Effects of Ammonia on Muscle Quality in Livestock and Poultry: Postprint

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Date: 2018-12-24T00:00:00+00:00

Abstract

Ammonia exposure in livestock housing represents one of the critical environmental factors influencing the growth and health of livestock and poultry. Such exposure not only impairs growth performance but also affects meat quality. Studies have demonstrated that ammonia can induce myostatin (MSTN) expression via the nuclear factor kappa enhancer binding protein (NF- B) signaling pathway while inhibiting the mammalian target of rapamycin (mTOR) signaling pathway, thereby suppressing skeletal muscle growth and protein synthesis. Additionally, ammonia can regulate the expression of genes associated with lipid metabolism, influencing body fat distribution. This review synthesizes current knowledge on ammonia production in livestock housing, ammonia metabolism in vivo, the impacts of ammonia exposure on growth performance and meat quality in livestock and poultry, and the potential molecular mechanisms underlying ammonia' s effects on meat quality, with the aim of providing novel evidence and perspectives for optimizing farming environmental conditions and improving meat quality.

Full Text

Effects of Ammonia on Meat Quality in Livestock and Poultry

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Abstract: Ammonia exposure in livestock housing represents a critical environmental factor affecting animal growth and health. Beyond impairing growth performance, ammonia exposure adversely influences meat quality in livestock and poultry. Research demonstrates that ammonia can induce myostatin (MSTN)

expression through the nuclear factor kappa enhancer binding protein (NF- B) signaling pathway while inhibiting the mammalian target of rapamycin (mTOR) pathway, thereby suppressing skeletal muscle growth and protein synthesis. Ammonia also regulates the expression of genes related to fat metabolism, altering body fat distribution. This review synthesizes current knowledge on ammonia production in livestock housing, ammonia metabolism in vivo, the impacts of ammonia exposure on growth performance and meat quality, and the potential molecular mechanisms underlying these effects, aiming to provide novel evidence and strategies for improving rearing environments and enhancing meat quality.

Keywords: ammonia; livestock and poultry; meat quality; NF- B signal pathway; mTOR signal pathway

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The intensification of livestock production and widespread adoption of enclosed housing systems for swine and poultry have made indoor air quality a crucial environmental determinant of animal health, production efficiency, and product quality. Ammonia, a colorless gas with a strong pungent odor, constitutes one of the primary pollutants in livestock facilities. Ammonia exposure irritates respiratory mucosa, triggering inflammatory responses and compromising barrier function. Upon absorption through alveoli into the bloodstream, ammonia binds to hemoglobin, causing tissue hypoxia and anemia. Elevated ammonia concentrations reduce feed intake, alter animal behavior, and severely impair growth performance and health [1-2]. While most research on ammonia toxicity has focused on digestive and respiratory tract injuries, hemoglobin disruption, immune dysfunction, and behavioral changes, recent studies have revealed detrimental effects on meat quality. This review examines ammonia sources in intensive production systems, its impacts on growth performance and meat quality, and the underlying molecular mechanisms, providing a theoretical foundation for healthy animal production and high-quality meat products.

1 Ammonia Production in Livestock Housing

Animal manure, urine, and feed residues represent the primary sources of ammonia in livestock facilities [3-4]. These substrates decompose at different rates: feces and feed residues contain approximately 80% organic nitrogen and require weeks for microbial conversion to ammonia, whereas urea/uric acid in urine decomposes within hours under ambient temperatures [5]. Ammonia production is influenced by multiple factors including dietary composition, ambient temperature, and excreta retention time [6-8]. Higher dietary crude protein levels increase nitrogen content in manure and urine, while prolonged retention times and temperatures approaching the optimum for microbial enzymatic activity enhance ammonia accumulation. Consequently, increasing ventilation rates and maintaining housing cleanliness effectively reduce ammonia concentrations [7].

2 Ammonia Metabolic Pathways in vivo

Environmental ammonia enters the bloodstream through respiratory gas exchange in alveoli, existing primarily as ammonium ions (NH4+) with a smaller fraction as free ammonia. The NH4+/ammonia ratio in blood depends on pH [9]. Endogenous metabolic processes, including amino acid deamination and catabolism of amines, purines, and pyrimidines, also generate ammonia [10], as does microbial activity in the digestive tract. Under normal physiological conditions, animals detoxify ammonia through two primary pathways: conversion to non-toxic alanine via reaction with pyruvate, and formation of glutamine from -ketoglutarate catalyzed by glutamate dehydrogenase [9]. Glutamine serves as both an essential amino acid component and amino group donor in biosynthetic pathways, and as an important energy substrate [11]. Given its large mass, skeletal muscle represents a critical tissue for ammonia detoxification via glutamine synthesis. Alanine and glutamine are transported to the liver and kidneys for urea/uric acid synthesis through the urea cycle, with final excretion in urine as urea/uric acid or ammonium salts [10].

3 Effects of Ammonia on Growth Performance

Ammonia exposure severely compromises growth performance, primarily by reducing feed intake, feed conversion efficiency, and body weight gain while increasing mortality. Reece et al. [12] reported decreased feed conversion and slaughter weight in broilers exposed to 25–200 mg/kg ammonia, with mortality increasing above 100 mg/kg. Li et al. [13] exposed 21-day-old broilers to 25, 50, and 75 mg/kg ammonia for 21 days, observing significant reductions in average daily feed intake (4.01%, 6.68%, and 9.56%) and average daily gain (8.25%, 11.83%, and 15.42%). Cheng et al. [14] found that growing pigs exposed to 20 mg/kg ammonia for 15 weeks showed no significant effects on growth rate or health, whereas Cao et al. [15] demonstrated that piglet average daily gain decreased with increasing ammonia concentration, dropping significantly by 22.07% at 80 mg/kg. Drummond et al. [16] exposed 4-week-old pigs to 50, 100, and 150 mg/kg ammonia for 4 weeks, resulting in significant weight reductions of 12%, 30%, and 29%, respectively. These findings indicate concentration-dependent effects of ammonia on feed intake and weight gain, with high concentrations exerting particularly severe impacts. High ammonia levels irritate eyes, nose, and mouth, disrupting feeding behavior and reducing intake [17]. Additionally, ammonia acts as a stressor, redirecting nutrients toward immune and defense tissues [18], thereby reducing energy deposition in muscle and impairing feed conversion efficiency.

4 Effects of Ammonia on Muscle Quality

Ammonia exposure reduces feed intake, decreasing protein and energy consumption, which influences the proportion of oxidative muscle fibers and intramuscular fat content, thereby affecting meat quality [19-20]. High ammonia con-

centrations also induce stress responses that reduce energy deposition in muscle [18] and alter muscle glycogen content. When glycogen depletion reaches a critical threshold, it causes pH changes post-mortem, resulting in pale color and reduced shelf life [21]. Pre-slaughter stress (including heat stress and electrical stunning) can cause acidic meat, decreased water-holding capacity, and pale, soft, exudative (PSE) meat [23-24]. Most research on ammonia' s effects on meat quality has used broiler models, with limited studies on swine and other species. In the 1970s, Quarles et al. [25] observed blisters in broiler breast muscle and reduced carcass grades at 25–50 mg/kg ammonia exposure. Sackett et al. [26] reported that 25 mg/kg ammonia tended to decrease breast muscle pH and increase drip loss, while higher concentrations (75–100 mg/kg) significantly reduced tenderness. Zhou [27] found that 20 mg/kg ammonia exposure for 2 weeks significantly reduced breast muscle pH at 45 minutes post-slaughter, while 50 mg/kg increased meat lightness (L* value). Wei et al. [28] demonstrated that 70 mg/kg ammonia significantly increased drip loss, decreased tenderness and redness (a* value) at 45 minutes post-slaughter, and increased L* and yellowness (b* values) at both 45 minutes and 24 hours post-slaughter. Our previous research showed that ammonia exposure significantly increased drip loss in broiler breast muscle without affecting meat color [13], and altered body fat distribution by reducing intramuscular fat content [29]. Collectively, these studies indicate that ammonia exposure compromises broiler meat quality by reducing quality grades, muscle pH, tenderness, and color intensity while increasing drip loss.

5 Potential Molecular Mechanisms of Ammonia-Induced Effects on Meat Quality

High environmental ammonia enters the body primarily through the respiratory system and secondarily via the digestive tract, elevating blood ammonia concentrations and altering signaling pathways and gene expression. These changes affect energy metabolism, muscle cell growth, intramuscular fat deposition, and muscle fiber type transformation, ultimately influencing muscle yield and meat quality.

5.1 Ammonia Induces Myostatin Expression to Inhibit Muscle Cell Growth

High ammonia concentrations in housing enter the bloodstream through respiration and alveolar exchange, causing transient elevation of blood ammonia. When concentrations exceed the metabolic threshold, blood ammonia stabilizes at elevated levels. High blood ammonia significantly upregulates myostatin (MSTN) expression in skeletal muscle [9,30]. MSTN acts as a potent inhibitor of protein synthesis, suppressing mammalian target of rapamycin (mTOR) activation through Akt-dependent and -independent mechanisms, thereby impairing skeletal muscle protein synthesis [31] and reducing meat yield. MSTN also regulates muscle fiber type transformation by activating Smad2 and Smad3 downstream of the transforming growth factor- (TGF-)/Smad3 pathway, modulating fork-

head box O (FoxO) and atrogin-1 to reduce myosin heavy chain expression [32], induce muscle atrophy [33], and promote conversion from type I to type II fibers [34]. Muscle fiber type is closely associated with meat quality traits including color, water-holding capacity, pH, tenderness, and intramuscular fat deposition. Oxidative fibers (types I and IIa) contain higher myoglobin and lipid content, producing darker meat with better moisture retention, whereas glycolytic fibers (type IIb) have high glycogen content, accelerating post-mortem pH decline through anaerobic glycolysis and lactate production [36], compromising meat preservation.

Qiu et al. [30] demonstrated that hyperammonemia activates the nuclear factor kappa enhancer binding protein (NF- B) signaling pathway to regulate MSTN expression (Figure 1 [Figure 1: see original paper]). High ammonia activates inhibitor of NF- B kinase / (IKK /) and downstream signaling molecules, releasing the p50-p65 heterodimer for nuclear translocation and binding to cis-acting elements on the MSTN promoter, thereby regulating transcription [37]. Current research on ammonia-induced MSTN expression via NF- B activation has primarily utilized in vitro cell models; however, the ammonia sensor in cells remains unidentified, the molecular mechanisms of IKK / activation are unclear, and relevant animal studies are scarce.

5.2 Ammonia Affects Muscle Cell Energy Metabolism

When blood ammonia remains elevated, the liver cannot efficiently convert ammonia to urea or uric acid. As ammonia freely diffuses across cell membranes, it serves as an important gaseous signaling molecule linking liver and muscle. Excess ammonia enters muscle tissue, combining with -ketoglutarate to form glutamate and glutamine, thereby reducing blood ammonia [38]. -Ketoglutarate is a crucial metabolite connecting carbon-nitrogen cycles and a key intermediate in the tricarboxylic acid cycle; its depletion reduces adenosine triphosphate (ATP) generation, impairing the energy-intensive process of protein synthesis [9] and decreasing muscle fiber number. This mechanism parallels sarcopenia and muscle dysfunction in cirrhotic patients with impaired hepatic function and elevated blood ammonia. Research indicates that leucine serves as a substrate for -ketoglutarate replenishment [39], suggesting that leucine supplementation may alleviate ammonia-induced muscle damage and mitigate adverse effects on meat quality.

Additionally, reduced ATP content increases the AMP/ATP ratio, activating AMP-activated protein kinase (AMPK) [40]. AMPK functions as a cellular energy sensor that critically regulates glucose and lipid metabolism [34]. AMPK activation enhances autophagy, clearing post-translationally modified muscle proteins and potentially disrupting actomyosin interactions, leading to muscle dysfunction [9]. Elevated AMPK activity also induces peroxisome proliferator-activated receptor co-activator 1 (PGC-1) expression, a transcriptional co-activator whose expression level in skeletal muscle is closely associated with muscle fiber type [34]. Thus, AMPK can influence meat quality by modulat-

ing PGC-1 expression and muscle fiber type composition. Studies show that AMPK overexpression in mouse skeletal muscle promotes conversion from type IIb to type IIa or IIx fibers [41].

5.3 Ammonia Affects Intramuscular Fat Deposition

Our previous research demonstrated that ammonia exposure altered body fat distribution in broilers, reducing intramuscular fat deposition in breast muscle while increasing abdominal fat [29]. Transcriptomic analysis revealed that ammonia regulates expression of genes involved in lipid metabolism [42-43] (Figure 2 [Figure 2: see original paper]). Cluster of differentiation 36 (CD36) is a fatty acid receptor on plasma and mitochondrial membranes that participates in fatty acid uptake, transport, and oxidation [44]. CD36 is highly expressed in skeletal muscle, which relies on fatty acid oxidation for energy [45]; CD36 knockout impairs fatty acid transport and oxidation, reducing muscle fat content [46]. Solute carrier family 27 member 1 (SLC27A1) is another key gene involved in fatty acid transport and metabolism, primarily expressed in muscle and adipose tissue. Wu et al. [47] found that SLC27A1 inactivation protected mice from diet-induced obesity. Long-chain-fatty-acid CoA ligase 1 (ACSL1) is associated with fat deposition capacity and meat quality; Joseph et al. [48] showed that ACSL1 participates in unsaturated fatty acid biosynthesis and fatty acid metabolism. Ankyrin repeats and suppressor of cytokine signaling box 2 (ASB2) is an important gene involved in accelerated post-mortem glycolysis; its product promotes myofibrillar protein degradation, and high expression correlates with increased tenderness [28]. Perilipin 2 (PLIN2) participates in intramuscular fat storage and mobilization; Conte et al. [49] found that high PLIN2 expression reduced muscle strength, while porcine studies showed that increased PLIN2 expression was associated with elevated intramuscular fat content [50]. These findings demonstrate that ammonia exposure can modulate tissue lipid metabolism at the transcriptional level, redirecting fat deposition and reducing muscle fat content, thereby compromising meat quality.

6 Conclusion

Ammonia concentration in livestock production environments directly affects the health of both workers and animals. Excessive ammonia exposure not only reduces feed intake and feed conversion efficiency, severely impairing production performance, but also significantly degrades muscle quality. Current research on ammonia's effects on meat quality has primarily utilized broiler models, with limited studies on other species. Systematic investigations are lacking on different animal breeds, ages, physiological stages, and cumulative effects of exposure duration. Most published studies have focused on phenotypic meat quality indicators, with insufficient exploration of underlying molecular mechanisms. Further in-depth research elucidating the mechanisms by which ammonia exposure affects muscle growth will provide new insights into skeletal muscle growth regulation and offer pathways for multi-dimensional approaches to improve meat

quality through integrated consideration of breed, nutrition, and environmental factors.

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