Atmospheric Ammonia Is Detrimental to the Performance of Modern Commercial Broilers

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ABSTRACT Atmospheric ammonia inhibits broiler performance. Quantified effects are based on older genetic stock with a BW of 2,000 g at 7 wk. In contrast, modern genetic stock reaches 3,200 g at 7 wk of age. To assess the impact on present day broilers, 2 trials were conducted exposing male broilers to graded levels (0, 25, 50, and 75 ppm) of aerial ammonia from 0 to 4 wk of age. Sixty, 1-d-old chicks were placed in environmentally controlled chambers, weighed weekly as a group, and processed with yield determined at 7 wk of age. Final BW was significantly depressed by 6 and 9% for the 50 and 75 ppm concentrations of ammonia as compared with 0 ppm. Also, mortality was significantly greater at the 75 ppm ammonia concentration, 13.9% compared with 5.8% for the 0 ppm treatment. Percentage yield of deboned meat per bird decreased slightly with increasing exposure to ammonia but was not statistically significant. Although current genetic stock reaches growout weights that are approximately 60% greater than those 2 decades ago, the relative quantified effects of ammonia exposure were similar. Additionally, statistical analysis of the results provided a simple equation, presented herein, for predicting the decline in BW of male broilers after exposure to ammonia.

(Key words: ammonia, broiler, body weight, performance, yield)

INTRODUCTION Ammonia concentration in a commercial broiler house commonly exceeds 50 ppm, especially during the brooding phase and when the same litter has been used for several flocks. New research has shown that house-to-house variability is high, even on a single broiler farm (Wheeler et al., 2003). Recommended ammonia levels in broiler houses are 25 to 50 ppm, the lower of which is set by US regulatory agencies, NIOSH and OSHA, for the 8-h human exposure limit.

Ammonia levels in broiler houses can reduce bird performance (Charles and Payne, 1966a,b; Reece et al., 1981; Moore et al., 1999) and increase susceptibility to disease and increase subsequent mortality (Kristensen and Wathes, 2000; Elliot and Collins, 1982). House management, season, humidity, stocking density, and litter properties influence ammonia concentrations. Although it is widely known that ammonia is detrimental to poultry welfare, the effect of ammonia on broiler performance is of practical importance to the poultry industry for determining production losses.

Today’s broiler can attain a BW of 3,200 g at 7 wk compared with those 2 decades ago at 2,000 g. Literature providing quantified effects of ammonia is based on the older genetic stock. Reece et al. (1981) exposed broiler chicks to atmospheric ammonia during a 0-to-4-wk period and observed reductions in BW up to 8% at 7 wk of age. Ammonia levels are highest during the first few weeks of growth, but as the birds grow larger, ventilation rates are increased, and ammonia levels in the house decrease. Increased ventilation reduces contaminant load inside the house but compromises energy efficiency (Wathes et al., 1997). Quantifying the effects of ammonia on today’s genetic stock of broiler allows industry personnel to realize the need for ammonia reduction and to promote strategic management practices for ammonia control. The control of ammonia levels will subsequently improve bird performance and health. The objective of this project was to determine the BW depression of broilers exposed to atmospheric ammonia as well as to assess any negative effects on livability and yield.

MATERIALS AND METHODS Two trials were conducted in which 60 male commercial broilers (508 Ross × Ross)2 at 1 d of age were placed in environmentally controlled chambers (trial 1: 5 cham-
bers, trial 2: 8 chambers), as described by Reece and Deaton (1969). The commercial hatchery provided routine vaccination of the chicks prior to pick up. At the research farm, chicks received a bursal booster via water at 14 d of age. Birds had ad libitum consumption of water and feed under continuous lighting. Starter feed crumbles were provided from 0 to 3 wk and pellets were provided for the 3-to-7-wk period. The starter diet contained 21.6% protein and had an ME level of 3,150 kcal/kg, whereas the finisher diet contained 19.8% protein with an ME of 3,200 kcal/kg, on a calculated basis. Lighting for the first week was approximately 71.8 lx and then 9.7 lx for the remainder of the growout.

Ventilation in each 14.5 m³ chamber was approximately 5,100 L/min. Chamber dry bulb temperatures and dew points at 1 to 4 d were 32 and 18°C, respectively. Temperatures were reduced 2°C per week through 28 d of age to reach a growout temperature of 21°C. Before bird placement, fresh kiln-dried pine shavings were put on the floor of each chamber to a depth of 10 cm. Placement of fresh shavings, although common for research experiments, is in contrast to US commercial broiler production, which reuses bedding material. Where houses are poorly ventilated, ammonia concentrations tend to rise over built up litter (Reece et al., 1979). For the purposes of this work, ammonia was introduced into the chambers as described below.

Anhydrous ammonia was metered continuously into 6 of the chambers to maintain 24-h levels of 25, 50, or 75 ppm ammonia. No ammonia was added to the remaining chambers to maintain near 0 ppm (control). Wheeler et al. (2003) measured average ammonia concentrations in a 48-h period over reused litter in a Pennsylvania commercial house that ranged from 85 to 129 ppm; diurnal variations in ammonia concentration for this house were as much as 20 ppm above or below the average determined concentration.

Broilers in each chamber were weighed weekly as a group. Mortality was recorded daily, and feed conversion was adjusted accordingly. Ammonia level was determined daily prior to disturbing the chamber atmosphere for animal care. Gastec® detector tubes (no. 3L and 3La) in conjunction with a Sensidyne/Gastec® pump (kit 800) were used to measure aerial ammonia concentrations. Glass tube, panel mount flow meters were used to control ammonia flow into the chambers.

Atmospheric addition of ammonia was discontinued at 28 d of age, and birds were moved to an environmentally controlled pen facility where a common atmosphere with virtually no ammonia was shared. At d 49, feed was withdrawn from the broilers for 8 h prior to and in preparation for mechanical processing. Birds were transported 3.2 km via coops to a pilot-scale processing facility. All birds were processed from each treatment replication. In each replicate pen, a minimum of 47 birds and a maximum of 60 birds were processed. Processing included stunning, scalding, and feather and visceral tissue removal. After obtaining a hot weight, the lower halves (leg portion) of the carcasses were removed, and the upper halves (breast, wings, and back) were placed in an ice bath overnight. Using an automated cone line, experienced personnel deboned the chilled upper halves to obtain breast meat muscles (pectoralis major and minor). Live weight was used in conjunction with hot carcass weight to determine overall yield (wog) and with breast tissue weight to determine breast meat yield.

Analysis of variance was performed for a split plot design in which the main unit had a randomized complete block design with 2 trials, 4 ammonia treatments, and additional replications within trials for chambers. The subunit is a repeated measure for weeks. The mixed procedure model of SAS software (SAS Institute, 2000) was used, and treatment means for BW, feed:gain (F:G) and yield were declared different based on least significant difference at $P \leq 0.05$. Chi-squared analysis was used on the mortality data.

RESULTS AND DISCUSSION

Comparisons of male broiler BW are given in Table 1 for near 0 (control), 25, 50, and 75 ppm aerial ammonia concentrations. Broilers in the control atmosphere had a mean weight of 1,421 g at 4 wk of age. Compared with the control group at 4 wk, the treatment with 25 ppm demonstrated a 2% BW deficit, which was not statistically supported. The 50 and 75 ppm ammonia treatments at 4 wk were comparable, in that each resulted in 17 and 21% respective reductions in BW when compared with controls. The control treatment BW mean at 7 wk was 3,211 g. At 7 wk, the 25 ppm treatment did not differ significantly from the control group, whereas the 50 and 75 ppm levels were depressed by 6 and 9%, respectively, compared with the 0 ppm treatment. Thus, there was a rebound in BW after ammonia treatment was discontinued at 4 wk of age. Reece et al. (1981) noted a similar recovery in BW once ammonia was removed so that at 7 wk male broilers had a final BW of 2,012 g.

Both the current work and that of Reece et al. (1981) agree with earlier work (Charles and Payne, 1966a) reporting reduced growth rate for broilers as well as delayed maturity in pullets after exposure to atmospheric ammonia. In other research, Charles and Payne (1966b) did not observe a rebound in BW where laying hens were subjected to a prolonged exposure (10 wk). Further, they noted that voluntary food intake of the hens was reduced in ammoniated atmospheres (approximately 100 ppm). For pullets exposed to 200 ppm ammonia, feed intake, growth rate, and egg production were less and mortality was greater compared with control (0 ppm) birds (Deaton et al., 1984). The severity of damage to the bird is apparently greater when birds are subjected to exposures that are prolonged and involve high concentrations of ammonia.
Body weight is plotted versus ammonia concentration in Figure 1, where lines represent trends for rate of BW decline, and means are denoted by a symbol. Weeks 0 and 1 lacked the trend depiction, as chicks experienced no apparent change in BW due to ammonia exposure. In the current study, aerial ammonia concentration and bird age separately ($P < 0.0001$) affected BW. The rate of BW reduction was greatest at wk 3 and 4 and was linear for the conditions at those times. There was an initial decline in BW at 2 wk, but the detriment was strongest at 3 and 4 wk and was less discernable at wk 5 to 7.

The trends in Figure 1 are a product of the following exponential equation:

$$ BW = \text{intercept}_{(\text{week})} \times \text{rate}^{(\text{ammonia concentration})} $$

Table 2 provides numeric values for weekly intercept and rate of BW decline for the 0 to 7 wk growout with ammonia concentrations to 75 ppm. Using these values and the above equation, one can easily calculate the detrimental effect of low levels of aerial ammonia on BW.

In 2002, approximately 8.59 billion broilers were produced in the US by over 50 major broiler integrators (National Agricultural Statistics Service, USDA, 2003). The results of this study demonstrated that lowering ammonia exposure improved BW. Thus, we can assert that managing the ammonia at or below 25 ppm resulted in a more consistent and uniform-sized product. Lower
variability in BW for mechanically processed broilers assists poultry companies in meeting emerging standards for well-being as promoted by consumer groups because mechanical equipment can “treat” birds of similar size more reliably.

Controlling ammonia concentrations in 10% of the integrators and growers would translate into an exceptional production increase. For example, assuming optimum ammonia management can increase the BW of the average broiler by 45 g (0.1 lb), profits (using the 2002 farm value of $0.305/lb, National Agricultural Statistics Service, USDA, 2003) would be increased $26.2 million. This can be calculated as follows:

\[(8.59 \text{ billion broilers}) \times (0.1 \text{ integrators and growers}) \times (0.1 \text{ lb}) \times (0.305/\text{lb}) = 26.2 \text{ million}\]

A 45-g increase seems reasonable, as we observed a 9% (290 g) depression for the 75 ppm treatment, more than 6 times the assumption used in the above example. Growers who use optimal management practices can increase their profits and produce a healthier product.

Yield and F:G were also numerically poorer at the increased levels of ammonia concentration, but there were no noted statistically significant effects for these parameters (Table 1). Feed conversion ratios generally increased with the increase in ammonia exposure, and at 7 wk of age, F:G ranged from 1.91 to 1.98. The 0 ppm ammonia treatment averaged 73.2% in overall yield, whereas the 75 ppm treatment had a 72.4% total yield. Similarly, the control treatment had a 19.8% breast meat yield, whereas the 50 and 75 ppm concentrations resulted in a 19.0% breast meat yield.

Treatment means for mortality (Table 1) at 7 wk generally increased as aerial ammonia concentration increased. The exception occurred with the 25 ppm treatment, which had 2.8% mortality. The control treatment mortality was 5.8%, which was not statistically different from the 50 ppm treatment (10.6%) but appeared different than the 75 ppm treatment (13.9%). In contrast, mortality reported by Reece et al. (1981) was 4% at the 50 ppm level, and no differences were noted for mortality in any of the treatments.

In conclusion, broilers exposed to concentrations greater than 25 ppm of atmospheric ammonia experienced a reduction in BW and generally had greater mortality. Means for yield and F:G did not meet significance criteria to indicate any detrimental effects of ammonia exposure. Body weight depression and lack of effect on F:G correspond with earlier research (Reece et al., 1981). Although the BW of today’s broiler is approximately 60% greater than 20 yr ago, the relative effects of atmospheric ammonia are similar. Decreased BW translates directly into diminished capital for broiler growers. With reductions in BW due to atmospheric ammonia quantified monetarily, the economic impact of ammonia exposure can be easily computed for ranges up to 75 ppm.

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**REFERENCES**


