Ammonia Emission Patterns from Livestock Housing and Their Hazards to Animal Health: Postprint

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Date: 2017-11-08T00:00:00+00:00

Abstract

With the continuous expansion of livestock and poultry farming scale and the rapid development of intensive production systems, large quantities of animal excreta are being discharged in a concentrated manner. Volatilized ammonia (NH3) not only causes substantial environmental pollution, but also severely compromises animal health, induces various diseases, and results in decreased production performance. Therefore, analyzing the patterns of ammonia emissions from livestock and poultry and their impacts on animal production and health is of great significance for controlling ammonia concentrations in livestock housing. This paper primarily expounds on the factors influencing ammonia emissions in livestock facilities and the emission patterns, analyzes the effects of ammonia on livestock and poultry health and the associated pathological mechanisms, and provides a reference for large-scale livestock and poultry production.

Full Text

Ammonia Emission Characteristics from Livestock and Poultry Houses and Their Harm to Animal Health

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Abstract

With the continuous expansion of livestock production and rapid development of intensive farming systems, large quantities of manure are being discharged in concentrated manner. Volatile ammonia (NH_3) not only causes substantial environmental pollution but also seriously compromises animal health, induces various diseases, and reduces production performance. Therefore, analyzing ammonia emission patterns and their impacts on livestock production and health is crucial for controlling ammonia concentrations in animal housing. This paper systematically reviews the factors influencing ammonia emissions from livestock facilities and their emission characteristics, analyzes the effects of ammonia on animal health and the underlying damage mechanisms, and provides a reference for large-scale livestock production.

Keywords: livestock and poultry; ammonia; emission characteristics; damage mechanism

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Ammonia nitrogen emissions have become a critical constraint on the sustainable development of livestock production in China. In pig production, for example, animals can only utilize 30%–55% of dietary nitrogen, with the majority excreted in feces and urine. Urinary nitrogen exists primarily as urea (97%), and fattening pigs emit approximately 70 g of ammonia per day [1-2]. China has been the world' s largest ammonia emitter for nearly two decades, with over 80% of emissions originating from livestock production and fertilizer application, according to the *China Air Quality Management Assessment Report 2016*. Total annual ammonia emissions in China reach approximately 10.2 million tons—exceeding the combined emissions of the United States and the European Union —and continue to rise with expanding production scales [6].

Ammonia in livestock housing is generated through two primary pathways: metabolic decomposition of protein after ingestion, and decomposition of urinary nitrogen. Most ammonia originates from urea breakdown in excreta. Poultry lack arginase and carbamoyl phosphate synthetase in their livers, preventing urea synthesis through the hepatic ornithine cycle. Instead, they synthesize purines in the liver and kidneys, which are converted to uric acid via xanthine oxidase. Due to their short digestive tracts, many nutrients are not fully utilized and are excreted in feces. Poultry manure contains up to 70% nitrogen, with uric acid and urea readily decomposed by urease into ammonia [8]. Consequently, ammonia concentrations and emissions are typically higher in poultry houses than in livestock barns [9].

2.1 Animal Growth Stage

As animals progress through growth stages, increasing body weight corresponds to higher feed intake and protein consumption, leading to greater production of uric acid and urea and, consequently, increased ammonia emissions. Hayes

et al. [10] monitored ammonia emissions across growth stages from suckling piglets to finishing pigs, finding average emissions of 11.3–11.9 g/d per pig in finishing barns versus only 1.1–1.7 g/d in nursery facilities. In broilers, ammonia emissions show a strong linear relationship with age and body weight, with these two factors being the most important determinants [11]. Investigations of broiler house ammonia concentrations and per-animal emissions revealed a 0.92 g difference between 1- and 23-day-old birds [12]. Variations in ammonia emissions across growth stages result not only from changes in body weight and feed intake but also from differences in dietary nutrient composition and housing structure.

2.2 Housing Structure

Research monitoring environmental parameters in three types of chicken houses found that ammonia concentrations in extra-large enclosed houses were approximately double those in conventional enclosed or open-sided houses, likely due to reduced ventilation rates along the long axis preventing effective removal of harmful gases [13]. Ventilation rates directly affect ammonia emissions; increased air velocity accelerates urea decomposition and raises ammonia emissions [14]. One study reported that increasing ventilation rate fivefold doubled NH_3 emission rates by enhancing surface gas exchange at the manure-air interface [15]. Notably, while increased air velocity accelerates urea decomposition and ammonia emission, the physical effect of air movement reduces in-house ammonia concentrations, resulting in significantly lower ambient levels despite higher emission rates [2].

Floor material significantly affects ammonia emissions in pig barns. Replacing concrete slats with metal or plastic slatted floors reduces emissions by 10%–40% [16]. Concrete surfaces are rougher than metal or plastic, increasing the area for manure adhesion and urea decomposition, and adhered manure is more difficult to clean from concrete than from smoother plastic surfaces, leading to greater residual manure in slatted areas and increased emissions. Slatted floor area also influences emissions: replacing fully slatted floors with partially slatted floors (37% slat area) reduced ammonia emissions by approximately 40%, and reducing slat area from 50% to 25% decreased daily ammonia emissions per finishing pig from 6.4 g to 5.7 g [17]. Larger slatted areas increase contact between manure and slats, and expanding slat area widens the manure pit beneath, increasing the surface area for urea reaction and ammonia emission.

Temperature affects ammonia emissions primarily by influencing urease activity in manure. Research shows that urease activity increases with temperature, remaining relatively high even below 90°C. Consequently, elevated temperatures in animal housing enhance urease activity in excreta, accelerating urea decomposition and ammonia emissions [18-19]. In pig barns, high-temperature periods (13:00–17:00) account for 33% of daily ammonia emissions [20]. During composting, internal temperatures exceeding 60°C at 14–28 days correspond to peak ammonia emissions [21]. Therefore, adequate ventilation is essential in

summer to reduce ammonia and other harmful gas concentrations.

2.4 Dietary Nutrient Composition

Appropriately reducing dietary crude protein levels while supplementing essential amino acids can improve animal growth performance while significantly decreasing ammonia emissions from excreta (Table 1 [22-31]). Studies demonstrate that reducing dietary crude protein by 1% in pig diets decreases ammonia emissions by 10%–13%, and reducing crude protein from 20% to 12% cuts barn emissions by 63% [32-33]. Lower dietary crude protein reduces urea and uric acid content in excreta and decreases blood urea nitrogen, which reduces urinary nitrogen production through circulation and subsequently lowers ammonia emissions [24,29].

Dietary fiber levels also influence ammonia emissions. Fermentation of dietary fiber in the hindgut promotes healthy gut microbiota, with some microbes producing short-chain fatty acids (propionate, butyrate) that lower cecal and fecal pH, inhibiting urease activity and reducing NH_3 emissions [34]. Additionally, beneficial bacterial growth enhances microbial protein utilization, reducing protein excretion and nitrogen waste [2]. Adding corn distillers grains to laying hen diets increased crude fiber by 2.47% and reduced total ammonia emissions by 48% over one week [35].

2.5 Feed Additives

Feed additives reduce ammonia emissions primarily by inhibiting urease activity, physical adsorption, and improving protein utilization. Plant extracts such as Yucca schidigera inhibit urease activity in manure, possibly through saponins that both suppress urease and chemically bind ammonia. One study reported that adding 125 mg/kg Yucca extract to lactating sow diets significantly reduced barn ammonia concentrations [36]. Physical adsorption methods involve adding natural or synthetic materials (activated carbon, clinoptilolite, modified alumina) with porous structures that effectively adsorb small gas molecules. Adding 5% zeolite to broiler diets (22–42 days) reduced ammonia emissions by approximately 50% [37]. Probiotics improve gut microbiota, increase beneficial bacteria, enhance bacterial protein utilization, and reduce fecal nitrogen excretion. Adding 0.05% lactic acid bacteria and yeast to growing pig diets reduced ammonia concentrations by 4.02–6.00 mg/L [36].

2.6 Manure Removal Methods and Frequency

As manure is the primary source of ammonia emissions, cleaning methods significantly affect in-house ammonia concentrations. Studies show that scraper systems increase ammonia concentrations by 9.97% compared to manual dry removal [38]. In sheep barns, manual removal and scraping twice daily reduced ammonia emissions by 52% and 63%, respectively, compared to deep-pit accumulation [40].

Understanding ammonia emission patterns and influencing factors is crucial for controlling barn concentrations. Emissions can be reduced by addressing both sources and sinks: nutritional measures to decrease fecal and urinary nitrogen excretion, and inhibition of urea decomposition through feed additives like urease inhibitors or Yucca extract and reduction of manure pH. Adsorbents in feed and manure can remove ammonia, while good ventilation, improved cleaning practices, and frequent manure removal effectively reduce concentrations. With increasing emphasis on environmental health in animal production, nutritional strategies for ammonia reduction will find broader application.

3.1 Animal Welfare

Chronic ammonia exposure induces abnormal behaviors in livestock and poultry. Animals instinctively avoid ammonia; in a 10-day exposure trial with piglets, two-thirds avoided environments with 100 mg/kg ammonia, preferring normal air (5–10 mg/kg) [41]. Respiratory chamber studies on broilers showed that increasing ammonia concentrations exacerbated hock joint and foot pad infections, lameness, and walking instability [42]. Ammonia causes ocular abnormalities, with broilers in high-concentration environments exhibiting wing-assisted eye rubbing [43]. In nursery pigs exposed to 50 mg/kg ammonia, blood macrophage and lymphocyte counts and corticosterone levels increased significantly, indicating a respiratory stress immune response [44]. At 70 mg/kg ammonia, broilers showed reduced serum globulin and lysozyme activity; lysozyme, secreted primarily by macrophages, is a key component of non-specific immunity [45]. Thus, ammonia impairs immune function, allowing pathogen invasion and respiratory disease, ultimately reducing production performance (Table 2 [42,44,46-55]).

3.2 Respiratory System

Ammonia most directly damages the trachea, ocular mucosa, and lung tissue. The severity of respiratory damage depends on both concentration and exposure duration. In broilers, 20 mg/m^3 ammonia for six weeks caused pulmonary edema, reduced feed intake, decreased growth performance, and increased disease susceptibility. At 70 mg/kg, broilers showed tracheal and pulmonary mucosal cilia loss with significantly increased pulmonary inflammatory cells [56]. High ammonia environments (75 mg/kg) caused broiler tracheal cilia shortening or loss, with upregulated mucin expression; excessive mucin secretion leads to tracheal obstruction, explaining coughing and gasping symptoms. Additionally, myosin and troponin expression increased in tracheal tissue; these proteins facilitate muscle contraction by promoting filament sliding, constricting airways to reduce ammonia inhalation [54].

Ammonia not only directly harms the respiratory tract but also increases airborne microbial aerosol concentrations and pathogen loads. Michiels et al. [52] investigated ammonia effects on PM2.5 levels and lung lesions in growing pigs, finding that increasing ammonia concentrations significantly elevated mortality and Mycoplasma pneumonia prevalence. At 15 mg/kg ammonia, pigs were sus-

ceptible to respiratory disease, while 35 mg/kg induced atrophic rhinitis [48]. Hamilton et al. [46] studied ammonia effects on suckling piglets at concentrations of 5, 10, 15, 25, 35, and 50 mg/kg, finding that 10 mg/kg caused the highest incidence of atrophic rhinitis.

3.3 Digestive System

As a stressor, ammonia damages intestinal mucosa and affects digestive enzyme activity and nutrient transporters in mucosal epithelium, impairing nutrient digestibility. In rats injected with acetamide (high blood ammonia), short-chain fatty acid oxidation in the colon was inhibited [57]. In broilers exposed to 75 mg/kg ammonia, small intestinal cytoskeletal protein expression decreased and mucosal epithelial morphology changed, manifested as shortened or absent villi, deeper crypts, and reduced growth performance. High ammonia upregulated proteins related to oxidative phosphorylation and apoptosis in intestinal mucosa, triggering oxidative stress and interfering with immune function and nutrient absorption [53]. At 70 mg/kg ammonia, duodenal, jejunal, and cecal pH increased significantly, with effects on intestinal development worsening over time. pH is a critical indicator of gut health; acidic conditions favor beneficial bacteria (Lactobacillus, Bifidobacterium) while inhibiting pathogens (E. coli, Salmonella). Major pathogenic bacteria thrive at pH 6.5–8.0, whereas beneficial bacteria prefer acidic conditions. Therefore, high ammonia disrupts microbial balance and promotes putrefactive bacteria proliferation (Figure 1 [Figure 1: see original paper][7,44,53-54,56,58]).

3.4 Liver Tissue

Ammonia serves as a substrate for amino acid synthesis and is produced via amino acid deamination. As a product of amino acid and protein metabolism, ammonia is primarily metabolized to urea in the liver. Our previous research found that as portal blood ammonium (NH_4^+) concentration increases, more NH_4^+ enters the liver, accelerating hepatic amino acid catabolism and increasing urinary nitrogen excretion because urea synthesis nitrogen is derived from blood ammonia and amino acid amino groups [59]. Additionally, urea synthesis from ammonia, CO_2, and water consumes approximately 45% of total hepatic energy expenditure [60]; excessive ammonia inhalation disrupts whole-body energy metabolism. High blood ammonia overloads the liver, causing fatigue, failure, and hypertrophy [61]. Ammonia also impairs hepatocyte antioxidant capacity, increasing reactive oxygen species (ROS) concentrations [62]. In broilers, high ammonia environments cause hepatic metabolic disorders, reduced antioxidant capacity, impaired hepatocyte regeneration, and potential cirrhosis [63].

3.5 Nervous System

Excessive ammonia enters the bloodstream and, when not fully converted to NH_4^+, crosses into brain tissue as ammonia gas. Ammonia toxicity severely damages neurons, microglia, and astrocytes, particularly causing astrocyte swelling

[64]. The primary ammonia detoxification pathway in brain tissue involves formation of glutamate and glutamine. Limited brain capacity to convert ammonia to glutamine leads to accumulation of both, causing cerebral dysfunction including increased brain water content and abnormal ion transport and neurotransmitter function [65]. Brain hypoxia or inhibition of cellular respiration from hyperammonemia increases brain lactate [66]. Cattle fed excess urea exhibit muscle tremors, ruminal stasis, tachycardia, dehydration, and convulsions [67].

Ammonia inhibits α-ketoglutarate dehydrogenase and pyruvate dehydrogenase in the tricarboxylic acid (TCA) cycle, reducing NADH and ATP generation in astrocyte mitochondria [58]. Treatment of rat astrocytes with 5 mmol/L ammonium chloride (NH_4Cl) severely impaired cellular energy metabolism and phosphorylation, causing ATP production to plummet [68]. Ammonia increases mitochondrial permeability transition pore (mPTP) opening, enhancing permeability, causing mitochondrial matrix swelling, incomplete oxidative phosphorylation, and blocked ATP synthesis [69-70]. Ammonia exposure in cultured astrocytes generates reactive nitrogen species, causing oxidative/nitrosative stress (ONS) associated with astrocyte swelling. This involves increased nitric oxide synthesis, partial coupling to N-methyl-D-aspartate receptor activation, and increased ROS production via NADPH oxidase. Enhanced ONS and astrocyte swelling increase glutamine synthesis, which accumulates in mitochondria and, upon degradation, impairs mitochondrial function [71].

[Figure 1: see original paper]

Ammonia hazards include direct respiratory effects from inhaled ammonia and systemic effects from elevated blood ammonia on the liver, nervous system, digestive system, and cellular metabolism. Most studies show that increasing ammonia concentrations reduce growth performance and exacerbate respiratory damage. However, results are not entirely consistent, possibly due to differences in exposure duration and ammonia tolerance across species and growth stages. With growing emphasis on animal welfare, ammonia thresholds are continually lowered beyond impacts on performance alone. This review of ammonia emission patterns and health effects provides a foundation for controlling disease and improving production environments.

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