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Growth Performance and Physiological Variables for Broiler Chickens Subjected to Short-Term Elevated Carbon Dioxide Concentrations

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Abstract: Four trials were conducted to evaluate growth responses, blood chemistry and heart characteristics of broiler chicks subjected to progressive concentrations (0, 3,000, 6,000, 9,000 ppm) of carbon dioxide (CO_2) gas from 1-14 days of age, which were then discontinued throughout the remainder of the trial (42 days of age). On days 14 and 42 of each trial, 20 birds per chamber were randomly selected for immediate analysis of blood partial pressure of CO_2 (pCO_2), blood partial pressure of O_2 (pO_2), blood pH, hematocrit (Hct), hemoglobin (Hb), blood electrolytes (Na^+, K^+, Ca^{2+} and Cl^-) and determination of heart characteristics. Body and feed weights were recorded at 0, 14, 28 and 42 days of age for growth performances. Final body weight (BW) gain and feed conversion were similar among the treatments, but cumulative mortality significantly increased as CO_2 increased ($P \leq 0.05$) from 3,000-9,000 ppm. Treatments did not alter blood pCO_2 and pO_2 concentrations at age 14 and 42 days of age. Increasing CO_2 up to 9,000 ppm failed to reveal differences for heart weight characteristics at 14 days of age, but total heart and left ventricle weights were increased at 42 days of age. These results indicate that subjecting chicks to progressive concentrations of CO_2 from 1-14 days of age does not adversely alter blood chemistry or cumulative growth performance, but increased the incidence of late-mortality.

Key words: Carbon dioxide, growth performance, blood chemistry, broiler, well-being

Introduction

Costs for fuel and electricity have increased dramatically in recent years, spurring growers to adopt energy conservation measures such as reduced ventilation during winter months to conserve fuel. Modern poultry housing is designed and constructed to reduce heat loss and improve energy efficiency, however when coupled with reduced ventilation, can result in elevated levels of carbon dioxide (CO_2), ammonia and other air contaminants, which may adversely affect the health and productivity of flocks.

Sources of CO_2 within a house include fuel combustion, bird respiration and ambient air content (typically 300-500 ppm). The combustion of 3.8l (1 gal) of liquid propane (LP) fuel produces approximately 3.1 m^3 (108 ft^3) of CO_2. A single radiant brooder (11,722 W or 40,000 $Btu \cdot h^{-1}$) uses approximately 1.9 $l \cdot h^{-1}$ of LP (0.5 $gal \cdot h^{-1}$) and would generate 1.6 m^3 (54.7 ft^3) of CO_2 via combustion (Czarick and Lacy, 2001). A typical broiler house measuring 12.2×152.4 m (40×500 ft) and containing 20 brooders would produce 31 m^3 (1,094.7 ft^3) of CO_2 every hour of brooder operation. Carbon dioxide concentrations may often exceed 3,000 ppm during brooding in modern housing as a result of reduced ventilation.

Air quality is essential to getting chicks off to a good start (Miles *et al*., 2004; Ritz *et al*., 2004). Subjecting broiler chickens to elevated CO_2 concentrations early during grow-out may adversely affect subsequent livability. Carbon dioxide may compete with oxygen to bind with hemoglobin contributing to hypoxia. In turn, CO_2 causes an increase in red blood cell production leading to increased resistance to blood flow (Owen *et al*., 1995) and enlargement of the heart right ventricle resulting in mortality. In addition, it has been documented that ascites syndrome is associated with high pCO_2 and low pO_2 values in venous blood, which in turn affect lung ventilation rate (Wideman *et al*., 1999).

Reece and Lott (1980) evaluated growth responses of broiler chicks exposed to 0, 3,000, 6,000 and 12,000 ppm of CO_2 from 1-28 days of age over a 49 day production period. Subjecting chicks to 12,000 ppm of CO_2 reduced final BW by 3.5% compared with the control group (0 ppm of added CO_2). The present study examined responses of broiler chickens subjected to various concentrations of CO_2 from 1-14 days of age on subsequent blood physiological variables, heart characteristics, growth performance and the incidence of mortality during a 42 days production period.

Materials and Methods

Bird husbandry: A total of 480 (Ross × Cobb 500) male chicks were obtained from a commercial hatchery and randomly distributed to 8 environmentally controlled chambers (60 chicks/chamber). Each environmental chamber had a floor area of 6 m^2 (2.3×2.6 m) with a chamber volume of 15.3 m^3. Chicks were vaccinated for Mareks disease, Newcastle disease and infectious bronchitis at the hatchery. Each chamber was equipped with fresh pine shavings, two tube feeders and a nipple watering system having seven nipples. A 3-phase feeding program was provided (1-14, 15-28 and 29-42 days of age) that consisted of corn-soybean meal based-diets formulated to either meet or exceed NRC (1994) nutrient recommendations. Starter feed was provided as crumbles and subsequent feeds were fed as whole pellets. Feed and water were offered *ad libitum*. Ambient temperature was maintained at 33°C at the start of experimentation and was reduced as the birds progressed in age to ensure comfort with a final temperature set point of 21°C at 35 days of age and thereafter. The lighting schedule consisted of continuous lighting with an intensity of 20 lx from placement to days 7, 19L:5D at 20 lx from 8-14 days, 20L:4D at 5 lx from 15-22 days and continuous lighting at 3 lx from 23-42 days. USDA animal care protocols were approved at the Mississippi State location.

Treatments: In each of 4 trials, 4 treatment groups consisted birds that were exposed to 0, 3,000, 6,000 or 9,000 ppm of carbon dioxide (CO_2) from 1-14 days of age. None of the treatment groups received CO_2 from 14-42 days of age. There were 2 replicate chambers per each treatment. Carbon dioxide administration was similar to procedures used in previous research (Reece and Lott, 1980). In each trial, CO_2 was continuously metered into 6 of the chambers to maintain 2 chambers each at 3,000, 6,000 and 9,000 ppm through panel-mount flowmeters. No CO_2 (0 ppm) was added to the remaining 2 chambers that served as controls, however CO_2 levels reflected ambient concentrations.

Carbon dioxide was measured daily at 0800, 1200, 1600 and 2000 h during the first 4 days and once a day thereafter through days 14 using a photoacoustic multigas monitor and analyzer (INNOVA-1312, Air Tech Instrument, Ballerup, Denmark). In addition, CO_2 concentration was measured before and again once or twice after disturbing the chamber atmosphere each day by animal caretakers. During the 14 days exposure period, the actual CO_2 concentration for each treatment was in good agreement with the treatment concentrations. The average concentration for the 0, 3,000, 6,000 and 9,000 ppm treatments were 350, 3,075, 6,120 and 9,155 ppm, respectively.

Body weight, blood collection and chemical analyses: Body and feed weights were recorded at 0, 14, 28 and 42 days of age for the computation of growth rate, feed intake and feed conversion. The incidence of mortality was recorded daily and feed conversion was corrected for mortality. Necropsies were performed on all birds that died during the trials. On days 14 and 42, blood samples were collected between 0800 and 0900 h on sampling day from a brachial vein of 20 randomly selected birds from each chamber and then the birds were returned to the appropriate chambers using a handling procedure described by Olanrewaju *et al.* (2006, 2007, 2008). Blood samples were collected directly into heparinized (50 $IU \cdot mL^{-1}$) monovette syringes. All bleeding were completed within 45 sec after birds were caught. Blood samples were drawn directly from the syringes into a blood gas/electrolyte analyzer (ABL-80 Flex, Radiometer America, Westlake, OH) for immediate analysis of blood partial pressure of CO_2 (pCO_2), blood partial pressure of O_2 (pO_2), blood pH, hematocrit (Hct), hemoglobin (Hb) and blood electrolytes (Na^+, K^+, Ca^{2+} and Cl^-). The pH, pCO_2 and pO_2, values were corrected to reflect a body temperature of 41.5°C (Burnett and Noonan, 1974).

Heart collection and analysis: On days 14 and 42, 20 randomly selected birds from each chamber were euthanatized by cervical dislocation for the heart characteristics. The heart was removed and the atria, great vessels and epicardial fat were trimmed off. The weight of total heart (TH), total ventricle (TV), right ventricle (RV) and left ventricle (LV) were determined. Calculation of the RV:TV ratio as a gross indicator of ascites and pulmonary hypertension syndromes (Cueva *et al.*, 1974; Peacock *et al.*, 1989; Wideman *et al.*, 1999) was determined.

Statistics: Four trials were conducted as a randomized complete block design with 4 concentrations of added CO_2. Chamber was considered as the experimental unit. Data were initially analyzed by analysis of variance (ANOVA) using PROC MIXED (SAS, 2004) for the effects of CO_2. Means comparison were assessed by LSD and statistical significance was established at $P \leq 0.05$. Linear and quadratic responses were measured using the General Linear Model procedure of SAS. Each treatment was represented by 8 replicate chambers (2 chambers/trial) with 4 trials being replicated over time. All mortality data were subjected to arc sine transformation.

Results

Body weight (BW), BW gain, feed consumption, feed conversion and mortality between 2 and 4, 4 and 6 and 2 and 6 weeks of age are given in Table 1. Body weight and BW gain were significantly reduced both linearly

Table 1: Live performance responses of male broilers subjected to various carbon dioxide concentrations from placement to 14 days of age during a 42 day production period[1,2]

		Added CO_2, ppm[3]					p-value[4]	
Days	Variables	0 (Control)[5]	3,000	6,000	9,000	SEM[6]	Linear	Quadratic
1-14	BW, kg	0.466[a]	0.454[b]	0.451[bc]	0.447[c]	0.0018	0.004	0.006
1-14	BW gain, kg	0.424[b]	0.441[a]	0.412[c]	0.407[c]	0.0022	0.037	0.041
1-14	Feed intake, kg	0.533	0.536	0.522	0.619	0.0035	0.091	0.104
1-14	FCR, kg:kg	1.265	1.281	1.282	1.273	0.0073	0.618	0.679
1-14	Mortality, %	1.0	1.0	0.3	1.4	0.22	0.804	0.913
1-28	BW, kg	1.676	1.643	1.620	1.627	0.0086	0.067	0.074
1-28	BW gain, kg	1.632	1.600	1.577	1.583	0.0085	0.066	0.077
1-28	Feed intake, kg	2.402	2.337	2.305	2.331	0.0156	0.147	0.156
1-28	FCR, kg:kg	1.473	1.461	1.462	1.473	0.0056	0.924	0.939
1-28	Mortality, %	2.7	2.2	2.3	4.5	0.43	0.186	0.214
1-42	BW, kg	3.012	2.957	2.949	2.970	0.0141	0.376	0.412
1-42	BW gain, kg	2.969	2.913	2.906	2.926	0.0141	0.367	0.483
1-42	Feed intake, kg	5.049	4.912	4.942	4.970	0.0309	0.525	0.654
1-42	FCR, kg:kg	1.704	1.688	1.700	1.700	0.0078	0.972	0.987
1-42	Mortality, %	9.6[b]	7.8[b]	10.7[b]	16.5[a]	0.99	0.025	0.029

[1]Values represent least-squares means of eight replicate chambers, [2]Means within a row that lack common superscripts differ significantly by LSD at $P \leq 0.05$, [3]CO_2 was added from placement to 14 days of age, [4]Probability values associated with linear and quadratic sources of variation, [5]No added CO_2 (Control group), [6]Pooled SEM for main effects (n = 8)

Table 2: Blood gas and electrolytes analysis of male broilers subjected to various carbon dioxide concentrations from placement to 14 days of age during a 42 day production period[1]

		Added CO_2, ppm[2]					p-Value[5]	
Days	Variables	0 (Control)[3]	3,000	6,000	9,000	SEM[4]	Linear	Quadratic
14	pH	7.39	7.39	7.38	7.38	0.02	0.384	0.461
14	pCO_2, mmHg	54.08	57.19	59.05	65.89	8.856	0.63	0.715
14	pO_2, mmHg	87.38	81.79	79.1	65.7	9.42	0.27	0.312
14	HCO_3^-, mmHg	32.02	33.35	36.77	41.69	4.328	0.476	0.617
14	Hct, %	17.18	19.3	19.35	19.48	1.819	0.582	0.674
14	Hb, $g \cdot dL^{-1}$	5.41	6.11	6.18	6.29	0.673	0.601	0.717
14	Na^+, $mEq \cdot L^{-1}$	143	143.2	142.8	144	1.53	0.852	0.785
14	K^+, $mEq \cdot L^{-1}$	4.37	4.99	5.04	5.25	1.286	0.699	0.843
14	Ca^{2+}, $mEq \cdot L^{-1}$	3.13	3.04	3.13	3.06	0.074	0.517	0.762
14	Cl^-, $mEq \cdot L^{-1}$	108.7	108.2	109.1	107.7	1.695	0.851	0.744
42	pH	7.4	7.39	7.38	7.37	0.027	0.865	0.718
42	pCO_2, mmHg	51.36	52.89	60.41	64.6	4.729	0.158	0.247
42	pO_2, mmHg	89.87	86.73	81.17	72.73	5.828	0.173	0.315
42	HCO_3^-, mmHg	29.69	31.32	36.64	40.14	4.214	0.455	0.518
42	Hct, %	19.31	20.13	20.85	21.11	0.876	0.47	0.526
42	Hb, $g \cdot dL^{-1}$	6.13	6.41	6.65	6.75	0.297	0.457	0.573
42	Na^+, $mEq \cdot L^{-1}$	144.5	144.2	144.2	145.2	0.788	0.781	0.638
42	K^+, $mEq \cdot L^{-1}$	5.57	5.7	5.74	6.42	0.434	0.513	0.644
42	Ca^{2+}, $mEq \cdot L^{-1}$	2.93	2.96	2.96	2.96	0.093	0.993	0.738
42	Cl^-, $mEq \cdot L^{-1}$	110.6	110	110.5	109.9	0.997	0.945	0.747

[1]Values represent least-squares means of eight replicate chambers. [2]CO_2 was added from placement to 14 days of age. [3]No added CO_2 (Control group). [4]Pooled SEM for main effects (n=8). [5]Probability values associated with linear and quadratic sources of variation

($p \leq 0.04$; $p \leq 0.04$) and quadratically ($p = 0.04$; $p \leq 0.04$) from 1-14 days. Feed consumption, feed conversion and mortality were similar between treatments from 1-14 days of age. From 1-28 and 1-42 days of age, BW, BW gain, feed consumption and feed conversion were not adversely affected by increasing the concentration of CO_2. However, from 29-42 days, the incidence of mortality increased both linearly ($p = 0.025$) and quadratically ($p = 0.029$) with gradient increments of CO_2. In addition, increasing CO_2 from 3,000-9,000 ppm led to a 228% increase (5.3 vs. 12.1%) in cumulative mortality from 1-42 days, which apparently may be due to pulmonary hypertension syndrome (Fig. 1). Blood gases and electrolytes analysis did not reveal significant differences ($P \geq 0.05$) between the control and CO_2 treated groups at 14 and 42 days of age (Table 2). Furthermore, heart characteristics on day 14 revealed no significant differences between the control and CO_2

Table 3: Heart characteristics of male broilers subjected to various carbon dioxide concentrations from placement to 14 days of age during a 42 day production period[1,2]

		Added CO_2, ppm[3]					p-value[4]	
Days	Variables	0 (Control)[5]	3,000	6,000	9,000	SEM[6]	Linear	Quadratic
14	HT, g	3.880	3.990	4.065	4.420	0.701	0.833	0.782
14	TV, g	2.502	2.547	2.637	2.657	0.335	0.959	0.645
14	LV, g	2.125	2.130	2.170	2.235	0.283	0.976	0.733
14	RV, g	0.377	0.417	0.467	0.422	0.055	0.928	0.846
14	RV:TV, g:g	0.1507	0.1637	0.1771	0.1588	0.008	0.241	0.377
42	HT, g	14.703[b]	15.150[ab]	15.369[ab]	16.457[a]	0.210	0.041	0.049
42	TV, g	10.595	10.862	10.993	11.237	0.517	0.659	0.711
42	LV, g	8.547[b]	8.785[ab]	8.886[ab]	9.118[a]	0.057	0.039	0.042
42	RV, g	2.048	2.077	2.107	2.119	0.212	0.85	0.758
42	RV:TV, g:g	0.193	0.191	0.192	0.189	0.014	0.270	0.311

[1]Means within a row that lack common superscripts differ significantly by LSD at $P \leq 0.05$, [2]Total heart = TH; Total ventricle = TV; Left ventricle = LV; Right ventricle = RV, [3]CO_2 was added from placement to 14 days of age, [4]Probability values associated with linear and quadratic sources of variation, [5]No added CO_2 (Control group), [6]Pooled SEM for main effects (n = 8)

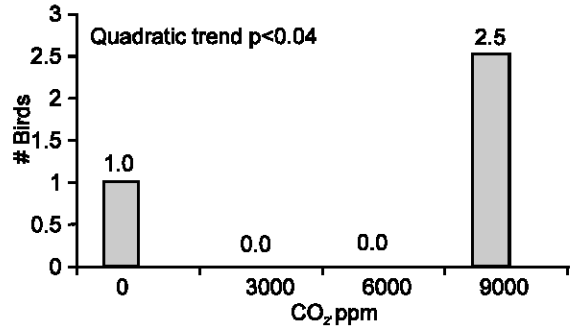

Fig. 1: Incidence of ascites per chamber from 29-42 days of age of birds subjected to progressive CO_2 concentrations from 1-14 days of age

treated groups in total heart (TH), total ventricle (TV), right ventricle (RV) or left ventricle (LV) weights (Table 3). Consequently, data were re-grouped by RV:TV (RV:TV < 0.25 = normal; RV:TV≥0.25 = abnormal) within each treatment in an attempt to detect CO_2-related differences that might have been masked by the chamber. The resulting comparisons did not reveal chamber or CO_2-related differences. However, increasing CO_2 from 3,000-9,000 ppm led to a significant (Linear $p \leq 0.041$; Quadratic $p \leq 0.049$) increase in total heart and a significant (Linear $p \leq 0.039$; Quadratic $p \leq 0.042$) increase in left ventricle weight at 42 days of age, but no significant differences in right ventricle weight and RV:TV ratio were observed.

Discussion

Broiler chickens subjected to CO_2 concentrations of up to 9,000 ppm had similar cumulative live performances compared with the control birds in the research reported herein. Subjecting chicks to progressive concentrations of CO_2 limited growth rate at 14 days of age, but growth rate differences were not apparent at 28 and 42 days of age. Reece and Lott (1980) subjected broilers to CO_2 additions of 0, 3,000, 6,000, or 12,000 ppm from 1-28 days, which were subsequently discontinued from 29-49 days of age. These authors reported similar results, showing that subjecting broilers to CO_2 up to 6,000 ppm did not alter 28 and 49 day BW gain and feed conversion, while exposure to 12,000 ppm of CO_2 limited BW by 8 and 3.5%, respectively, at 4 and 7 weeks of age. In the present study, the addition of CO_2 had a pronounced effect on late-mortality. Increasing CO_2 from 3,000-9,000 ppm increased cumulative mortality, which is most likely attributed to pulmonary hypertension syndrome. In addition, elevated CO_2 from 3,000-9,000 ppm increased total heart and left ventricle weights, but right ventricle weight and RV:TV ratios were not affected by the treatments. Ratio of the right ventricle to the total ventricle mass has been used to quantitatively correlate increased blood pressure in the right ventricle with the incidence of pulmonary hypertension syndrome (Wideman and French, 1999). Broilers having RV:TV ratios averaging < 0.28 is indicative of normal pulmonary arterial pressure, whereas RV:TV values ≥ 0.28 denote pulmonary hypertension syndrome (Julian, 1987; Wideman and French, 1999; Wideman, 2001). Other research has shown that broilers exposed to 6,000 ppm of CO_2 up to 5 weeks of age did not develop the incidence of ascites syndrome (McGrovern *et al.*, 2001). In conclusion, subjecting broilers to elevated CO_2 concentrations from 1-14 days resulted in similar cumulative growth performance, but increased the incidence of late-mortality and altered heart characteristics.

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