ENVIRONMENT AND HEALTH

Variation in Toe-Web Response of Turkey Poults to Phytohemagglutinin-P and Their Resistance to *Escherichia coli* Challenge

G. R. BAYYARI, W. E. HUFF, J. M. BALOG, and N. C. RATH

USDA, Agricultural Research Service, Poultry Production and Product Safety Research, Poultry Science Center, University of Arkansas, Fayetteville, Arkansas 72701

ABSTRACT One thousand 5-wk-old male turkeys from each of two commercial strains (A and B) were grouped into low, medium, and high responders based on the cutaneous basophil hypersensitivity (CBH) response obtained 24 h after toe-web inoculation with 100 μg of phytohemagglutinin-P (PHA-P). The CBH response for Strain A was higher than strain B (P = 0.00001) and ranged from 0 to 1.95 mm, with a mean of 0.66, whereas the CBH response for Strain B ranged from 0 to 1.67 mm with a mean of 0.38. At 6 wk of age, 36 birds from each of the six response groups were inoculated into the left thoracic air sac with 1.5 × 10^7 cfu of an early log phase broth culture of *Escherichia coli*. Samples of 5 or 10 birds were necropsied from each of the six groups at 7, 14, 28, and 42 d postinfection (PI). Birds were scored for air-sacculitis/pericarditis (AS) and turkey osteomyelitis complex (TOC). Overall mortality of birds inoculated with *E. coli* was 31%. There were no mortalities in unchallenged controls. Strain A had significantly higher Week 1 mortality, marginally higher overall mortality (P = 0.1), and higher AS scores than Strain B. There were no TOC lesions detected until 7 d PI, after which all mortalities had TOC lesions in multiple sites. The differences in CBH response within each strain were not clearly correlated to *E. coli* susceptibility. However, these data suggest that air sac inoculation of *E. coli* can provide a useful model for the study of TOC. The greater incidence of disease in Strain A indicates that an enhanced inflammatory response may increase susceptibility to *E. coli* septicemia.

(Key words: turkey osteomyelitis complex, hypersensitivity, *Escherichia coli*, air sacculitis, pericarditis)

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INTRODUCTION

Turkey osteomyelitis complex (TOC) is a disease defined by the U.S. Food Safety and Inspection Service (FSIS) to include processed turkeys that have a green liver, arthritis, synovitis, or soft tissue abscesses with or without osteomyelitis (Barnes et al., 1990; Clark et al., 1991; Bayyari et al., 1994; Droual et al., 1996; Mutalib et al., 1996). Besides being an important cause of late mortality and production losses, this disease is also viewed by the FSIS as a potential public health concern. Whole turkeys with hidden foci of infection could unknowingly be marketed and the bacteria in these lesions could be incorporated into processed turkey products. These infections often contain *Staphylococcus aureus* and *Escherichia coli*, which create the potential for food poisoning.

In addition to these predominant species, many other opportunistic species of bacteria have been associated with such lesions (Clark et al., 1991; Bayyari et al., 1994), suggesting that the disease may be more the result of inefficient phagocytosis or bactericidal activity rather than bacterial virulence (Bayyari et al., 1997a). Turkeys with TOC have been shown to have fewer lymphocytes and decreased in vivo and in vitro T cell function than clinically healthy birds (Bayyari et al., 1997a). It is not known whether these differences are innate, acquired prior to infection, or simply a result of the physiological response to infection. It has been suggested that selection of both chickens and turkeys for fast growth has resulted in decreased humoral and cellular immune responses (Han and Smyth, 1972; Saif et al., 1984; Sacco et al., 1991, 1994; Miller et al., 1992; Tsai et al., 1992; Qureshi and Havenstein, 1994). Divergent selection for increased immune responses has also been shown to result in decreased body weights (Siegel and Gross, 1980; Siegel et al., 1982; van der Zijpp, 1983; Okada et al., 1988; Martin et al., 1990; Afraz et al., 1994). Previous work in this laboratory has revealed that turkeys selected for fast growth had a lower cutaneous basophil hypersensitivity (CBH) response to phytohemagglutinin-P (PHA-P), lower lymphocyte counts, and lower relative spleen weights than their smaller parent line (Bayyari et al., 1997b). We have hypothesized that such selection may result in birds that are generally more susceptible...
to the opportunistic bacterial infections that result in TOC lesions, and that this disease may be evidence of a subset of birds with the least resistance to these infections. The purpose of this experiment was to determine whether the CBH response to the cutaneous injection of PHA-P, a relatively simple method of determining the cellular immune responsiveness of turkeys (McCorkle et al., 1983; Scott and Siopes, 1994), could be used to define a population within commercial turkey strains that is more susceptible to bacterial infection leading to TOC.

MATERIALS AND METHODS

Toe-Web Hypersensitivity

One thousand male poults from each of two commercial strains, Strain A and Strain B, were wing-banded at day of hatch and placed in floor pens. Poults were maintained on a standard turkey starter ration that met or exceeded the nutrient requirements established by the NRC (1994) for 5 wk, at which time CBH was measured using the methodology of Corrier and Deloach (1990). Briefly, the right foot was cleansed with 70% ethanol and the thickness of the toe web between the third and fourth digits was measured using a micrometer. One hundred microliters of a 100 mg/mL solution of phytohemagglutinin-P (PHA-P)3 in sterile 0.85% saline was injected intradermally. After 24 h, the toe webs were cleansed and measured again. Cutaneous basophil hypersensitivity response was determined as the difference in skin thickness before and 24 h postinjection. Previous studies demonstrated no differences between these values and CBH response calculated as the difference between response to PHA-P in the right foot and response to a control injection of saline in the left foot at 24 h postinjection. Responses of each strain were ranked from highest to lowest and compared using the test procedure of SAS® software (SAS Institute, 1988). Significant differences between treatments were separated using Duncan’s multiple range test (Duncan, 1955). Comparisons were made between Strain A and Strain B using the t test procedure of SAS® software. Statements of significance are based on the probability level of 0.05 unless otherwise stated.

E. coli Challenge

Thirty-six birds from each CBH response group (low, medium, and high) of both Strain A and Strain B were housed in six floor pens. Body weights were measured at the beginning and end of the experiment and feed consumption was determined. Blood smears were prepared for differential WBC counts from each bird. A pathogenic E. coli isolate was grown in tryptose phosphate broth (TPB)4 for 2.5 h in a 37 C shaking water bath and diluted 1:1 with TPB. The inoculum contained 7.5 x 107 cfu/mL. A 200-μL aliquot was injected into the left cranial-thoracic air sac. Birds were held in lateral recumbency with wings and legs extended, and the inoculum injected using a 0.5-in, 28-gauge insulin needle.

Dead birds were collected twice daily, weighed, and examined for lesions of colibacillosis and TOC. At 7, 14, 28, and 42 d postinfection (PI), 5 or 10 birds from each group were bled, euthanatized, and examined for lesions of colibacillosis and TOC. Gross lesions of air sacculitis/pericarditis were scored using a modification of the system described by Piercy and West (1976). Gross bone and joint lesions, liver samples, and pericardium from each bird were cultured for E. coli in MacConkey agar.4

Relative differential WBC counts were determined from whole blood smears from 5 birds from each group before challenge and at 7 d PI, and from 10 birds from each group at 14 d PI. Blood smears were prepared using the methodology described by Campbell (1988). Total relative leukocyte counts included heterophils, lymphocytes, monocytes, basophils, and eosinophils. Impression smears were made from a thin bone section cut from the top of the proximal tibia of each bird and were stained with the Diff Quik Stain Set.5

Livers, spleens, heart, and bursae of Fabricius were weighed. All organ weights were expressed as a percentage of body weight and subjected to arc sine transformation. All data were analyzed by ANOVA (Snedecor and Cochran, 1967) using the General Linear Models procedure of SAS® software (SAS Institute, 1988). Significant differences between treatments were separated using Duncan’s multiple range test (Duncan, 1955). Comparisons were made between Strain A and Strain B using the t test procedure of SAS® software. Statements of significance are based on the probability level of 0.05 unless otherwise stated.

RESULTS

The mean CBH response at 24 h postinjection with PHA-P was higher for Strain A than for Strain B (P = 0.00001). The CBH response for Strain A ranged from 0 to 1.95 mm, with a mean value of 0.66, whereas the CBH response for Strain B ranged from 0 to 1.67 mm with a mean value of 0.38 (Figure 1).

Body weights of high response Strain B birds inoculated with E. coli were lower than those of unchallenged controls and BW of high response Strain B birds were lower than those of low response birds (Table 1). Body weights of each Strain A response group were lower than the corresponding BW of the Strain B response group. All groups had significantly higher relative liver and heart weights than the unchallenged control. The high response Strain B group and all of the Strain A response groups had higher relative spleen

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3Sigma Chemical Co., St. Louis, MO 63178-9916.
4Difco, Detroit, MI 48232-7058.
5Baxter Healthcare Corp., Miami, FL 33152-0672.
weights than the unchallenged controls (Table 1). The bursa:spleen ratio of all challenged groups was lower than that of the unchallenged control, and the bursa:spleen ratio of the Strain A low response group was lower than that of the Strain B low response group (Table 1).

Overall mortality of challenged birds was 31% and there were no deaths in unchallenged controls (Figure 2). The first death occurred at 1 d PI and mortality peaked at 3 d PI; at this time there were more deaths of high responders than of low responders in both strains, but this difference was not significant (Table 2). Strain A had significantly higher Week 1 mortality and marginally higher overall mortality ($P = 0.1$) than Strain B (Figure 2). Pericarditis, airsacculitis, perihepatitis, and peritonitis were seen in mortalities starting 2 d PI. Green livers were first seen 7 d PI, and only in euthanatized birds, not mortalities. After 7 d PI, all dead birds had lesions of TOC including osteomyelitis and soft tissue abscesses in multiple sites. At 42 d PI, Strain A had significantly higher cumulative incidence of green-discolored liver ($P = 0.01$), and airsacculitis/pericarditis lesion scores ($P = 0.02$) than Strain B (Table 3). Strain A had a marginally higher incidence of TOC ($P = 0.1$) and isolation of $E. coli$ from pericardium or blood ($P = 0.08$). All challenged turkeys had significantly higher levels of mortality and airsacculitis than unchallenged control birds (Table 4). All challenged Strain A turkeys had a higher incidence of TOC lesions than the unchallenged controls (Table 4). Impression smears of the proximal tibia of mortalities showed evidence of inflammation (Figure 3) and bacterial infection (Figure 4) starting 2 d PI in birds with no gross signs of osteomyelitis.

At 7 d PI, heterophil percentages were marginally higher ($P = 0.1$) in the medium response groups of both

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**FIGURE 2.** Cumulative mortality over 6 wk of turkeys challenged with $Escherichia coli$. Week 1 mortality was significantly higher in Strain A turkeys.

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**TABLE 1.** Effect of $Escherichia coli$ challenge on cumulative BW, liver, bursa of Fabricius, spleen, and heart relative weights, and the ratio of bursa weight to spleen weight of turkeys segregated into high, medium, and low cutaneous basophil hypersensitivity (CBH) response groups from Strains A and B

<table>
<thead>
<tr>
<th>Group</th>
<th>BW (g)</th>
<th>Liver (% BW)</th>
<th>Bursa (% BW)</th>
<th>Spleen (% BW)</th>
<th>Heart (% BW)</th>
<th>Bursa:Spleen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control$^1$</td>
<td>4,704 ± 434$^a$</td>
<td>2 ± 0.1$^b$</td>
<td>0.12 ± 0.006</td>
<td>0.12 ± 0.005$^b$</td>
<td>0.5 ± 0.01$^b$</td>
<td>96 ± 4$^a$</td>
</tr>
<tr>
<td>B-Low</td>
<td>4,177 ± 417$^a$</td>
<td>3 ± 0.2$^a$</td>
<td>0.11 ± 0.005</td>
<td>0.15 ± 0.01$^a$</td>
<td>0.6 ± 0.03$^a$</td>
<td>82 ± 5$^a$</td>
</tr>
<tr>
<td>B-Med</td>
<td>3,863 ± 391$^{ab}$</td>
<td>3 ± 0.2$^a$</td>
<td>0.10 ± 0.007</td>
<td>0.14 ± 0.008$^{ab}$</td>
<td>0.6 ± 0.04$^a$</td>
<td>76 ± 6$^{bc}$</td>
</tr>
<tr>
<td>B-High</td>
<td>3,116 ± 299$^{bc}$</td>
<td>3 ± 0.3$^a$</td>
<td>0.10 ± 0.007</td>
<td>0.17 ± 0.01$^a$</td>
<td>0.7 ± 0.06$^a$</td>
<td>68 ± 5$^{bc}$</td>
</tr>
<tr>
<td>A-Low</td>
<td>2,374 ± 249$^{cd}$</td>
<td>3 ± 0.2$^a$</td>
<td>0.10 ± 0.005</td>
<td>0.16 ± 0.009$^a$</td>
<td>0.7 ± 0.05$^a$</td>
<td>64 ± 4$^c$</td>
</tr>
<tr>
<td>A-Med</td>
<td>2,702 ± 295$^{cd}$</td>
<td>3 ± 0.2$^a$</td>
<td>0.10 ± 0.006</td>
<td>0.18 ± 0.02$^a$</td>
<td>0.6 ± 0.04$^a$</td>
<td>70 ± 5$^{bc}$</td>
</tr>
<tr>
<td>A-High</td>
<td>1,963 ± 141$^e$</td>
<td>3 ± 0.2$^a$</td>
<td>0.11 ± 0.005</td>
<td>0.17 ± 0.01$^a$</td>
<td>0.7 ± 0.04$^a$</td>
<td>74 ± 5$^{bc}$</td>
</tr>
</tbody>
</table>

$^a$$^{cd}$Means within a column with no common superscript differ significantly ($P ≤ 0.05$).

$^1$Control represents unchallenged Strain B turkeys.
TABLE 2. Mortality at 3 d postinfection of turkeys separated into high, medium, and low cutaneous basophil hypersensitivity (CBH) response groups and challenged with air sac inoculation of Escherichia coli. Unchallenged controls had no deaths.

<table>
<thead>
<tr>
<th>Strain</th>
<th>High responders</th>
<th>Medium responders</th>
<th>Low responders</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>10 (28%)</td>
<td>8 (22%)</td>
<td>6 (17%)</td>
</tr>
<tr>
<td>B</td>
<td>6 (17%)</td>
<td>3 (8%)</td>
<td>5 (13%)</td>
</tr>
<tr>
<td>Total</td>
<td>16 (23%)</td>
<td>11 (15%)</td>
<td>11 (15%)</td>
</tr>
</tbody>
</table>

strains than in unchallenged controls. There were no significant differences in lymphocyte percentages; however, challenged groups had counts from 14 to 40% lower than those of unchallenged controls (Table 5). There were no differences in the prechallenge percentages. The 7 d postchallenge heterophil:lymphocyte ratio of Strain A medium response birds was higher ($P = 0.08$) than that of all other groups except the Strain B medium response group (Table 5). All challenged birds had lower percentages of monocytes than did unchallenged controls; however, these differences were significant only in Strain B birds and in the low response group of Strain A. (Table 5). There were no significant differences in relative percentages among leukocytes at 14 d PI (data not shown). The relative mean numbers of lymphocytes, monocytes, and total leukocytes counted in 50 fields of a peripheral blood smear monolayer before inoculation with $E. coli$ were significantly lower in Strain A. The number of heterophils was marginally lower in Strain A ($P = 0.09$) (Table 6). The relative mean number of lymphocytes counted 7 d PI was significantly lower in Strain A but decreased in Strain B (Table 6).

DISCUSSION

Field reports imply that Strain A turkeys have higher early mortality, but lower incidence of leg problems and condemnations at processing than Strain B turkeys. In this study, Strain A had a higher CBH response to PHA-P. This higher response was accompanied by higher Week 1 mortality and higher air sacculitis/pericarditis lesion scores. These results suggest that an enhanced inflammatory response was involved in the pathogenesis of this experimentally produced colisepticemia.

It has been previously reported that genetically selected strains of chickens with a high antibody response to SRBC had more weight loss, air sacculitis, and mortality when challenged with $E. coli$ than birds with a low antibody response (Gross et al., 1980; Dunnington et al., 1991). Divergent selection based on a measurement similar to that used in this study, the delayed-type hypersensitivity wattle reaction to Bacillus Calmette-Guerin antigen, demonstrated that a high response was correlated with decreased resistance to Marek's disease virus challenge (Afraz et al., 1994). These studies indicate that birds with high immune responses may sometimes be more susceptible to disease than birds with an average response. In the study

TABLE 3. Mean cumulative percentage incidence of turkey osteomyelitis complex (TOC) lesions, green liver, isolation of Escherichia coli from blood or pericardium, and mean air sacculitis/pericarditis scores of Strain A and Strain B turkeys 42 d after air sac inoculation with $E. coli$

<table>
<thead>
<tr>
<th>Strain</th>
<th>TOC lesions (%)</th>
<th>Green liver (%)</th>
<th>$E. coli$ Isolation (%)</th>
<th>Air sac scores $^1$</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>22</td>
<td>14</td>
<td>43</td>
<td>2.9 ± 0.18</td>
</tr>
<tr>
<td>B</td>
<td>14</td>
<td>4</td>
<td>31</td>
<td>2.3 ± 0.2</td>
</tr>
<tr>
<td>$P$ value</td>
<td>0.1</td>
<td>0.01</td>
<td>0.08</td>
<td>0.02</td>
</tr>
</tbody>
</table>

$^1$Mean and SE. Each bird was given a score of 0 to 5 based on severity of lesions.

FIGURE 3. Impression smears of the proximal tibia of mortalities starting 2 d postinfection showed evidence of inflammation in bones not grossly affected with osteomyelitis.

TABLE 4. Mean cumulative percentage incidence of turkey osteomyelitis complex (TOC) lesions, green liver, isolation of Escherichia coli from blood or pericardium, and mean air sacculitis/pericarditis scores 42 d after air sac inoculation of low, medium, and high cutaneous basophil hypersensitivity response groups from Strains A and B with $E. coli$

<table>
<thead>
<tr>
<th>Group</th>
<th>Mortality (%)</th>
<th>Mean air sac score ± SE</th>
<th>$E. coli$ isolation (%)</th>
<th>Green liver (%)</th>
<th>TOC lesions (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control$^1$</td>
<td>0$^b$</td>
<td>0.2 ± 0.1$^c$</td>
<td>4$^b$</td>
<td>4</td>
<td>0$^b$</td>
</tr>
<tr>
<td>A-Low</td>
<td>36$^a$</td>
<td>2.9 ± 0.3$^a$</td>
<td>42$^a$</td>
<td>14</td>
<td>28$^a$</td>
</tr>
<tr>
<td>A-Med</td>
<td>31$^a$</td>
<td>3.0 ± 0.3$^a$</td>
<td>42$^a$</td>
<td>11</td>
<td>19$^a$</td>
</tr>
<tr>
<td>A-High</td>
<td>40$^a$</td>
<td>2.8 ± 0.3$^a$</td>
<td>46$^a$</td>
<td>17</td>
<td>20$^a$</td>
</tr>
<tr>
<td>B-Low</td>
<td>22$^a$</td>
<td>1.9 ± 0.3$^b$</td>
<td>27$^a$</td>
<td>5</td>
<td>8$^a$</td>
</tr>
<tr>
<td>B-Med</td>
<td>28$^a$</td>
<td>2.6 ± 0.3$^ab$</td>
<td>31$^a$</td>
<td>3</td>
<td>17$^ab$</td>
</tr>
<tr>
<td>B-High</td>
<td>34$^a$</td>
<td>2.5 ± 0.4$^ab$</td>
<td>37$^a$</td>
<td>3</td>
<td>17$^ab$</td>
</tr>
<tr>
<td>$P$ Value</td>
<td>0.01</td>
<td>0.0001</td>
<td>0.01</td>
<td>0.1</td>
<td>0.07</td>
</tr>
</tbody>
</table>

$^a,b$Means within a column with no common superscript differ significantly as stated.

$^1$Control represents unchallenged Strain B turkeys.
reported here, weight loss in response to bacterial challenge was markedly increased in the highest responders of both strains. Stimulation of the immune system with bacterial endotoxin is known to decrease performance of chicks (Klasing and Austic, 1984; Klasing et al., 1987; Cook et al., 1993). Gross et al. (1980) reported that a strong immune response was associated with increased susceptibility to both E. coli and S. aureus challenge and with decreased BW. They suggested that animals having near average immune responses in a specific test were more likely to resist infection than animals with high or low responses to any specific factor, and that animals that develop infections are near the genetic fringes of the population for some immune response.

The 7 d PI heterophil percentages of the medium response groups in this study were higher than those of the lowest and highest response groups. This result may predict an increased ability to respond to challenge in the medium response group, as an early heterophil response has been linked to resistance to E. coli challenge (Gross, 1962). In this study, there was no significant increase in the resistance of the medium response birds; however, at 3 d PI, the Strain B medium response birds had approximately half of the mortality of the high and low responders. The relative mean number of prechallenge white blood cells counted in 50 fields was lower for Strain A turkeys and may represent leukopenia associated with disease susceptibility or may reflect a greater tissue infiltration of leukocytes in response to PHA-P. The increase in peripheral blood monocytes at 7 d PI in Strain A may reflect the higher incidence of disease in these birds.

It has been shown that cell-free culture filtrates of E. coli and Pasteurella multocida can induce an inflammatory response that is indistinguishable from that produced by bacterial inoculation into the air sac (DeRosa, et al., 1992; Ficken et al., 1991). In addition, pretreatment with cyclophosphamide reduces the inflammatory changes in the air sac of inoculated birds. In our study, damage to air sac tissue and vasculature caused by inflammation may have led to bacteremia and higher mortality in Strain A turkeys.

Because the segregation of each strain into high, medium, and low responders failed to predict their response to E. coli challenge, this test may not be sensitive enough to be a useful predictor of the susceptibility of turkeys to TOC. However, this study does demonstrate that air sac inoculation of E. coli can be a useful model for the study of TOC and may provide insights into the natural pathogenesis of this disease. It is remarkable that every mortality after 7 d PI had osteomyelitis in multiple sites. Recently, Droual et al. (1996) reported the association of an apparently enteric-origin E. coli infection and TOC lesions, and suggested that the more typical respiratory E. coli infection may not be involved in the pathogenesis of TOC. Previous studies of TOC have reported an

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre 7d</th>
<th>Pre 7d</th>
<th>Pre 7d</th>
<th>Pre 7d</th>
</tr>
</thead>
<tbody>
<tr>
<td>HET</td>
<td>48 ± 4</td>
<td>39 ± 3</td>
<td>1.3 ± 0.2</td>
<td>1.3 ± 0.4</td>
</tr>
<tr>
<td>LYM</td>
<td>35 ± 1</td>
<td>51 ± 1</td>
<td>0.7 ± 0.0</td>
<td>1.7 ± 0.6</td>
</tr>
<tr>
<td>H:L</td>
<td>40 ± 8</td>
<td>43 ± 8</td>
<td>12 ± 3</td>
<td>12 ± 3</td>
</tr>
<tr>
<td>MONO</td>
<td>49 ± 1</td>
<td>48 ± 1</td>
<td>12 ± 3</td>
<td>12 ± 2</td>
</tr>
</tbody>
</table>

a-bMeans within a column with no common superscript differ significantly from control (P ≤ 0.05).

Control represents unchallenged Strain B turkeys.
P = 0.1.
P ≤ 0.08.

FIGURE 4. Impression smears of the proximal tibia of mortalities starting 2 d postinfection showed evidence of bacterial infection in bones not grossly affected with osteomyelitis.
association between pericarditis and green liver discoloration (Tilley and Barnes, 1990) and between pericarditis and increased incidence of TOC (Bayyari et al., 1994). The severe airsacculitis and pericarditis seen in this challenge study were associated with all of the typical lesions described by the FSIS as defining TOC, including osteomyelitis, synovitis, soft tissue abscesses, and green liver, which suggests that respiratory *E. coli* infections and a subsequent septicemia may also be involved in the pathogenesis of TOC.

### REFERENCES


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**TABLE 6.** Differential white blood cell counts* for turkeys before (Pre) and 7 d after air sac inoculation with *Escherichia coli*

<table>
<thead>
<tr>
<th>Strain</th>
<th>HET Pre</th>
<th>HET 7 d</th>
<th>LYM Pre</th>
<th>LYM 7 d</th>
<th>MONO Pre</th>
<th>MONO 7 d</th>
<th>TOTAL Pre</th>
<th>TOTAL 7 d</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>87 ± 6</td>
<td>205 ± 53</td>
<td>71 ± 5</td>
<td>98 ± 5</td>
<td>8 ± 2</td>
<td>30 ± 9</td>
<td>183 ± 8</td>
<td>346 ± 61</td>
</tr>
<tr>
<td>B</td>
<td>108 ± 10</td>
<td>141 ± 36</td>
<td>97 ± 9</td>
<td>120 ± 9</td>
<td>28 ± 4</td>
<td>18 ± 5</td>
<td>240 ± 20</td>
<td>345 ± 44</td>
</tr>
<tr>
<td></td>
<td>0.09</td>
<td>0.7</td>
<td>0.02</td>
<td>0.04</td>
<td>0.03</td>
<td>0.2</td>
<td>0.01</td>
<td>0.8</td>
</tr>
</tbody>
</table>

*Data represent the mean and SE of the numbers of each cell type counted on 50 fields of a whole blood monolayer using 1,000× magnification.

*LYM = lymphocyte; HET = heterophil; MONO = monocyte; TOTAL includes lymphocytes, heterophils, monocytes, eosinophils, and basophils.


