Research Notes

Graded Atmospheric Oxygen Level Effects on Performance and Ascites Incidence in Broilers[1]

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ABSTRACT The effects of graded atmospheric O_2 concentration (12, 14, 16, 18, and 20.6%) on chick performance and propensity to develop ascites were investigated using commercial male broilers. Chicks were housed in calorimetry chambers for 2 wk with incoming air diluted with N to provide the desired O_2 concentration at thermoneutral (TN) ambient temperature. Day 14 body weight, weight gain, feed consumption, and gain-to-feed ratio increased ($P < 0.01$) as O_2 concentration incrementally rose from 12 to 20.6%. Body weight was 138 g for the lowest atmospheric O_2 level compared to 371 g for 20.6% O_2. The greatest treatment difference occurred between the 12 and 14% O_2 concentrations. Growth depression appeared related to feed consumption. Ascites heart ratio (AHR), ascites score (AS), right ventricular mass (RVM), and hematocrit (HCT) all increased ($P < 0.01$) as O_2 concentration decreased. Blood HCT appeared to be a more sensitive indicator of physiological change attributable to atmospheric O_2 than AHR, AS, or RVM. The data reported herein suggests that 19.6% atmospheric O_2 is the minimal allowable level for housing birds within a relatively stress-free, TN environment to avoid cardiac and HCT changes related to ascites.

(*Key words*: ascites, ascites heart ratio, hematocrit, oxygen concentration threshold, broiler)

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INTRODUCTION

Over the years genetic selection has elevated broiler productivity markedly. Occasionally, however, undesirable characteristics such as ascites susceptibility have also emerged. Ascites syndrome, initially thought to be attributable to high altitude (Hernandez, 1987), also occurs at lower elevations (Dale and Villacres, 1986; Huchzermeyer and DeRuyck, 1986). Although advances in genetic selection (Julian and Wilson, 1984) have been made, ascites occurrence in high-yielding broilers continues. Experimentally, ascites is usually induced with combinations of temperatures below the accepted thermoneutral (TN) ambient temperature range and simulated high altitude. High altitude can be simulated by use of hypobaric chambers (Owen et al., 1990) or by O_2 dilution (Beker et al., 1995).

Julian (1993) and Witzel et al. (1990) exposed 1-wk-old broilers to simulated altitudes of 1,980, 2,590, and 2,895 m and observed that for each ascites incidence at 1,980 m, two occurred at 2,590 m and six occurred at 2,895 m. Additionally, 6-wk-old birds exposed to 2,895 m weighed 530 g less than those reared at 1,980 m. Ascites syndrome is primarily caused by a hypoxia-induced pulmonary hypertension. Teeter and Wiernusz (unpublished data) observed that the O_2 requirement per g of lean and fat accretion to 42 d of age commercial type birds in 1994 averaged 3.9 and 1.2 L, respectively. This finding suggests that as growth increases, particularly lean tissue, the need for O_2 consumption will increase. Van der Hel et al. (1988), compared 1-d-old chicks exposed to 15% O_2 with control birds exposed to 20.9% O_2. Ascites was observed in the low-O_2 exposure birds at 21 d, and at 32 d, they weighed 600 g less than control birds with a packed cell volume 50% higher than that of control birds. Beker et al. (1995) demonstrated an inverse relationship between oxygen consumption and ascites incidence ($R^2 = 0.96$) in birds housed within calorimetry chambers while exposed to 17.6% O_2. Presumably, other factors influencing metabolic rate, such as ambient temperature (AT) (Julian et al., 1989; Shlosberg et al., 1992) and immune challenge (Klasing et al., 1987) would also elevate ascites incidence. Frequently, the aforementioned studies investigating ascites used combinations of altitude and cold stress. Consequently, little data are available where atmospheric O_2 has been titrated in a relatively stress-free environment, such that

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Abbreviation Key: AHR = ascites heart ratio; AS = ascites score; AT = ambient temperature; HCT = hematocrit; RVM = right ventricular mass; RV = right ventricle; TN = thermoneutral.

TABLE 1. Composition of diet used in the study

Ingredient	Percentage (as fed basis)
Corn	52.96
Soybean meal	37.67
Vegtable oil	5.16
Dicalcium phosphate	2.00
Calcium carbonate	1.24
Salt	0.41
Vitamin mix[1]	0.25
Trace minerals[2]	0.10
DL-Methionine	0.21
Coban	0.05
Selenium premix	0.0015
Calculated analysis	
ME kcal/kg	3,150
Crude protein (%)	22.50

[1]Mix supplied the following per kilogram of diet: vitamin A, 38,500 IU; vitamin D_3, 11,000 IU; vitamin E, 55 IU; vitamin B_{12}, 0.066 mg; riboflavin, 33 mg; niacin, 165 mg; D-pantothenic acid, 55 mg; menadione, 11 mg; folic acid, 3.3 mg; pyridoxine, 13.75 mg; thiamin, 6.66 mg; D-biotin, 0.28 mg.

[2]Mix supplied the following per kilogram of diet: manganese, 120 mg; zinc, 100 mg; copper, 10 mg; iodine, 2.5 mg; calcium, 135 mg.

the atmospheric O_2 effects may be independently examined. The objectives of the study reported herein were to examine dose titration effects of atmospheric O_2 on ascites incidence and broiler performance independent of cold stress.

MATERIALS AND METHODS

Ninety-four male broilers were obtained from a commercial hatchery and transferred to open-circuit respiratory chambers. The metabolic chambers and general operational procedures have been described elsewhere (Belay and Teeter, 1993; Wiernusz and Teeter, 1993; Beker et al., 1995). Chicks were wing-banded, weighed, and divided into 15 groups of six chicks each and one group of four. Chick groups were then assigned at random to 16 respiratory chambers housed within two thermostatically controlled rooms (eight chambers/room). The ratio of air to nitrogen was varied such that chicks were exposed to 12, 14, 16, 18, and 20.6 % O_2. Birds were initially housed at 32°C AT, with 24 h of fluorescent light with feed (Table 1) and water available ad libitum. Temperature was decreased 2°C at 7 d. Chicks were closely monitored for mortality three times daily, and all mortalities were removed, weighed, and examined grossly for ascites lesions. Group feed consumption and individual live weights were determined at the end of the study such that the gain-to-feed ratio could be determined. The study was terminated on d 14 with all remaining birds weighed, bled for hematocrit via wing vein puncture, and euthanized by CO_2 (AVMA, 1986). Packed cell volume (HCT) was determined by microcentrifugation. At necropsy, each chick was examined for gross lesions of ascites and scored (AS) as follows: 0 = no ascites, 1 = fluid in pericardial sac and dilated right ventricle, 2 = small amount of ascitic fluid in abdominal cavity, 3 = large amount of ascitic fluid accumulation in the abdominal cavity. Individual hearts and right ventricle (RV) were removed and weighed to obtain heart weight and RV mass (RVM) and the ascites heart ratio (AHR; RVM/heart weight; expressed as percentage) was determined for each bird.

Statistical Analysis

Data for all response variables were subjected to ANOVA using the general linear models procedure of SAS software (SAS Institute, 1985). When the F-test was significant, treatment means were separated using least significant difference (Steel and Torrie, 1960). Regression equations were calculated relating HCT to atmospheric O_2 concentrations. Broken line and exponential models were fit using a nonlinear program of SAS software (1985). Broken line regression (Hinkley, 1971) was used so that the minimum amount of atmospheric O_2 associated with the onset of ascites in broilers could be estimated.

RESULTS AND DISCUSSION

The study was completed with consistent delivery of the desired O_2 concentration to the metabolic chambers. Results are displayed in Tables 2 and 3. Final BW, BW gain, feed consumption, and gain-to-feed ratio for 0 to 14 d increased quadratically ($P < 0.01$) as the O_2 concentration rose from 12 to 20.6%. The BW responses are in general agreement with the results reported by Van der Hel et al. (1988), Maxwell et al. (1990), and Witzel et al. (1990) in which birds reared at lower atmospheric O_2 were observed to have reduced BW gain. A previous study conducted in our laboratory (Vanhooser et al., 1995) also demonstrated that low environmental O_2 severely reduced weight gain and feed efficiency of broiler chicks, presumably due to inadequate O_2 to support normal metabolic efficiency and reduced feed consumption. In the

TABLE 2. Means and SE of feed consumption (Feed) BW, weight gain (gain), and gain-to-feed ratio of broiler chicks reared to 14 d of age by atmospheric O_2 concentration

	O_2 concentration (%)					
Variable	12	14	16	18	20.6	Probability
BW (g)	138 ± 20.96[c]	287 ± 12.83[b]	353 ± 7.41[a]	356 ± 7.41[a]	371 ± 18.15[a]	0.0001
Gain (g)	92.00 ± 20.67[c]	243 ± 12.65[b]	308 ± 7.31[a]	312 ± 7.31[a]	329 ± 17.90[a]	0.0001
Feed (g)	205 ± 9.58[c]	280 ± 9.58[b]	399 ± 8.29[a]	396 ± 8.29[a]	384 ± 20.32[a]	0.0001
Gain:feed	0.46 ± 0.05[b]	0.78 ± 0.03[a]	0.78 ± 0.02[a]	0.79 ± 0.02[a]	0.86 ± 0.05[a]	0.0001

[a–c]Means within a row with no common superscript differ.

TABLE 3. Means and SE of hematocrit (HCT), ascites score (AS), right ventricular (RV) mass, and ascites heart ratio (AHR) of broiler chicks reared to 14 d of age by atmospheric O_2 concentration

	O_2 concentration (%)					
Variable	12	14	16	18	20.6	Probability
HCT (%)	48.92 ± 0.46[a]	42.24 ± 0.46[b]	35.77 ± 0.57[c]	32.60 ± 0.40[d]	31.88 ± 0.98[d]	0.0001
AS	3.00 ± 0.39[a]	2.23 ± 0.19[b]	0.67 ± 0.14[c]	0.38 ± 0.14[c]	0.25 ± 0.14[c]	0.0001
RV(g)	0.79 ± 0.04[a]	0.82 ± 0.04[a]	0.46 ± 0.03[b]	0.38 ± 0.03[b]	0.34 ± 0.08[b]	0.0001
AHR	52.36 ± 2.00[a]	43.37 ± 2.00[b]	24.00 ± 1.74[c]	22.91 ± 1.74[c]	20.99 ± 4.25[c]	0.0001

[a–d]Means within a row with no common superscript differ.

present study, AHR, AS, RVM, and HCT all increased quadratically ($P < 0.01$) as the O_2 concentration decreased from 20.6 to 12%. A marked HCT increase at lower O_2 concentration is in agreement with earlier reports (Hernandez, 1987; Maxwell et al., 1990; Vanhooser et al., 1995). Hernandez (1987) further reported that hemoglobin and HCT increased by 40% in ascetic broilers vs. nonascitic broilers housed at 2,630 m above sea level. Increased AHR at reduced atmospheric O_2 is in general agreement with the findings of Cueva et al. (1974).

The first physiological change observed in our laboratory with high-altitude exposure has consistently been the change in HCT (Beker et al., 1995). Consequently it was used here to guide altitude consequences (Figure 1). Regressing blood HCT on atmospheric O_2 concentration reveals a good relationship as a second order polynomial:

$$\text{HCT (\%)} = 137.36 - 10.42 \times O^2 + 0.26 \times O_2^2$$
$$(R^2 = 0.92;\ P = 0.0001)$$

where HCT = percentage hematocrit, and O_2 = oxygen concentration.

Figure 1 depicts the HCT response of broilers to graded atmospheric oxygen concentrations. The HCT reading increased by 35% as the level of O_2 decreased from 20.6 to 12%. Nonetheless, as is also shown in Table 3, HCT values were similar for atmospheric O_2 concentrations between 18.0 and 20.6%. A plateau value from the nonlinear regression procedure provided an estimated requirement of 19.6% atmospheric O_2 as a breakpoint. This finding suggests that the breakpoint for ascites-related changes, in the absence of other stress factors, is 19.6% O_2. However, it should be emphasized that genetic change in this area is still evolving. Most breeding companies report progress is due to selection for ascites resistance.

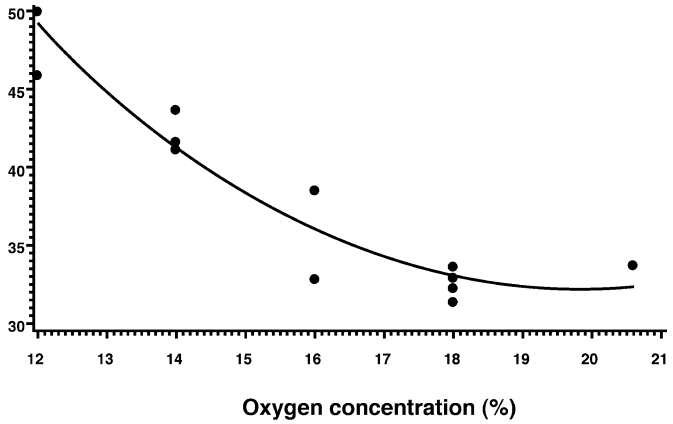

FIGURE 1. Plot of hematocrit (%) versus atmospheric O_2 concentration (%).

In conclusion, O_2 concentration plays a major role on the onset of ascites even at accepted thermoneutral temperatures. Reductions in atmospheric O_2 concentration effectively induced ascites. It is important to keep the O_2 concentration above 19.6% to minimize ascites-related anomalies and to maximize performance at accepted TN conditions.

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