and lipids. Oxidative stress is an active area of research in veterinary medicine and is involved in many organ diseases including sepsis, mastitis, acidosis, ketosis, enteritis, pneumonia, cancer, respiratory and joint diseases. In this review, the negative effects of oxidative stress caused by many factors in poultry and mammals were evaluated by comparing oxidative stress and antioxidant enzyme levels in the light of current literature.

Anahtar Kelimeler: Memeli, kanatlı, oksidatif stres, antioksidan enzim

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Introduction

Animals use more than 90% of the total oxygen molecule (O₂) in the electron transport chain (ETS) to sustain their lives. In ETS, molecular oxygen is reduced to water by taking electrons from NADH and FADH₂, which are derived from fuels (glucose, fatty acid, and the carbon skeleton of amino acids). In this process, the strong oxidizing power of the oxygen molecule is converted into the high-energy phosphate bond of Adenosine Triphosphate (ATP). ATP is important for specific metabolic processes such as reactions that require molecular oxygen but do not match the formation reaction of ATP, catabolism of amino acids, detoxification of drugs, and synthesis of steroid hormones. 5-10% of the inhaled molecular oxygen tends to form highly Reactive Oxygen Species (ROS) (Halliwell, 1990; Kohen and Nyska, 2002; Thomas 1995).

Under normal conditions, both mammals and poultry have enzymatic or nonenzymatic systems called "antioxidants" that can reduce, stop, clear and prevent cell damage of low molecular weight ROS and other prooxidants (Sen et al., 2010; Shinde et al., 2012). However, in the organism exposed to large amounts of physical, toxic chemical and biological materials, these metabolites undergo various metabolic processes and cause the formation of ROS such as singlet oxygen, hydroxyl radicals, peroxy radicals and hydrogen peroxide in the cell membrane, tissue, organs, protein, carbohydrate, lipid and DNA. It causes oxidation in its metabolism, causing the formation of many diseases from gastrointestinal diseases to infertility, from cardiovascular diseases to respiratory and excretory system disorders (Bollengier et al., 1998; Sen et al., 2010; Shinde et al., 2012). Oxidative stressors can be toxic chemicals, wastes (heavy metals, industrial chemicals) and viral (hepatitis viruses, HIV/AIDS, SARS, MERS viruses) or microbial origin (*Mycobacterium tuberculosis*, *Streptococcus* and *Pseudomonas*, *Shigella*, *Campylobacter* and *Salmonella*). Mammals and poultry, which are exposed to these factors, are directly or indirectly adversely affected and face acute or chronic diseases. Polyunsaturated fatty acids have been added to poultry and mammalian feeds to reduce these negative effects. However, it has been found that this disrupts the polyunsaturated fatty acid composition of animals and increases their susceptibility to lipid oxidation. In addition, it has been supported by studies that these products affect the consumability, shelf life and quality of these products negatively (Bollengier et al., 1998; Yeşilbaş, 2009).

In this context, in this review, stress sources caused by many factors and the effects of these sources on oxidation and antioxidants in liver tissue in mammals and poultry were compared.

Free Radicals and Sources of Oxidative Stress in Animals

Stress is defined as the reflex reaction of the organism to harmful environmental conditions, which inevitably occur in animal breeding and can lead to many undesirable consequences, from various diseases to

death (Yarsan and Gülçin, 2003). Oxidative stress refers to the excessive production of reactive oxygen species (ROS) in the cells and tissues and antioxidant system cannot be able to neutralize them. Imbalance in this protective mechanism can lead to the damage of cellular molecules such as DNA, proteins, and lipids (Güven 2003). Evidence indicated that oxidative stress plays a pathogenic role in chronic inflammatory diseases. Damage of oxidative stress such as oxidized proteins, glycosylated products, and lipid peroxidation results in neuron degenerations mostly reported in brain disorders (Güven et al., 2019; Güven et al., 2021). The liver, the largest organ in the body after the skin and also the largest compound gland, is located in the abdominal cavity. It has various vital functions such as bile production, storage of fat, glycogen, and some vitamins (Vitamins A and B), detoxification, synthesis (fibrinogen, protombin, globulin etc.), phagocytosis and blood production in the embryonal period, and disposal of metabolic wastes. The liver is an exocrine gland because it discharges its secretions into the duodenum through the bile ducts, and an endocrine gland by giving the synthesized substances directly to the blood (Bioulac-Sage, et al, 2007). When animals convert the nutrients they take into energy using oxygen, free radicals with one or more unpaired electrons are formed. These radicals react rapidly with other molecules to share their missing electrons. It takes an electron of the molecules that react with them and makes it reactive, and these reactions continue as a chain (Güven et al., 2003; Siegel, 1985). The harmful effects of free oxygen radicals are removed from the body by antioxidants. Those that are not removed are stored in tissues and play a role in the pathogenesis of many diseases such as aging in organs, atherosclerosis, neurodegenerative diseases, cancer, allergy, diabetes, mastitis, ketosis, joint problems and cataracts. These radicals that cause oxidative stress are superoxide anion (O₂⁻), hydrogen peroxide (H₂O₂), hydroxyl radical (OH⁻), singlet oxygen (O₂), nitric acid (NO), peroxy radical (ROO), hydroperoxy radical (HO₂) is the alkoxy radical (RO) (Kohen and Nyska, 2002). Reactive oxygen species such as singlet oxygen, hydroxyl radicals, peroxy radicals and hydrogen peroxide are continuously produced by various oxidase enzymes within cells due to the degradation of the superoxide anion form formed by electron loss during mitochondrial respiration (Arthur, 2000). It contains free oxygen radicals, especially reactive oxygen, which are formed as a result of endogenous and exogenous reactions. These radicals, which have a very short lifespan, are a source of oxidative stress that reacts rapidly in the cell. Oxidative stress is caused by physical (heat, cold, effort) and chemical wastes (heavy metals) and viral (hepatitis viruses, HIV/AIDS, SARS, MERS viruses) or microbial origins (*Mycobacterium tuberculosis*, *Streptococcus* and *Pseudomonas*, *Shigella*, *Campylobacter* and *Salmonella*). There are many parameters for the degree of oxidative stress in the

organism. The measurement of aldehyde products formed as a result of lipid peroxidation is one of the most used methods. Aldehydes, which are formed by the breakdown of lipid hydroperoxides and have biological activity, are metabolized at the cell level or diffuse from their initial domains and spread damage to other parts of the cell. Lipid peroxidation ends with the conversion of lipid hydroperoxides to aldehydes and other carbonyl compounds and volatile gases such as ethane and pentane in both mammals and poultry (Arthur, 2000; Canoruç et al., 2001).

Physical Factors

Many factors such as hot and cold weather, frequency of being kept together, excessive effort, transportation, noise, behaviour of the caregiver, diseases can cause stress in animals. After a while, this turns into oxidative stress in the cells, as a result of which the number of free radicals normally formed in the cells increases. The most important stress factors in animal husbandry are climatic (heat, cold), environmental (light, dark, transport, altitude), nutritional (excessive salt, food shortage), physiological (electric shock, anesthesia), physical (immobility, crowd),

poor care conditions (health of breeders, number of animals per unit area, poor hatching conditions, errors during transportation, feeding, etc.), social (changes in group structure) and psychological (fear, noise) factors (Canoruç et al., 2001; Mench et al., 1986).

Moving, high effort

It is known that regular physical activity (exercise) has many beneficial effects on health. Under normal conditions, exercise is a condition that relaxes the metabolism, but the excess temporarily increases the production of reactive oxygen derivatives (ROS) in the skeletal muscle. It has been reported that different types of aerobic or anaerobic acute exercises increase the production of free radicals, affect the antioxidant defense system in different ways and create oxidative stress (Radak et al., 2007).

Temperature and light

It is stated that when the temperature rises or falls against the organism, it negatively affects the use of nutrients, reducing performance and productivity, increasing the incidence of diseases by weakening the antioxidant defense system, and as a result of all these, it causes economic losses, especially in the poultry industry (Bollengier et al., 1998)

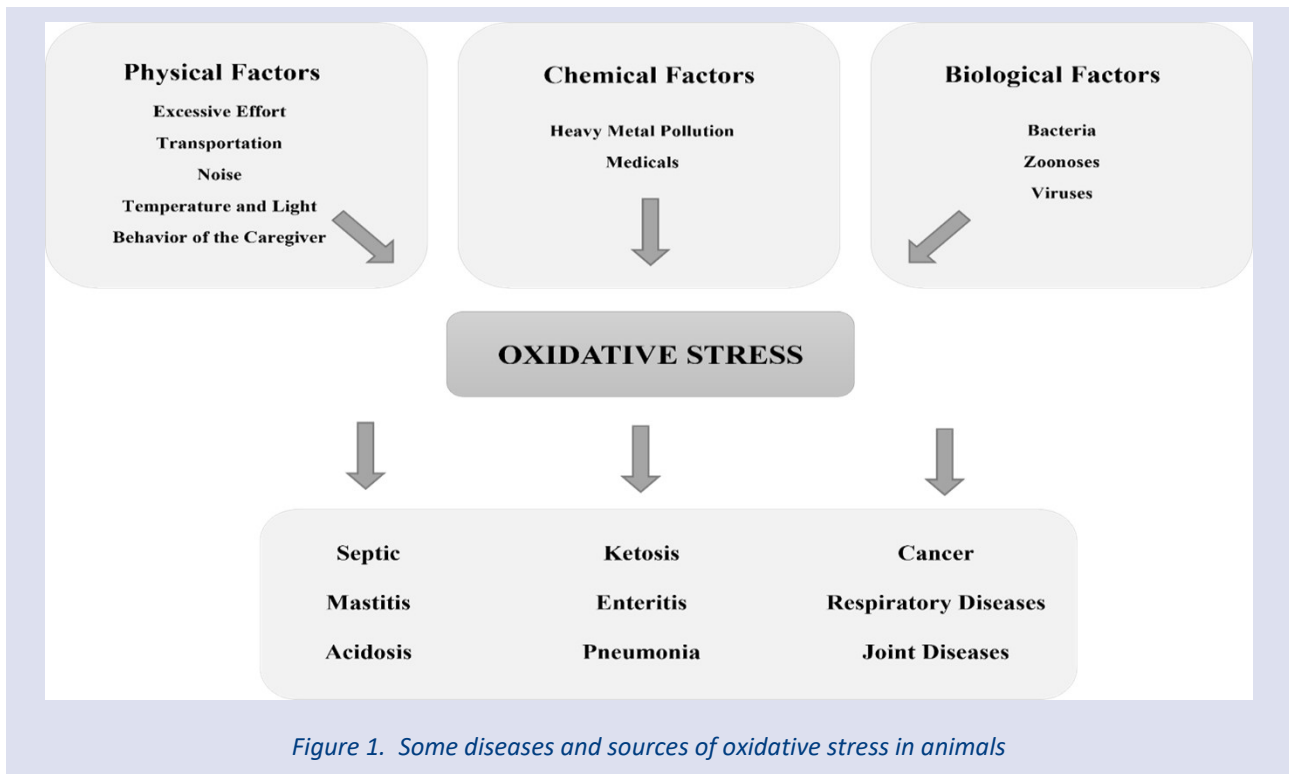


Figure 1. Some diseases and sources of oxidative stress in animals

Chemical Factors

Studies have shown that heavy metal pollution poses a greater threat to ecosystems day by day, threatening the life of all living things. Here, the common mechanism determining toxicity is oxidative stress formation (Tunca, 2012). Oxidative stress caused by chemicals is toxic and lethal. Heavy metal pollution, especially mercury (Hg), lead (Pb), cadmium (Cd) and arsenic (As), poses a threat to all living things. Fe; While it is a more effective metal in promoting oxidative reactions, Cu-catalyzed reactions

have not been fully elucidated yet (Güven et al., 2009; Shind et al., 2012)

Biological Factors and Enzymatic Oxidations

Bacteria, some zoonoses and viruses are the leading biological factors that cause oxidative stress by causing various diseases in mammals and poultry. In the case of oxidative stress, aldehydes (MDA), the main by-product of lipid peroxidation, accumulate in tissues and peripheral circulation (Palanisamy et al., 2011).

Antioxidant System

Industrial chemicals that cause environmental pollution, drugs, additives, harsh physical conditions, biological factors may reduce the catalysts of enzymes in the organism or impair their activities by changing them chemically. The effects of harmful reactive species are kept under control by different natural defense systems in the body (Arman et al., 2019). The enzymes that control the formation of free radicals in animals and that are specialized to prevent the harmful effects of these molecules are called antioxidants. Antioxidant compounds increase the body's defense capacity with their unique mechanism of action. It does this in two ways: first, it involves inhibitory mechanisms such as removing initiating reactive compounds such as hydrogen peroxide and metals that catalyze reactions such as free iron, and reducing oxygen concentration; the second includes direct mechanisms such as collecting free radicals, suppressing their activities by adding protons to them, renewing-repairing radicalized antioxidants or molecules, and breaking auto-oxidation (Güven et al;2003; Benzie, 2003). Enzymes such as Glutathione (GSH), Glutathione peroxidase (GSH-Px), Glutathione S-transferase (GST), Katalase (CAT), Superoxide dismutase (SOD) in the primary antioxidant category. Secondary antioxidants are compounds such as vitamin C, vitamin E, uric acid, bilirubin and polyphenols that capture oxygen radicals and break radical chain reactions (Halliwell and Gutteridge,1999; Kaya and Güven, 2008).

GSH

It is a non-protein tripeptide with multiple cellular functions such as detoxification of electrophilic xenobiotics and removal of ROS from cells. As a protective agent, GSH acts as a cofactor for enzymes involved in the synthesis of leukoerythrin and some enzymes working in different metabolic pathways, including glyoxylases. GSH is also involved in protein folding and degradation of proteins carrying disulfide bonds such as insulin (Halliwell and Gutteridge, 1993). Tissue GSH level is not only regulated by the enzymes involved in the synthesis, it is also very important that the amino acids containing thiol are sufficient. GSH, which can be synthesized in vivo and partially absorbed from the small intestine, is an endogenous and exogenous antioxidant. GSH-radical (GS-) is formed by the oxidation of glutathione. GS- combines with another GS- to form oxidized GSH (GSSG), which is reduced to GSH by NADPH-dependent GSH-reductase. GSH is also the most abundant intracellular thiol (Kidd, 2004). Glutathione, a water-soluble thiol found in very high concentrations in many cells, protects biological membranes against lipid peroxidation. This protection takes place enzymatically. Glutathione also reacts with many harmful oxidants such as singlet oxygen (1O_2), superoxide anion ($\cdot O_2^-$), hydroxy ($\cdot OH$) radicals without enzyme catalysis (Larson, 1988).

GSH-Px

Thiol groups are cellular antioxidants that act through enzymatic reaction and scavenging free radicals. In addition to being a cofactor of the peroxidase (GSH-Px) enzyme family, glutathione is involved in many metabolic

processes that ensure ascorbic acid metabolism, maintain intercellular communication and generally prevent oxidation and cross-linking of sulfhydryl(-SH) groups of proteins. The GSH-Px enzyme that converts the reduced form of glutathione (GSH) to its oxidized state (GSSG) (Shinde et al., 2012; Siegel, 1985).

GST

Glutathione S-transferase (EC.2.5.1.18) is a multifunctional enzyme that provides homeostasis by catalyzing the first step in the formation of mercapturic acid, the end product in the detoxification metabolic pathway. GST is found in the cytosol and membranes of all tissues in the body, such as liver, lung, kidney, muscle, breast, testicles and large intestines and plasma. It protects cells against oxidative damage by providing conjugation of glutathione with free radicals, lipid peroxides and xenobiotics. Thus, it protects the organism from many pathological conditions such as heart, liver, muscle, cancer, diabetes and rheumatoid arthritis caused by oxidative stress. GSTs detoxify a broad spectrum of xenobiotics, including chemotherapeutic drugs, environmental carcinogens, and endogenous molecules. The resulting glutathione conjugate is less toxic and is excreted in soluble form (Sen et al, 2010; Yeşilbağ, 2009). It is found in mammals, insects, fish, birds, annelids, mollusks and many microorganisms. It is present in the cytosol and membranes of all tissues in the body, such as liver, lung, kidney, muscle, breast, testes, large intestines and plasma, and protects cells against oxidative damage by conjugating glutathione with free radicals, lipid peroxides and xenobiotics. Thus, it protects the organism from many pathological conditions such as heart, liver, muscle, cancer, diabetes and rheumatoid arthritis caused by oxidative stress (Arı and Dere, 2003; Koç and Alptekin, 1990). GST is a multi-substrate enzyme. GSH has a G site specific to its co-substrate and an H site to which hydrophobic electrophilic substrates are attached. The thiol group of GSH faces the open part of the pocket. The group that binds to other substrates is this thiol group. GST, which is a multi-substrate enzyme, is calculated at different CDNB and GSH concentrations in calculating the K_m and V_{max} values of enzymatic reactions with and without inhibitor (Anton and Johannes 1990; Boyer, 1989; Güven et al., 2003).

SOD

Superoxide dismutase, which is in the primary antioxidant category, is capable of destroying free radicals. Toxic H_2O_2 , which is formed as a result of the activity of the SOD enzyme, is converted into water and oxygen by the effect of the enzyme "catalase". This enzyme reduces the effect of these radicals by catalyzing the conversion of superoxide anion to hydrogen peroxide and oxygen. In this case, Zn, which forms the active site of the SOD enzyme, is an important mineral. However, in reality all aerobic organisms have been found to contain SOD (Duthie et al.,1989).

CAT

The enzyme catalase, known as a metalloenzyme, is one of the most effective protein catalysts that promotes

the redox reaction. Although H₂O₂ does not react specifically with most of the molecules of biological importance, it plays a role as a precursor in the formation of more reactive oxidants such as the OH radical. Peroxidases also have the same properties as catalase enzyme (Larson, 1988)

Antioxidant Levels in Mammals and Poultry

In the world where global warming is felt more and more every hour, environmental disasters occur due to the change in the ecological qualities of the environment. In the natural environment, pollutants pass into the body of living organisms through nutrients and environmental factors. (Sharifi-Rad et al., 2020). Radical changes in environmental factors, taken toxins and drugs cause liver to be affected the most, as they are metabolized in the liver. Meanwhile, liver cells work to keep the production of oxygen species in balance. Imbalance and oxidative damage following the redox reaction often lead to a range of diseases without jaundice, such as subclinical hepatitis, inflammatory necrotic hepatitis, liver cirrhosis, and cancer (Zhu et al., 2015).

While the liver eliminates the bacteria coming through the blood with its filter function, it harms the harmful substances that cannot be removed from the kidneys with detoxification. It also undertakes many tasks such as the metabolism of carbohydrates, proteins, fatty acids and vitamins.

Table 1. Antioxidant levels in poultry liver/blood tissue

Enzymes	CHICKEN	GOOSE	QUAIL
MDA	5.46±0.38 (nmol/mg protein)	8.9	12.29±002
GSH	0.113 (nmol/g doku)	5.37±0.14 (µmol/g doku)	0.011±0.2 (nmol/mL)
GSH-Px	28.02 (U/ml)	47.5±14 (U/g prot)	0.05±0.01 (U/g Hb)
GST	25.08 (U/ml)	956.70±33.97 (nmol/min mg prot)	15,86 EU/m prot
CAT	3.500 (nmol/dk/mg doku)	186±22 (k/g prot)	1544±229 U/gtissue
SOD	0.127 (nmol/dk/mg doku)	58±2.8 (U/g prot)	1.24±2.16 (U/gHb)
Liter	(30,31)	(10)	(32-34)

The liver is one of the organs most affected by oxidative stress. Therefore, it is possible to see the metabolism and effects of antioxidant enzymes predominantly in this organ (Thomas, 1995; Güven, 2021). Responses to stress in birds under stress are considered to help the animal cope and survive. Poultry can neutralize the free radicals they produce under normal physiological conditions with enzymatic and non-enzymatic antioxidant defense systems. For example, while alpha tocopherol (Vit E) prevents H₂O₂ production by breaking the lipid peroxidation chain by providing electrons to the free radical (oxidant) producing step of the lipid peroxidation

reaction, GPx can neutralize the formed H₂O₂ radical by converting it to water (H₂O) (Çelebi et al., 2016). It has been suggested that the formation of oxygen-centered free radicals with temperature change and the oxidative stress that occurs when the production of cytotoxic oxidants increases, may partially mediate temperature-induced cell damage (Lord-Fontaine et al., 2002). In a study, the concentration of thiobarbituric acid reactive products (TBARS) increased significantly in the liver but did not change in the heart, indicating that the liver was more sensitive to oxidative stress during temperature exposure than the heart (Lin et al., 2002). This may be related to the liver's high content of unsaturated fatty acids and relative changes in the antioxidant system. Direct absorption of light by a molecule can cause electron transfer processes that can produce superoxide anion. It is necessary to maintain a balance between the body temperature of poultry and the temperature of the environment they are in. A large part of the energy generated by the physical activities of the animals in the poultry, especially their feeding, is excreted from the body. Some of the generated energy stays in the body. In order for the excess energy to be discharged, the house temperature must be at a temperature that will absorb the wasted heat. Animals exposed to temperatures above or below the thermoneutral temperature limits are exposed to cold or heat stress and lose their ability to dissipate heat quickly and effectively at high temperatures. In addition, high environmental temperature reduces appetite and decreases live weight gain by reducing feed consumption (Altan et al., 2003; Esmail, 2002).

In the case of sudden and rapid exertion, oxidative stress occurs due to oxygen consumption. Reddy and Fernandes (1999) stated that training done on the treadmill for 8 weeks, 6 days a week, for 45-50 minutes in rats caused a significant increase in antioxidant enzyme activities of liver, kidney and heart muscle compared to the control group. Transport in farm animals is considered one of the most critical points in animal production, as it causes yield losses and affects animal welfare (Grandin, 2000; Mormede et al., 1982). Increased free radicals during transport cause peroxidation of cell lipid membranes. In this process, it was observed that malondialdehyde (MDA) and nitric oxide (NO) levels increased as a result of contractions, energy production and increased oxygen input to the organism. On the other hand, the oxidant-antioxidant balance may be disrupted in favour of radicals as a result of the increase in epinephrine and other catecholamines due to stress, and the increase in enzyme activities such as lactic acid, lactate dehydrogenase, and creatine phosphokinase (Freeman and Crapo, 1982; Pregel et al., 2005). It has been suggested that the deteriorated oxidant-antioxidant balance plays a role in the pathophysiology of diseases that occur after transport in cattle (Urban Chmiel, 2006; Yılmaz and Bahçelioglu, 2000).

Toxic halogenated hydrocarbons and nitrogen oxides known as air pollutants in contaminated drinking water

are also sources of oxidative stress. Yilmaz and Bahçecioglu in a study they conducted, found that liver MDA levels were higher and GSH-Px and glucose-6-phosphate dehydrogenase activities were lower in rats with liver cirrhosis with carbon tetrachloride (CCl₄) compared to controls (Yilmaz and Bahçecioglu, 2000). This means that hydrocarbons such as CCl₄ and bromotrichloromethane (CBrCl₃) are effective in the initiation of oxidative damage in biological systems (Chen and Tappel 1996; Güven and Kaya, 2005). In a study conducted to determine the effect of acute cadmium intoxication on tissue antioxidant enzyme activity in rat testis, antioxidant enzyme activities were found to be significantly higher in the cadmium administered group compared to the control group ($p < 0.05$). In addition, microscopic findings indicating testicular damage were observed in this group (Delibaş and Aydın, 1996). In the experimental cadmium toxicity study, it is stated that live

weight loss and oxidative damage occur in rats (Güven et al., 2003). In the study they conducted in cattle infected with *Theileria annulata*, it was determined that the decrease in CAT, GSH-Px, vitamin E and Vitamin C and the increase in MDA levels are indicators of the development of oxidative stress in the disease (Kızıl et al., 2011). While changes occur in MDA levels, antioxidant enzyme activities and NO levels in cirrhotic liver tissue with *Fasciola hepatica*, it has been stated that there may be resistance to lipid peroxidation due to the severe fibrotic nature of the liver tissue and impaired cell membrane structure (Benzer and Ozan, 2003).

Responses to stress in animals under all kinds of stress are considered to help the animal cope and survive. Unfortunately, the chronic course and persistence of stress negatively affect the immune and reproductive systems, as well as metabolism and energy balances.

Table 1. Antioxidant levels in liver blood/tissue in mammals

Enzymes	RAT	MICE	RABBIT	COW	SHEEP
MDA	234.2±76.85 (nmol/g (dry weight))	0.010±0.001 (µmol/mg prot)	4.13±0.68 (nmol/gr)	7.52 ±0.49 (nmol/mL)	2.57±0.07 (nmol/mL)
GSH	233±0.16 (nmol/g (dry weight))	436.1±9.87 (nmol/mg doku)	0.6±0.01 (nmol/gr)	3.70 ±0.22 (nmol/g Hb)	0.120±0.01 (nmol/mg doku)
GSH-Px	34.3±8.00 (nmol/g (dry weight))	3655.56±741.4 (U/mg prot)	40.09±11.19 (IU/gr.prot)	25.49 ±1.78 (U/g Hb)	36.90±1.25 (Umg prot(dry weight))
GST	2.3±0.52 (µmol (CDNB dry weight))	57±1,92 (nmol/dk/mg protein)	2.8 (IU/mg prot)	24.84 ±0.59 (k/g Hb)	0.112 Unit/ml
CAT	29±2.75 (k/mg prot dry weight)	1784.88±599.7 (U/mg prot)	21.3 (IU/mg prot)	0.20 ±0.012 (U/g Hb mL ⁻¹)	1.222±0.72 (nmol/dk/mg doku)
SOD	11.92±0.41(U/g protein)	680.67±135.7 (U/mg prot)	21.3(IU/mg prot)	-	0.510±0.05 (nmol/dk/mg doku)
Lit	(10,35)	(36-37)	(38-39)	(40)	(41,43)

Conclusion

The organism has various regulatory control systems and changeable behavioural programs. Free radicals and oxidants are involved in physiological signaling pathways, although an imbalance between pro-oxidant and antioxidant systems in favor of the former leads to major biomolecular damage (Mas-Bargues et al., 2021; Güven, 2021). The formation and activity of these oxygen species, which can attack intracellular and extracellular components by damaging cell functions, must be controlled. Knowing the reactive species and reaction mechanisms is very important in terms of controlling them. It has been shown that the liver is the most suitable tissue for the detection of lipid peroxidation (Güven et al., 2003; Güven and Kaya; 2005; Urbanova, 1974). In addition, antioxidants may give different results from one animal species to another due to differences in tissue, sex

and environmental factors (Gather et al., 1976; Yaralioglu and Özdemir, 2018). In this context, in this review, which was prepared by conducting a wide literature review, the functioning of the oxidant-antioxidant system and malondialdehyde (MDA) and nitric oxide (NO) levels and GSH, GSH-Px, GST, CAT and SOD values in poultry and mammal liver tissue samples were tried to be revealed. The possible reason for the differences may be the biological structure of the organism and their different responses to the same living conditions and different oxidant damage mechanisms between species. Due to their physiology, laying hens have a higher metabolism than ruminants, and a high level of laying physiology makes these animals prone to oxidative stress (Şimşek et al., 2013).

In conclusion, liver tissue lipid peroxidation and antioxidant mechanisms differ between mammals and poultry and within themselves. More studies are needed

to protect and control the animal species of these parameters. In addition, it may be important for animals to consume rations consisting of feed materials containing antioxidative system supporters that they need in order to ensure healthy, desired level of development and productivity.

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