A Review: Oxidative Stress during Lactation in Dairy Cattle

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Introduction

Lactation is an important period in dairy cows from the point of view of physiological changes taking place, which in turn produce measurable changes in the diagnostic parameters of the blood. Oxidative stress plays a key role in the onset or progression of numerous human and animal diseases. High metabolic demands during lactation can impact the oxidative status of dairy cows. Researchers have reported higher oxidative stress in high producing dairy cows when compared to average producing dairy cows. Stage of lactation has also been found to affect the oxidative status of the animal. Dairy cows undergo oxidative stress mainly during the peripartal period. The period of transition between late pregnancy and early lactation is associated with lipid and protein metabolic changes [1]. Oxidative stress is also considered a physiological stress on the secretary activity of the parenchyma and the onset of milk secretion is accompanied by high-energy demand and increased oxygen demand.

Excessive production of free radicals together with damage at the cellular level is controlled by cellular antioxidant defense systems. Antioxidants can be defined as defense substances that delay, prevent or eliminate oxidative damage to a target molecule. Antioxidant enzyme systems can be includes (e.g., superoxide dismutase, glutathione peroxidase and catalase) and non-enzymatic systems (e.g. vitamin E and selenium). SOD and GPx shows significant variation between breed, seasonal effect and breeding seasons with or without supplementation of antioxidant.

Superoxide Dismutase

Superoxide dismutase (SOD) accelerates the dismutation of the toxic superoxide radical to hydrogen peroxide and is considered the first intracellular defense against reactive oxygen species. The cytosol of all eukaryotic cells contains CuZn-SOD. Determination of SOD is important in the evaluation of antioxidant status, under physiological or pathological conditions [2]. SOD is one of the components of intrinsic antioxidant system. It is responsible for dissemination of superoxide radicals. SOD catalyses the conversion of superoxide radical to hydrogen peroxidase.

Significantly higher level of plasma concentration of SOD has been observed in mid lactating cows than early lactation [3]; and from 3 weeks before parturition to 9 weeks after parturition has also been reported [4]. Higher erythrocyte SOD activity during lactation than pregnancy has also been observed [5]. The increased SOD actually observed in the animals of mid-lactation showed high individual variation, possibly difference of age, parity, level of milk production and other inherent stress, might have caused such as high SOD level; further it is to be considered that SOD enzyme is transient in its action and actively shows a spurt on spontaneous induction of stress which may possible until other homeostasis enzyme have removed the association ROS and vice versa. The rise in SOD activity during early and mid lactation is a marker of oxidative stress. Higher serum SOD activity might be due to physiological upgrading of this enzyme in an attempt to neutralize/mitigation of superoxide radical challenges and adaption of animals to oxidative stress in an attempt to improve the antioxidant status.

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Glutathione Peroxidase

Glutathione peroxidase (GPX) plays an important role in cellular antioxidant defense. Determination of GPX activity might be beneficial in the evaluation of selenium status and in the evaluation of antioxidant status [2].

The enhanced lipid mobilization to satisfy the increased energy requirement for milk production disrupts several inflammatory and immune functions [6] and promotes free radicals production by leukocytes and endothelial cells [7]. In physiological conditions, the antioxidant defense system, provided by enzymes and antioxidants, scavenges reactive oxygen species (ROS), thus limiting or preventing oxidative damage. An increased production of free radicals or deficiencies of antioxidants may lead to oxidative stress, which impairs physiological functions, thus contributing to health disorders in lactating animals [8]. As a matter of fact, oxidative stress can increase the susceptibility of dairy cattle to several diseases and metabolic disorders, particularly during the transition period.

Gpx catalyzed the conversion of H2O2 to H2O produced in the course of normal cellular events. It is also catalyses the reduction of fatty acid hydroperoxides and 1-monoacglycerol hydroperoxides. Another GPxs in RBCs termed phospholipid hydroperoxides glutathione peroxidase participate in reduction of more complex phospholipid hydroperoxides using GSH. Selenium deficiency can be diagnosed by measuring decreased RBC GPX activity in some species, however caution indicated in using this activity as a direct indicator of selenium status because polymorphism in GPX activity may be present [9].

The maximum stress during early lactation is attributed due to peak lactation and more colostrum secretion by mammary gland. There may be further decrease in GPX level at mid lactation as compared to control values. Possible reason for this might be the physiological adjustment and adaption of animal body to lesser production during mid-lactation and supply of nutrients. A decrease of mean blood GSH-Px in dairy cows during mid-lactation might be due to a loss of homeostatic control in the postpartal period [10].

In contrast, opposite patterns, by means of, higher GPX level during late gestation as compared to both stages of lactation [10], and, in terms of a significant higher glutathione peroxiaase (GPx) level in mid lactating cows than early lactation, and a decrease in GPX level during early lactation than during advanced pregnancy but no significant difference have been observed [3,11]. So it can be concluded that the numbers of factors including various physiological and environmental factors may be responsible for the oxidative stress. Superoxide dismutase catalyses the dismutation of superoxide radical to hydrogen peroxide which is further metabolized to water by GSH-Px enzyme.

This review gives evidence that the lactating animals undergo substantial metabolic and physiology adaptation during transition from non-lactation and early lactation to mid lactation that contribute to dysfunctional host inflammatory responses [12]. The rise of oxidative stress markers could be due to pregnancy and early lactation which are considered as stressful stages accompanied by a high metabolic demand and elevates the requirements for tissue oxygen [13] and causes an increase of reactive oxygen species production. During lactation in order to sustain lactogenesis and fatty acid consumption from the mother’s fat reserve and production of hydrogen peroxide that has been enhanced by intense lipolysis and mobilization of fatty acids from the body deposits [2].

The Se status is most frequently assessed either directly from Se concentrations, or indirectly from GSH-Px activity assessment [14]. Selenium is a very important essential trace element for proper intrauterine and postnatal development of calves. Although Se passes both placental and mammary barriers, placental transfer is more effective than the transport of Se into milk [15].

References


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