Research Progress on the Protective Effects of Vitamin E against Animal Organism Damage: Postprint

Authors: Sun Yuecheng, Zhang Jing, Song Wentao, Anshan Shan

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Abstract

Vitamin E is an essential fat-soluble vitamin for animal organisms and an important antioxidant substance in the body. Recent research findings have demonstrated that Vitamin E plays a significant role in protecting animal organisms from damage, maintaining the stability of basic tissue structures, safeguarding reproductive performance, and enhancing immune function. This article summarizes the protective effects of Vitamin E against damage to animal organisms induced by harmful substances such as oxidative stress, mycotoxins, and pesticides, providing a theoretical basis for research on the mechanisms of Vitamin E' s action as a detoxifying and protective agent in animal organisms and for its further promotion and application in animal production.

Full Text

Research Progress on the Protective Effects of Vitamin E Against Animal Body Damage

SUN Yuecheng, ZHANG Jing, SONG Wentao, SHAN Anshan
Institute of Animal Nutrition, Northeast Agricultural University, Harbin 150030, China

Abstract

Vitamin E is an essential fat-soluble vitamin for animal organisms and an important antioxidant substance in the body. Recent research has demonstrated that vitamin E plays significant roles in protecting animal bodies from damage, maintaining stable basic tissue structure, safeguarding reproductive performance, and enhancing immunity. This paper summarizes the protective effects of vitamin E against animal body damage induced by harmful substances such

as oxidative stress, mycotoxins, and pesticides, providing a theoretical basis for further research on the mechanisms of vitamin E as a detoxifying and protective agent in animals and for its expanded application in animal production.

Keywords: vitamin E; oxidative stress; mycotoxin; pesticide residue; antioxidation

Animal oxidative stress damage represents a major factor limiting normal animal growth during production. In practice, contamination of agricultural and livestock products by mycotoxins and excessive pesticide residues have become increasingly serious problems. Statistics indicate that approximately 25% of global grain supplies are contaminated with mycotoxins to varying degrees, while incidents of pesticide residue 超标 (exceeding standard limits) also occur frequently. After ingestion by humans and animals, these toxic substances readily cause oxidative stress damage to liver function, intestinal function, and reproductive performance. Consequently, identifying effective antioxidant substances that can protect against and treat such damage has become increasingly important. As a traditional feed additive, vitamin E has been used alone or in combination with other substances to protect animal organisms, yielding promising research results. This paper summarizes the protective effects of vitamin E against animal body damage induced by oxidative stress, mycotoxins, pesticides, and other harmful substances, providing a theoretical basis for investigating its mechanisms of action as a detoxifying and protective agent and for promoting its further application in animal production.

1 Protective Effects of Vitamin E on Oxidative Stress Damage in Animals

Vitamin E, also known as -tocopherol, is a crucial antioxidant that blocks free radical chain reactions [2]. Current research demonstrates considerable potential for vitamin E in preventing and treating diseases such as cardiovascular and cerebrovascular diseases and tumors [3-4]. This benefit stems from its phenolic compound properties; the hydroxyl group at position 6 of its chromanol ring serves as an active site that can donate active hydrogen to scavenge free radicals. By directly acting as an electron donor, vitamin E eliminates excess free radicals, interrupts free radical chain reactions, and effectively prevents lipid peroxidation in tissues, thereby protecting cells and tissues from free radical attack [5]. This mechanism has led researchers to recognize natural vitamin E as a highly effective antioxidant [6]. The attack of polyunsaturated fatty acids on cell membranes by free radicals constitutes an important cause of cellular lipid peroxidation reactions. As a non-specific chain-breaking antioxidant, vitamin E can protect polyunsaturated fatty acids in cell membrane phospholipids and plasma lipoproteins from oxygen free radical attack [7-8], explaining its protective effects against body damage at the cellular level. Additionally, the combined action of vitamin E with other antioxidant substances is believed to effectively inhibit the oxidation of low-density lipoproteins within cell membranes, particularly oxidation mediated by free radicals [9].

Experimental results show that animal organisms maintain normal immune responses through regulation by relevant neural and endocrine systems. Supplementation with appropriate levels of vitamin E can prevent excessively low or high immune reactions by regulating related hormone levels, thereby ensuring homeostasis and maintaining normal growth [10]. Simultaneously, vitamin E influences lipid peroxidation by affecting tissue antioxidant enzyme and non-enzyme systems, regulating the activity of enzymes such as superoxide dismutase (SOD) and catalase (CAT) in tissue cells, and reducing oxidative stress from oxygen free radicals on cell membranes [11].

As a commonly used feed additive with efficient antioxidant effects, vitamin E has been widely applied in animal production, livestock product processing, human health foods, and clinical medicine. During actual animal production, animals experience varying degrees of oxidative stress due to changes in dietary nutrition levels and living environments, leading to inflammatory infiltration of neutrophils, stimulation of protease secretion, and production of large quantities of oxidative intermediates that cause oxidative stress damage [12]. Supplementing feed with appropriate levels of vitamin E can effectively enhance animal antioxidant capacity while significantly alleviating and improving symptoms such as organ damage and abnormal blood biochemical indicators caused by oxidative stress.

Combined use of vitamin C and vitamin E in mouse diets can effectively scavenge free radicals, reduce oxidative stress, halt lipid peroxidation, and protect against chemical liver injury induced by carbon tetrachloride (CCl4) [13]. Vitamin E can significantly increase the activity and concentration of certain antibodies in animals. Utilizing this characteristic, supplementing vitamin E in pregnant ewes' diets did not significantly affect serum lysozyme activity [14]. The protective effects of vitamin E also manifest in alleviating intestinal damage that may occur in animals under high-altitude hypoxic environments, though the specific mechanisms remain unclear and may involve signaling pathways such as hypoxia-inducible factor (HIF), Toll-like receptor 4 (TLR4), and nuclear transcription factor- B (NF- B) [15]. Wang Jin [16] used dexamethasone (DEX) to simulate oxidative stress in broiler chickens and found that oxidative stress significantly induced DNA damage, increased DNA methylation levels, and reduced performance; dietary vitamin E supplementation improved growth performance and alleviated DNA oxidative damage but did not improve DNA methylation status.

2 Protective Effects of Vitamin E on Mycotoxin-Induced Toxic Damage in Animals

During agricultural production, improper storage of feed raw materials has led to increasingly prominent problems of excessive mycotoxin content in feed. Researchers have applied vitamin E to alleviate mycotoxin-induced toxic damage, demonstrating certain protective and ameliorative effects on inflammatory damage, blood biochemical changes, and histological alterations caused by myco-

toxin contamination.

Aflatoxin (AF), a natural toxic molecule produced by Aspergillus fungi, can cause liver cancer and gastrointestinal diseases even at low intake levels, and severe cases may lead to organ dysfunction or failure [17]. Combined application of aflatoxin B1 (AFB1) and vitamin E minimizes damage to renal cortical histological structure and significantly improves antioxidant capacity, suggesting broad prospects for vitamin E in preventing kidney damage. Khan et al. [18] found that AFB1 exposure significantly decreased body weight and egg production in White Leghorns while increasing oxidative damage to red blood cells and elevating embryonic mortality and malformation rates; combined use of vitamin E and AFB1 significantly reduced AF residues in animals but provided only partial protection against other indicators. Therefore, vitamin E supplementation in laying hen diets offers only partial protection against AFB1-induced damage. Vitamin E did not significantly affect PHA-P lymphocyte proliferation response or antibody titers against sheep red blood cells (SRBC) induced by AFB1, suggesting limited immunological protective effects, though this may be due to the single dose used in the experiment and the effects of higher doses remain uncertain [19]. Additionally, feeding AFB1-contaminated corn in agricultural production reduces growth performance and relative immune organ weight in ducks, while dietary vitamin E supplementation significantly improves these parameters and reduces mortality [20].

Ochratoxin A (OTA) is another fungal metabolite widely present in food and feed production that poses serious hazards to livestock. OTA residues significantly reduce fetal weight in pregnant rabbits and birth weight of offspring, causing significant decreases in blood biochemical parameters such as red blood cell count. Histopathological examination reveals that OTA causes fatty degeneration, hemorrhage, and congestion in liver tissue, glomerular contraction and renal tubular degeneration, and significant changes in intestinal villus length [21]. Vitamin E supplementation can partially improve tissue damage and exert therapeutic effects [21]. Additionally, adding vitamin E (100 mg/kg) significantly ameliorates the reduction in blood biochemical parameters caused by OTA exposure in broiler chickens, repairing autoimmune and hematopoietic functions, thus recommending supplementation of certain vitamin E levels in broiler diets [22]. Combined use of vitamin E with other substances also demonstrates its important value in feed applications. Abidin et al. [23] administered L-carnitine and vitamin E separately or in combination to White Leghorn cockerels exposed to OTA, proving that both improved OTA-induced changes in hematological and serum biochemical parameters and reduced OTA residues in feed. Combined use of vitamin E and silymarin significantly improved blood physiological parameters affected by OTA, including total red blood cell count (RBC) and hemoglobin concentration, while significantly reducing serum total protein (TP) content and increasing serum alanine transferase (ALT) activity. This demonstrates that combined use of vitamin E with these agents may have potential preventive and ameliorative effects against OTA-induced toxic damage [24].

Zearalenone (ZEN), a non-steroidal estrogenic mycotoxin produced by stalk rot, readily causes DNA damage in organisms. Using vero cells (monkey kidney cells), researchers analyzed ZEN' s effects on chromosome aberration and inhibition of gap junction intercellular communication through unconventional DNA synthesis, verifying its in vitro genotoxicity and epigenetic effects. Results showed that ZEN exposure significantly increased unconventional DNA synthesis in vero cells, induced chromosome aberration, and inhibited gap junction intercellular communication. Administration of vitamin E effectively restored the gap junction intercellular communication suppressed by ZEN, proving its therapeutic and reducing effects on ZEN-induced oxidative stress damage in vero cells [25].

T-2 toxin, a type A trichothecene mycotoxin produced by various Fusarium species, is commonly found in agricultural and livestock products, particularly in feed. After T-2 toxin ingestion, organisms readily develop clinical symptoms including diarrhea, vomiting, hemorrhage, and neurological diseases, while bone marrow hematopoietic function is adversely affected, causing blood disorders [26-27]. Ahmadi et al. [28] found that sublethal dose injection of T-2 toxin significantly reduced B lymphocyte (CD19+) counts in mice; combined use of vitamin E and selenium at 12 and 72 hours enhanced restoration of CD19+ counts after 24 hours, while vitamin E alone did not regulate CD19+ changes. This proves that vitamin E can promote selenium' s effects on altering B lymphocyte subsets suppressed by T-2 toxin. Current data indicate that when feed DON content reaches 4 mg/kg and T-2 toxin content reaches 3 mg/kg, genetic toxicity symptoms such as porcine lymphocyte DNA fragmentation may be induced; vitamin E supplementation can partially reduce DNA damage and help maintain lymphocyte DNA integrity [29].

3 Protective Effects of Vitamin E on Pesticide and Insecticide-Induced Toxic Damage in Animals

Beyond mycotoxins, feed hygiene and safety also include contamination and residues of pesticides and insecticides from excessive application in agricultural production, which cause damage to animals and even humans after absorption. As an additive, vitamin E exhibits protective effects against damage caused by organophosphorus pesticides and insecticides, whether used alone or in combination. Olsvik et al. [30] showed that vitamin E supplementation effectively prevented chlorpyrifos toxicity by promoting fatty acid accumulation in Atlantic salmon (Salmo salar) and resisting inhibition of glucose metabolism, thereby improving chlorpyrifos-induced toxic damage. The subacute toxicity of organophosphorus pesticide diazinon increases reactive oxygen species content and causes liver lipid peroxidation and destruction of the antioxidant defense system in mice; vitamin E supplementation restored specific enzymes in mouse liver cells to normal, proving its effectiveness in improving oxidative stress and liver tissue damage [31]. Methyl parathion' s subacute and acute toxicity primarily affects the animal liver. Uzunhisarcikli et al. [32] administered methyl

parathion to male rats via gavage for 7 weeks, resulting in significant weight loss and increased liver weight. Combined use of vitamin E and vitamin C in diets effectively improved blood biochemical parameters in rats but did not produce significant pathological changes in hepatocyte damage caused by methyl parathion, suggesting it may not fundamentally alter organ damage but rather provide preventive and functional barrier effects [32]. Cengiz et al. [33] proved that feeding 1.45 g/L deltamethrin increased total fat content and significantly increased polyunsaturated fatty acid proportions in Nile tilapia (Oreochromis niloticus L.) liver; combined deltamethrin and vitamin E administration reduced unsaturated fatty acid peroxidation, thereby protecting cell membranes.

Vitamin E plays an important role in ensuring normal reproductive function in mammals and also exhibits protective effects against reproductive dysfunction caused by some exogenous toxicants. Bayar et al. [34] confirmed that deltamethrin causes necrosis of fish spermatogenic cells, nuclear pyknosis, reduced spermatogenic cell numbers, decreased spermatocyte counts, degeneration of spermatogonia, and increased testicular macrophage numbers; vitamin E reduced some deltamethrin-induced histopathological changes but did not provide complete protection.

4 Protective Effects of Vitamin E on Other Types of Damage in Animals

Vitamin E's antioxidant function plays a certain role in the detoxification process. Dimethylformamide (DMF) poisoning readily causes liver lipid peroxidation. Jiang Chao et al. [35] found that adding different levels of vitamin E to diets significantly reduced malondialdehyde (MDA) content in DMF-poisoned mouse livers, proving vitamin E' s antagonistic effect against DMF-induced liver lipid peroxidation damage. Cadmium poisoning increases free radical production, particularly in nervous tissue, causing oxidative stress damage. Ge Guo et al. [36] used vitamin E and vitamin C separately and in combination in mouse models, finding both exerted significant protective effects on mouse substantia nigra neurons; the intervention effects ranked as vitamin E > combined vitamin E and C > vitamin C, providing experimental theoretical basis for preventing cadmium poisoning in certain regions. Sodium fluoride (NaF) inhibits digestive enzyme activity in the pancreas, readily causing histological deformation and pathological changes in NaF-related biochemical indicators and vitamins. Agha et al. [37] added vitamin E, methionine, and L-carnosine to diets and co-fed them for 35 days, which resisted NaF poisoning-induced histological, hematological, immunological, and DNA damage. Additionally, supplementation with natural vitamin E can enhance the immune response of specific Th1-type antigens to tetanus toxoid in immune organs and alleviate damage to these organs [38].

5 Summary

In summary, vitamin E plays important roles in detoxification and protection of animal organisms, primarily manifested in antioxidant effects, prevention of tissue cell damage, enhancement of animal immunity, and improvement of tissue metabolism, all of which provide effective protection and alleviate damage such as oxidative stress. Therefore, vitamin E has gained widespread recognition and application in actual agricultural and livestock production. With deepening research and expanding research fields, vitamin E can continue to deliver new utilization value beyond its traditional applications as a food additive, feed additive, and health product. However, certain practical issues remain: 1) The specific mechanisms of vitamin E as a detoxifying and protective agent in vivo remain unclear; 2) Whether vitamin E provides barrier protection before toxic damage occurs or detoxification effects after damage remains uncertain; 3) Vitamin E only partially ameliorates toxic damage rather than completely eliminating its effects. Relevant researchers continue conducting experiments to expand its broader application prospects and anticipate achieving more fruitful research results in the future.

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