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Review Article

Oxidative Stress in Animals Exposed to Different Stressful Conditions

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Abstract

The terms oxidative stress, oxidative damage, free radical and antioxidant have become an integrated part of the variety of scientific discussions in issues related to chemistry, biology and research in biosciences. Oxidative stress commonly occurs following heat stress in tropical regions and affects animals and Glutathione protects cells from oxidative damages. Glutathione has been mediating the initial response for acquiring tolerance to heat stress. Glutathione serves vital functions in animals, antioxidant defense, scavenging free radicals and other reactive species, removing hydrogen and lipid peroxides, preventing oxidation of bio-molecules, signal transduction and gene expression and DNA and protein synthesis, and proteolysis. The ATP-ase and cholinesterase enzymes take the major role in metabolic pathways inside the living cell. There was a significant inhibition of total ATP-ase activity and cholinesterase enzymes in animals under heat stress conditions.

Keywords: Oxidative stress; Antioxidants; Glutathione; Choline-Esterase; Adenosine triphosphate; Heat stress; Animals

Introduction

The dietary of antioxidant nutrients is important in protecting tissues against free radical damage, since free radical reactions are the integral part of normal metabolism. The antioxidant function enhances immunity by maintaining the structural and functional integrity of immunity system. The reduction in immunity will affects animal production efficiency through increased susceptibility to diseases, thereby leading to increased animal morbidity and mortality [1]. The protection against free radical damage by the dietary of antioxidant nutrients has become very important in the studies related to ruminant production and reproduction. Antioxidant status gives complementary information about the metabolic status of the animal rather than metabolic parameters alone [2]. Oxidative stress markers can be divided into non enzymatic antioxidants and antioxidant enzymes such as Superoxide Dismutase (SOD), catalase and glutathione peroxidase [3].

The important role of some antioxidants like glutathione, Adenosine-Tri Phosphate (ATP) and cholinesterase enzymes activities in relation to heat stressed animals was reviewed.

Oxidative stress and heat stress conditions

High ambient temperature and humidity are the major constraint on animal productivity in tropical and subtropical areas. Oxidative stress commonly occurs following heat stress in tropical regions and affects animals and Glutathione protects cells from oxidative damages. The oxidative balance is affected during heat stress periods. Fast production of free radicals and reactive oxygen species and/or a decrease in antioxidant defense mechanisms result in oxidative stress [4]. The effect of heat stress is known to induce oxidative stress which induces production of Reactive Oxygen Species (ROS). The high production of ROS and a decrease in antioxidant defense, leads to cause of many diseases and leading to the onset of health disorders in cattle [5].

The role of glutathione as oxidative stress

Glutathione (GSH) is an antioxidant in plants, animals, fungi, and some bacteria and archaea. Glutathione is capable of preventing damage to important cellular components caused by reactive oxygen species such as free radicals, peroxides, lipid peroxides, and heavy metals [6] GSH is a tripe tide with a gamma peptide linkage between the carboxyl group of the glutamate side chain and the amine group of cysteine, and the carboxyl group of cysteine is attached by normal peptide linkage to a glycine. Glutathione (GSH, γ- glutamyl - L-cysteinyl-glycine) as antioxidant either in reduced (GSH) or oxidized (GS-SG) form is one of the key constituent in the ant oxidative system with a significant function in reactive oxygen species scavenging and act as redox buffer to keep the cellular redox state in balance [7]. GSH is an antioxidant, preventing damage to important cellular components caused by reactive oxygen species such as free radicals and peroxides. GSH may also function in maintaining the integrity of the red cell by reducing sulphydryl groups of hemoglobin, membrane proteins and enzymes that may have become oxidized [8]. An important function of Glutathione in the red cell is the detoxification of low levels of hydrogen peroxide which may form spontaneously or as a result of drug administration [9]. GSH participates in leukotriene synthesis and is a cofactor for the enzyme glutathione peroxidase. It is also important as a hydrophilic molecule that is added to lipophilic toxins and waste in the liver during biotransformation before they can become part of the bile [10]. Glutathione is also needed for the detoxification of methylglyoxal, a toxin produced as a by-product of metabolism. GSH is the major endogenous antioxidant produced by the cells, participating directly in the neutralization of free radicals and reactive oxygen compounds, as well as maintaining exogenous antioxidants such as vitamins C and E in their reduced (active) forms [11]. GSH has a vital function in regulation of the nitric oxide cycle,

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which is critical for life but can be problematic if unregulated [12]. GSH is used in metabolic and biochemical reactions such as DNA synthesis and repair, protein synthesis, prostaglandin synthesis, amino acid transport, and enzyme activation [13]. GSH effectively scavenges free radicals and other reactive oxygen species (e.g., hydroxyl radical, lipid peroxyl radical, peroxynitrite and H2O2) directly and indirectly through enzymatic reactions and in such reactions. GSH is oxidized to form GSSG, which is then reduced to GSH by the NADPH-dependent glutathione reductase. In addition, GSH plays a role in diverse biological processes as protein synthesis, enzyme catalysis, trans-membrane transport, receptor action, intermediary metabolism and cell maturations [14]. Moreover, GSH is necessary for the hepatic action of insulin-sensitizing agents indicating their critical role in regulating lipid, glucose and amino acid utilization [15]. Free glutathione is mainly present in its reduced form (GSH), which can be converted to the oxidized form (GSSG) during oxidative stress. Glutathione reeducates can convert GSSG to GSH. Reduction and conjugation reactions are the most important functions of GSH [16]. The GSH protects cell membranes from oxidative damage by removing of produced reactive species [17].

Heat stress and glutathione level

The adverse effect of the heat stress had a negative impact on enzymatic activity. Different kinds of stress result in reduction in the concentration of reduced Glutathione (GSH) in animal organs [18]. A decrease in GSH and an increase in GSSG were found in the blood of heat-stressed cattle [19]. The authors found that mean GSH concentrations for thermo-neutral and heat stress were 3.2±0.65 and 2.7±0.62 mmol/L of RBC, respectively and reduced GSH concentrations were associated with reduced feed intake during heat stress period. The same authors concluded that heat stress reductions in feed intake and thermoregulatory effects may induce oxidative stress in cattle. The concentrations GSH in the liver and kidney of three genetic groups of rabbits decreased insignificantly due to stress of the influence of displacement of animals from cage to cage daily for 30 days [20]. Same authors concluded that this decrease in the concentrations of GSH in the liver and kidney of rabbits is due to adaptative processes in biochemical reaction of stress and/or by long time of experiments. Heat exposure results in decreases in GSH levels and consequently increase in free radicals leading to increase lipid per oxidation (oxidative stress) in rats [21]. Short-term heat stress was found to decrease GSH levels and increase lipid per oxidation in killifish and when increased acclimation temperature increased total GSH levels and increased glutathione peroxidase and GSH reductase activities [22]. Plasma total antioxidant activity decreases when cows are placed in environmentally controlled chambers and exposed to 29.5°C temperatures for a period of 7 days [23]. The same authors reported that as the Temperature Humidity Index (THI) approaches levels dangerous to livestock, total antioxidant activity declines. In addition same authors reported that hyperthermia results in a transient decrease followed by an increase in GSH levels in the blood, an increase in excretion of hepatic GSH, and an increase in lipid per oxidation. Liver GSH in male rats was significantly decreased after heat stress in both age groups treated with N-acetylcysteine. Serum glutathione level in the ram and its changes during normal and heat stress conditions were determined and reported that glutathione levels change during different environmental conditions [24]. Glutathione has been mediating the initial response for acquiring tolerance to heat stress [25]. The decreased values of GSH on long exposures to temperature stress indicate utilization of this antioxidant, either to scavenge oxiradicals or act in combination with other enzymes, was more than its production capacity under heat stress [26]. Liver supplies most of the plasma glutathione and it is removed from plasma by transpeptidase action which is mostly located in the kidney therefore, glutathione levels may be influenced by different physiological conditions [17].

Heat stress and adenosine triphosphate (ATP) enzyme activity

The Adenosine Triphosphatase (ATP-ase) and cholinesterase enzymes take the major role in metabolic pathways inside the living cell. The ATP-ase and cholinesterase enzymes are irreversibly inhibited by organic phosphorus compounds and most of pesticides. Adenosine Triphosphate (ATP) is a complex organic chemical that provides energy to drive many processes in living cells, e.g. muscle contraction, nerve impulse propagation, chemical synthesis. Found in all forms of life, ATP is often referred to as the molecular unit of currency of intracellular energy transfer. When consumed in metabolic processes, it converts either to Adenosine Diphosphate (ADP) or to Adenosine Monophosphate (AMP). It is also a precursor to DNA and RNA, and is used as a coenzyme. From the perspective of biochemistry, ATP is classified as a nucleoside triphosphate, which indicates that it consists of three components: a nitrogenous base (adenine), the sugar ribose, and the triphosphate. The ATP-ase plays an important role in the production of energy used by the living organisms [27]. The ATP-ase as integral part of the cell membrane, plays an important role in the active transport across cell membrane and considered the primary driving force for other membrane-associated transport system or channels such as Ca^{+2} channel, Na^{+}/Ca^{+2} and Na^{+}/H^{+} antiprotons, and Na^{+}, dependent transporters for phosphate, glucose and amino acids [28]. There was a significant inhibition of total ATP-ase activity of small and large intestinal mucosa of broiler chickens exposed to heat stress (41°C, 65% relative humidity for 6h) as compared to thermo-neutral (25°C, 65% relative humidity) conditions [29].

Heat stress and cholinesterase enzyme activity

Cholinesterase levels in serum are useful as an indicator to possible insecticide poisoning detection of deficiency of enzyme level [30]. Cholinesterase serves a pivotal role in regulating the transmission of nerve impulses by rapid hydrolysis of the neurotransmitter. Cholin-E enzyme activity in some central nervous system regions of albino rat newborns was decreased markedly at day 7, 14 and 21 after exposure to high temperature exposure (40°C two hours daily) as compared to thermo-neutral temperature (25°C) [31]. The cholin-E activity, which controls the acetylcholine content and cholinergic activity, was markedly decreased as a result of heat exposure. Heat stress provoked a decrease in the ch-E activity of the cerebrum region of the gerbil [32]. Contrary to these observations, [33] reported that the activity of ch-E was significantly increased in the brain of rats kept at high ambient temperature (40°C). So, the heat stress may cause the disturbance in the cholinergic functions. These changes may, in turn, cause impairment in the development of neurons, oligodendrocytes and the tissues of the CNS [34]. In conclusion, the heat stress at 40±1°C deleteriously affected the monoamine content and cholinesterase

activity in the examined CNS regions and the withdrawal for 1 week beyond day 21 failed to return these perturbations to normal values.

Conclusion

Glutathione, adenosine triphosphate and cholinesterase enzymes activities are important enzymes as antioxidant for ameliorating the oxidative stress in heat stressed animals.

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