Effects of Dietary Antioxidants on Muscle Tenderness and Mechanism of Action: Postprint

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Abstract

Tenderness is one of the important sensory indicators of muscle quality, determining its nutritional and eating qualities. During intensive animal production, the inevitably generated oxidative stress exerts significant adverse effects on muscle quality; therefore, exploring effective measures to mitigate the impact of oxidative stress on muscle tenderness is of paramount importance. Based on the current research status of meat tenderness both domestically and internationally, this paper primarily elaborates on the effects of oxidative stress and antioxidants on muscle tenderness and their underlying mechanisms, aiming to provide a reference for the efficient and rational utilization of antioxidants to improve muscle tenderness.

Full Text

Effects and Mechanisms of Dietary Antioxidants on Meat Tenderness

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Abstract: Tenderness is one of the most important sensory indicators of muscle quality, determining both the nutritional and eating quality of meat. However, oxidative stress inevitably generated during intensive animal production can exert significant adverse effects on meat quality. Therefore, exploring effective measures to mitigate the impact of oxidative stress on meat tenderness is crucial. Based on current research on meat tenderness both domestically and internationally, this paper primarily elaborates on the effects of oxidative stress and antioxidants on muscle tenderness and their underlying mechanisms, aiming to provide a reference for the efficient and rational utilization of antioxidants to improve meat tenderness.

Keywords: meat quality; tenderness; diet; antioxidants

Since China' s reform and opening up, the livestock industry has developed rapidly. According to the *China Statistical Yearbook* (2016), national meat production reached 86.25 million tons in 2015. With economic development and evolving consumer preferences worldwide, demands for meat products have increased continuously, making the improvement of meat tenderness a long-standing research focus. Tenderness refers to the shear force required to cut muscle with a knife or the overall perception of hardness in the mouth when chewing cooked meat, reflecting its softness and ease of mastication. It is one of the most important indicators for evaluating meat quality, as well as the least stable and most easily disturbed quality parameter [1-2]. Tenderness is typically evaluated through sensory assessment or instrumental methods. During meat production, processing, storage, and transportation, oxidative stress induced by intensive production, malnutrition, long-distance transport, extreme environments, and frozen storage increases peroxidation reactions in the body, making muscle highly susceptible to oxidative deterioration, reducing tenderness and color stability, and shortening shelf life [3]. Consequently, adding exogenous antioxidants to feed to inhibit muscle oxidation has become an effective method for extending shelf life and improving meat quality. Current research on feed antioxidants has primarily focused on their effects on animal antioxidant capacity, growth performance, and general meat quality indicators, while studies on their specific effects on muscle tenderness and underlying mechanisms remain relatively limited. Therefore, elucidating and summarizing the mechanisms by which antioxidants affect muscle tenderness is essential for their rational and efficient utilization.

1 Effects of Oxidative Stress on Meat Tenderness

In animal production, various factors including farming conditions, pre-slaughter environment, and transport stress can cause excessive free radical concentrations in the body, leading to oxidative stress that adversely affects animal health, production performance, and product quality [4]. Oxidative stress causes muscle oxidative deterioration, manifested as discoloration, off-flavors, formation of toxic substances, reduced shelf life, and compromised nutritional value and tenderness [5]. High levels of free radicals reduce muscle sensory quality and tenderness, cause loss of protein function, and deplete essential amino acids such as phenylalanine and tryptophan, making it crucial to understand free radical activity in meat [6-8].

During metabolic reactions, a small portion (2-5%) of consumed oxygen is converted into reactive oxygen species (ROS) [9], which play key regulatory roles in maintaining homeostasis through interactions with proteins, fatty acids, and nucleic acids. However, excessive free radicals in muscle readily react with the abundant polyunsaturated fatty acids in biological membrane structures, initi-

ating chain reactions that generate a series of lipid oxidation products. ROS, reactive nitrogen species, and lipid oxidation products can all induce oxidative modification of proteins or enzymes, altering protein structure and properties and consequently changing enzyme activity, thereby modifying meat tenderness [10-11].

Yang et al. [12] reported that the tenderness of longissimus dorsi muscle in fattening pigs under high temperature stress (30°C) was significantly lower than that of the control group (22°C). Yan et al. [13] found that electric shock driving stress significantly reduced the tenderness of longissimus dorsi muscle in Northeast Min pigs post-slaughter, affecting meat quality. In living animals, relatively low oxidative stress and high antioxidant capacity exist, whereas post-slaughter, particularly during the conversion of muscle to meat, high oxidative stress and relatively low antioxidant capacity prevail to reverse oxidation reactions [14]. This difference between the two states underscores the importance of oxidative stress for meat quality. Due to the complex and diverse factors causing oxidative stress, different oxidative stress conditions exert varying degrees of impact on muscle tenderness, and thus the mechanisms and effects of different antioxidants also differ.

2 Mechanisms of Antioxidants on Meat Tenderness

The foundation of meat tenderness formation lies in the content, physicochemical properties, and internal biochemical status of muscle fibers, connective tissue, and intramuscular fat. All factors affecting muscle tissue structure and internal status can indirectly influence tenderness, including animal breed and genetics, sex, age, feed nutrition, management practices, slaughter and processing methods, and post-slaughter aging. The primary cause of tenderness changes is the degradation of myofibrillar proteins such as desmin, titin, and nebulin. Protein degradation in animals involves multiple protease systems, with the calpain-calpastatin system being the most important [15-16].

Dietary antioxidants improve meat tenderness by enhancing the antioxidant capacity of animals and post-slaughter muscle, inhibiting oxidation of proteins, enzymes, and fats by ROS and reactive nitrogen species, and protecting the structural and functional integrity of muscle proteins and enzymes [17]. Pan [18] conducted an in vitro experiment inducing myofibrillar protein oxidation, demonstrating that oxidation during storage increased carbonyl content, cross-linking degree, and particle size of myofibrillar proteins while reducing total sulfhydryl content, with myosin being most susceptible to oxidative cross-linking. Adding compound antioxidants during meat storage could block protein oxidation pathways and improve tenderness. Li et al. [19] found that dietary vitamin E supplementation significantly improved pork tenderness, while ultrastructural analysis of muscle fibers showed that vitamin E significantly increased sarcomere length and I-band length and decreased myofibril diameter, all of which are closely related to meat tenderness. Zhang et al. [20] reported that licorice extract could inhibit free radical attack on calpain, increasing its activity and

promoting muscle protein degradation, suggesting that antioxidants affect meat tenderness by enhancing calpain activity during aging.

3 Effects of Dietary Antioxidant Supplementation on Meat Tenderness

Adding exogenous antioxidants to feed or muscle can directly or indirectly scavenge free radicals in the body or muscle, improving antioxidant performance. Exogenous antioxidants are diverse, including natural antioxidants such as vitamin E, vitamin C, and -lipoic acid; plant extracts such as isoflavones, tea polyphenols, lycopene, and spice extracts; and other antioxidant compounds.

3.1 Vitamins

Numerous studies have shown that adding certain vitamins to animal feed can improve antioxidant performance in animals and muscle. Vitamin E, a tocopherol compound, possesses multiple biological functions including antioxidant activity, growth promotion, and reproductive enhancement. As an important component of lipid membranes, vitamin E protects membrane structure and function by scavenging free radicals, thereby preventing cytoplasmic leakage and increasing meat tenderness [21]. Vitamin C can scavenge free radicals by donating electrons or restore the antioxidant function of vitamin E, exhibiting synergistic effects. However, many studies have found that vitamin C and vitamin E can act as pro-oxidants when transition metal ions are present or at high supplementation levels, indicating that antioxidants may also exert pro-oxidant effects under certain conditions.

Wang [22] demonstrated that different doses of vitamin E and vitamin C affected tenderness differently across muscle locations. Vitamin C supplementation at 150 and 300 mg/kg significantly improved leg muscle tenderness but had no significant effect on breast muscle tenderness, whereas dietary vitamin E at 100 mg/kg significantly improved breast muscle tenderness. This study also confirmed that individual supplementation of vitamin C or vitamin E could significantly improve antioxidant performance in broilers and muscle during cold storage. Research indicates that vitamin E must accumulate to a certain concentration in animal tissues to affect meat quality, with tissue concentration influenced by dietary supplementation level and duration [23-24]. These findings suggest that different doses of vitamin E and vitamin C may have differential effects on muscle quality across animal body parts, and excessively high or low supplementation may fail to improve certain meat quality indicators.

3.2 Plant Extracts

Many plants and their extracts have demonstrated strong antioxidant functions, primarily attributed to their phenolic and aldehyde contents, including lycopene, tea polyphenols, honeysuckle extract, resveratrol, guava powder, mulberry leaves, and oregano. However, most current research has focused on their

effects on animal antioxidant capacity and growth performance, with few reports on their impact on meat tenderness and mechanisms. Lycopene, a carotenoid found mainly in ripe red fruits such as tomatoes and guavas, exhibits strong antioxidant properties [25]. Jiang [26] showed that lycopene had good antioxidant function both in vivo and in vitro, and dietary supplementation improved lamb tenderness, though not significantly. Tea polyphenols, bioactive substances in tea, primarily include catechins, flavonols, and anthocyanins [27]. According to Ministry of Agriculture Announcement No. 2045, tea polyphenols are approved antioxidants for use in livestock feed. Chao et al. [28] investigated the effects of tea polyphenols on antioxidant capacity and meat quality in fattening pigs, finding that 0.04% dietary supplementation had no significant effect on tenderness but significantly improved antioxidant performance. Yao et al. [29] found that 0.009% and 0.013% tea polyphenol supplementation significantly improved pork antioxidant capacity and tended to improve tenderness, though not significantly. Dietary supplementation with honeysuckle extract, resveratrol, guava powder, and mulberry leaves significantly improved muscle tenderness [30-33]. However, some studies found that oregano essential oil had no significant effect on lamb or pork tenderness [34-35]. These discrepancies may be related to antioxidant type, dosage, experimental duration, and animal factors.

3.3 Other Antioxidants

Many single antioxidants or antioxidant-containing products have demonstrated good antioxidant functions in vivo or in vitro, such as -lipoic acid, microbial additives, and dihydropyridine, though their effects on meat tenderness vary considerably. Guo [36] proved that -lipoic acid significantly improved antioxidant capacity and meat tenderness in broilers. Zhang et al. [37] examined the effects of feeding lactic acid bacteria solution on pork quality and antioxidant capacity, showing that it significantly improved both tenderness and antioxidant performance. Bian et al. [38] found that dietary dihydropyridine supplementation increased pork tenderness, though not significantly. Li et al. [39] showed that feeding rice bran significantly reduced pork tenderness, while supplementation with additives containing glutathione, dihydropyridine, and zinc sulfate significantly improved tenderness. Different antioxidants may have multiple biological functions and varied mechanisms of action on muscle, warranting further investigation into their effects on meat tenderness.

Feed antioxidants primarily function by directly or indirectly scavenging or reducing excessive free radicals in animals, maintaining dynamic free radical balance, improving tissue and muscle antioxidant capacity, and inhibiting lipid oxidation, protein oxidative denaturation, and enzyme activity decline, thereby improving meat tenderness. Antioxidants as feed additives have attracted considerable research attention, with numerous publications focusing on exploring new antioxidants and their effects on animal health, production performance, and overall meat quality. However, systematic research on their specific effects on meat tenderness and underlying mechanisms remains lacking. Future studies

should deeply explore the mechanisms of antioxidants on meat tenderness while ensuring animal health and safety, providing theoretical foundations for rational and effective antioxidant use to reduce adverse effects of stress on meat quality.

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Note: Figure translations are in progress. See original paper for figures.