Association between gizzard lesions and increased caecal Clostridium perfringens counts in broiler chickens

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Available online: 18 Jan 2007
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The aim of this study was to investigate the relationship between mucosal gizzard lesions and caecal *Clostridium perfringens* counts. Gross pathological changes in the gizzard and small intestine, and caecal *C. perfringens* counts from 1217 meat-type chickens were recorded during the course of six experiments and were statistically analysed. *C. perfringens* counts increased significantly (*P* < 0.001) with the severity of mucosal gizzard lesions. Mucosal gizzard lesions were more prevalent than necrotic enteritis. Correcting for the pen and necrotic enteritis within experiment, mucosal gizzard lesions explained 31.8% of the variation in *C. perfringens* counts. Mucosal gizzard lesions and age together explained 59.1% of the variation in *C. perfringens* counts. The mean ages of birds with moderate and severe mucosal gizzard lesions were 1.7 and 0.8 days lower than the mean age of birds with necrotic enteritis, respectively. The association between mucosal gizzard lesions and high *C. perfringens* counts might be of importance when attempting to improve production efficiency, health and the welfare of the chickens.

**Introduction**

A condition designated “gizzard erosions” and/or “gizzard ulceration” (GEU) has been reported in commercial broiler chickens in connection with avian adenovirus infection (Ono *et al.*, 2001, 2003a,b), mycotoxin-contaminated feed (Hedman *et al.*, 1995; Hoerr, 2003), vitamin B₆ deficiency (Daghir & Haddad, 1981), suboptimal levels of vitamin E (Janssen & Germs, 1973), inadequate levels of sulphur-containing dietary amino acids (Miller *et al.*, 1975), high levels of dietary copper (Poupoulis & Jensen, 1976), pelleted feed (Ross, 1979), as well as inclusion of certain fish meals in the diets and the consequent presence of histamine and gizzerosine (Harry & Hurst, 1976; Okazaki *et al.*, 1983; Sugahara *et al.*, 1988; Sharma & Pandey, 1990; Tisljar *et al.*, 2002). The koilin layer of gizzards from affected birds is fissured, thickened, spongy and discoloured (Fossum *et al.*, 1988).

*Clostridium perfringens* type A and type C are known to cause necrotic enteritis (NE) in chickens (Parish, 1961; Helmboldt & Bryant, 1971; Long *et al.*, 1974; El-seedy, 1990; Wages & Opgengart, 2003). In the Nordic European countries it is *C. perfringens* type A that has been isolated from the intestine of both healthy and diseased chickens (Hofshagen & Stenwig, 1992; Naurby *et al.*, 2003; Engström *et al.*, 2003).

Lesions in the koilin layer and the mucosa of the gizzard have been observed in birds, in randomly sampled individuals without other significant gross lesions (Stuve *et al.*, 1992). One of the authors of the present paper has been involved in several studies on factors that may affect the risk of *C. perfringens*-associated NE in broiler chickens. During these studies, GEU lesions were frequently observed and recorded (M. Kaldhusdal, unpublished observations). Also, in some cases, gizzards with such lesions have been examined for *C. perfringens* with positive results (unpublished data). Furthermore, *C. perfringens* was detected in the koilin layer of gizzards presenting erosions, but it was supposed that this was a secondary infection as a consequence of koilin layer damage induced by other factors (Fossum *et al.*, 1988). GEU has not previously been associated with *C. perfringens* counts in the intestinal tract and/or *C. perfringens*-associated NE.

The aim of this study was to investigate the relationship between GEU and caecal *C. perfringens* counts.

**Materials and Methods**

**Data collection.** This was an analytical retrospective study of data collected from six experimental studies that were designed primarily to study the association between *C. perfringens*-associated NE and various immunological, dietary and environmental factors (Table 1).

- Experiment 1 included eight pens with 146 chickens in each pen. Three chickens from each pen at three different ages were selected at random (Table 2). A total of 72 chickens were included in the study for bacteriological and pathological examination.
- Experiment 2 included 12 pens with 144 chickens in each. At five different ages, three chickens were picked randomly from each pen for examination (Table 2). Since data from two chickens are missing, results from 178 chickens were included.
- Experiment 3 included 12 pens with 32 chickens in each pen. On two successive days, at six different ages, five chickens were randomly picked for examination (Table 2). Results from 357 chickens were included in this experiment since data from three were missing.

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Table 1. Summary of the six experiments

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Number of pens</th>
<th>Number of chickens at start</th>
<th>Number of chickens at end</th>
<th>Total number of chickens</th>
<th>Diet</th>
<th>Antimicrobial feed additives</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
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<td>72</td>
<td>8</td>
<td>1168</td>
<td>Broilers</td>
<td>None</td>
<td>Amprolium/C27</td>
</tr>
<tr>
<td>2</td>
<td>12</td>
<td>178</td>
<td>5</td>
<td>1728</td>
<td>Broilers</td>
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<td>None</td>
</tr>
<tr>
<td>3</td>
<td>12</td>
<td>357</td>
<td>12</td>
<td>384</td>
<td>Broilers</td>
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<td>None</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
<td>95</td>
<td>4</td>
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<td>Broilers</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>5</td>
<td>12</td>
<td>215</td>
<td>5</td>
<td>1728</td>
<td>Broilers</td>
<td>None</td>
<td>Amprolium/C27</td>
</tr>
<tr>
<td>6</td>
<td>12</td>
<td>144</td>
<td>12</td>
<td>2152</td>
<td>Broilers</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

- Experiment 4 included all chickens kept in one large pen. At four different ages, 24 chickens were picked randomly for examination (Table 2). Data from 95 chickens are included in this experiment (data from one bird was missing).
- Experiment 5 included 12 pens with 144 chickens in each. At five different ages, four chickens from each pen were picked at random for examination (Table 2). Since results from 25 chickens were missing, 215 chickens were included in this experiment.
- Experiment 6 included 12 pens with 144 chickens in each pen. At five different ages, five chickens were randomly picked from each pen for examination (Table 2). A total of 300 were included in this experiment.

Animals, housing and diets. All experiments were started with 1-day-old Ross 208 meat-type chickens supplied by a commercial hatchery. No antibiotic growth promoters or ionophorous anticoccidials were added to diets used in these experiments.

Experiments 1, 2, 5 and 6 were based on a NE disease model developed at The National Veterinary Institute in Oslo (M. Kaldhusdal, unpublished data) to induce C. perfringens-associated enteritis and hepatitis. The main characteristics of this model include the use of a putatively predisposing feeding regime, high stocking density, housing of birds on litter in pens, and spontaneous C. perfringens infection of the birds.

Experiment 3 was conducted in floor pens. These birds were offered a wheat and soybean-based diet supplemented with various ingredients assumed to promote intestinal C. perfringens proliferation, and which differed from the diet used in Experiments in 1, 2, 5 and 6. The stocking density started at 27.3 birds/m² and was reduced as the birds were removed for sampling and examination, until there were no birds left in the pens.

Experiment 4 was conducted in a commercial broiler house managed in accordance with general practice and recommendations from the hatchery and the poultry adviser employed by the processing plant. The feed was produced by a commercial feed mill, using its current formulation of feed for broilers, but without anticoccidial or anti-bacterial additives. The stocking density started at 29.3 birds/m² and was reduced to approximately 27.3 birds/m² after removal of birds for sampling and examination.

Bacteriology and pathology. The birds were stunned followed by cervical dislocation, and opened immediately. The caecal contents were collected for bacteriology from a subset of birds (1217 individuals) that were examined for organ lesions. In pen experiments, the same number of birds was examined for C. perfringens in each pen. Caeal contents (0.5 to 5.0 g) were weighed and serially diluted in 0.9% saline, assuming a specific weight of caecal material of 1.0 kg/dm³, and 0.1 ml each sample was plated on 5% blood agar (CM 271; Oxoid). The plates were incubated for 1 to 2 days in an anaerobic atmosphere (AN 0025 A; Oxoid) at 37°C. C. perfringens appeared as large (3 to 5 mm) irregular colonies with a large double zone of haemolysis. The figures from the bacterial counts were recorded as colony forming units.

The gizzard and small intestine were examined to record pathological changes in the mucosa. Gross gizzard lesions were scored as follows (Figure 1):

- None: no lesions.
- Moderate: focal lesions in the koilin layer (cuticle) without pronounced presence of spongy material.
- Severe: spongy material within the gizzard cuticle.

The gross appearance of spongy material is illustrated in Figure 1c. Gross lesions of the small intestine were scored as follows:

- No NE lesions present: no mucosal ulcers or pseudomembranes were seen.
- NE lesions present: at least one mucosal ulcer or pseudomembrane was seen.

An ulcer was defined as a grossly visible lesion characterized by a distinctly delineated depression of the mucosal surface, indicating loss
of stromal tissue. A pseudomembrane was defined as a gross lesion characterized by adherence of discoloured, amorphous material to the mucosal surface. Only lesions of at least 1 mm were recorded as ulcers or pseudomembranes. Pseudomembranes were spatially associated with ulcers, or appeared separately. Pseudomembranes (Kaldhusdal & Hofshagen, 1992) and ulcers (unpublished data; Løvland et al., 2004) are associated with high counts of intestinal \textit{C. perfringens}, and are considered specific for \textit{C. perfringens}-associated NE. Both types of lesions have previously been used as indicators of NE in a vaccine study (Lovland et al., 2004).

The distribution of NE lesions between intestinal regions was studied in a total of 267 birds from the six experiments, including birds that were not examined for \textit{C. perfringens} (\textit{C. perfringens} counts were available from 130 of these birds).

### Parasitology.

In each experiment, birds with and without NE lesions (a total of 281 individuals) were examined for intestinal coccidia. Smears were prepared from scrapings of the intestinal mucosa (from lesions if present), and examined for oocysts and schizonts by light microscopy.

### Mortality.

The number of birds that died or were killed was recorded each day in Experiments 1, 2, 4, 5 and 6. The accumulated mortality rate at the end of each trial was calculated as a percentage of the birds at the start.

### Statistical analysis.

All the assumed continuously distributed variables are expressed as mean values with 95% confidence intervals or standard deviations. The confidence intervals were constructed using the Student procedure (Kleinbaum, 2003). Categorized variables are expressed in contingency tables (Agresti, 1990).

Comparison of groups with regard to assumed continuously distributed variables was performed using analysis of covariance (Kleinbaum, 2003). In order to study the associations between the dependent variable \textit{C. perfringens} and the independent variables GEU and age, multiple regression analysis nested for the pen and for NE within experiment was used (Kleinbaum, 2003).

Contingency table analysis was used for analysis of the relationship between GEU and NE (Agresti, 1990). SAS software was used to perform the analyses.

### Table 2. Distribution of age groups within each experiment

<table>
<thead>
<tr>
<th>Age (days)</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>Total number of chickens</th>
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<tbody>
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</tr>
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<td>17</td>
<td>0</td>
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<td>30</td>
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<td>30</td>
</tr>
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<td>0</td>
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<td>96</td>
</tr>
<tr>
<td>33</td>
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<td>0</td>
<td>48</td>
<td>0</td>
<td>0</td>
<td>48</td>
</tr>
<tr>
<td>35</td>
<td>24</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>24</td>
</tr>
<tr>
<td>Total</td>
<td>72</td>
<td>178</td>
<td>357</td>
<td>95</td>
<td>215</td>
<td>300</td>
<td>1217</td>
</tr>
</tbody>
</table>

### Figure 1.

Open gizzards. The smooth inner surface of a healthy gizzard (A) can be compared with the focal, superficial lesions (*) appearing in a moderately affected gizzard (B). In a severely affected gizzard (C), the lesions appear as extensive spongy material (s) and dilated isthmus (i).
Table 3. Comparison of degree of pathological changes observed in the gizzard and small intestine related to C. perfringens count and age

<table>
<thead>
<tr>
<th>Pathological change location</th>
<th>Degree of change</th>
<th>C. perfringens count (log_{10} colony-forming units/g)</th>
<th>Age (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gizzard</td>
<td>None (n = 196)</td>
<td>4.05 (2.15), 3.75–4.36</td>
<td>20.2 (7.3), 19.1–21.2</td>
</tr>
<tr>
<td></td>
<td>Moderate (n = 564)</td>
<td>5.27 (2.50), 5.06–5.47</td>
<td>23.7 (5.6), 23.2–24.1</td>
</tr>
<tr>
<td></td>
<td>Severe (n = 457)</td>
<td>6.92 (1.78), 6.76–7.09</td>
<td>24.6 (5.2), 24.1–25.0</td>
</tr>
<tr>
<td>Small intestine</td>
<td>None (n = 1087)</td>
<td>5.38 (2.35), 5.23–5.52</td>
<td>23.2 (6.1), 22.8–23.6</td>
</tr>
<tr>
<td></td>
<td>Moderate/severe (n = 130)</td>
<td>8.30 (1.22), 8.09–8.51</td>
<td>25.4 (4.4), 24.7–26.2</td>
</tr>
</tbody>
</table>

Data presented as mean value (standard deviation), 95% confidence interval.

Results

C. perfringens counts were found to increase significantly (P < 0.001) with increasing severity of GEU and the appearance of NE. Additionally, the severity of GEU and the frequency of NE increased significantly (P < 0.001) with the age of the chickens. GEU lesions were distinctly more prevalent than NE at 10 days, the earliest NE case was detected on day 16.

In the group without NE lesions in the small intestine, C. perfringens increased significantly (P < 0.001) with the severity of the changes in the gizzard. Among birds with NE, C. perfringens was neither significantly different (P = 0.06) nor did it consistently increase with the severity of the pathological changes in the gizzard (Table 4).

Correcting for the pen within the experiment, the pathological changes in the gizzard explain 27.1% of the variation in C. perfringens, whereas NE explains 21.6%. When corrected for both the pen and NE within experiments, gizzard lesions were found to explain 31.8% of the variation in C. perfringens counts. C. perfringens was found to increase significantly (P < 0.001) with age (Figure 3). However, the relationship between the two variables was not found to be linear. The regression between the two variables was estimated to be given by the equation: C. perfringens = 2.19 + 0.17 age–0.011 (age–23.4)^2–0.00037 (age–23.4)^3. Corrected for the pen and changes in the small intestine within experiments, age was found to explain 17.9% of the variation in C. perfringens.

Changes in the gizzard and age corrected for changes in the small intestine and the pen within the experiment were found to explain 51.3% of the variation in C. perfringens.

The prevalence of NE was not found to increase consistently with increasing degree of GEU (Table 5).

A dilated isthmus (Figure 1e) and proventriculus was found in some of the birds with the most severe gizzard lesions. Forty-three per cent of the birds with NE presented NE lesions in the duodenum only, 40% in the duodenum and jejunum simultaneously, and 17% in the jejunum only. No NE lesions were observed in the ileum, caecum and colorectum.

Coccidial oocysts were not detected in any intestinal smear. No schizonts were found in 278 of 281 (98.9%) smears examined. One or a few structures that were similar to schizonts, but not positively identified as such, were detected in three (1.1%) smears.

Mortality rates for Experiments 1, 2, 4, 5 and 6 were 4.4%, 2.6%, 2.1%, 2.1% and 2.3%, respectively. Significant occurrence of clinical disease was not detected in any of the experiments, and the mortality in Experiment 3 was also low.

Discussion

The main finding in this study is the association between gizzard lesions and intestinal C. perfringens counts. The association between GEU and C. perfringens counts was present among birds without NE, indicating that the association is not due to confounding with NE. GEU seemed to appear earlier than the NE lesions.

A significant association between GEU and C. perfringens counts was only present among birds without NE. The main reason is that the C. perfringens

Figure 2. Percentage of chickens presenting lesions in the gizzard and necrotic enteritis as a function of age, with 95% confidence interval.
Table 4. Comparison of degree of pathological changes observed in the gizzard, with and without lesions in the small intestine, related to C. perfringens counts

<table>
<thead>
<tr>
<th>Lesions in the small intestine</th>
<th>Degree of changes in the gizzard</th>
<th>C. perfringens (log_{10} colony-forming units/g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>None (n = 18)</td>
<td>7.88 (1.71), 7.03–8.73</td>
</tr>
<tr>
<td></td>
<td>Moderate (n = 89)</td>
<td>8.47 (1.07), 8.25–8.70</td>
</tr>
<tr>
<td></td>
<td>Severe (n = 23)</td>
<td>7.96 (1.23), 7.43–8.50</td>
</tr>
<tr>
<td>No</td>
<td>None (n = 178)</td>
<td>3.67 (1.78), 3.40–3.92</td>
</tr>
<tr>
<td></td>
<td>Moderate (n = 475)</td>
<td>4.67 (2.22), 4.47–4.87</td>
</tr>
<tr>
<td></td>
<td>Severe (n = 430)</td>
<td>6.87 (1.79), 6.70–7.03</td>
</tr>
</tbody>
</table>

Data presented as mean value (standard deviation), 95% confidence interval.

counts of almost all birds with NE were very high, whether GEU was present or not. This association between NE and C. perfringens counts is in accordance with previous findings (Kaldhusdal & Hofshagen, 1992; Lovland et al., 2004). Furthermore, our results show that NE may be found also in birds without GEU. This finding indicates that the presence of GEU in an individual is not a prerequisite for the presence of NE in that individual.

Moderate and severe GEU were detected in birds that were on average 1.7 and 0.8 days younger than those with NE, respectively. The first case of NE was detected 6 days later than the first cases of GEU, which were present already on the first sampling day. These findings are in accordance with an assumption that GEU is a risk factor for NE. However, we found that although GEU was associated with increased C. perfringens counts, the presence of GEU in a bird did not affect the likelihood of concurrent NE in the same bird. Our study did not collect data on the GEU and NE frequencies among birds younger than 10 days of age. We were therefore unable to evaluate the GEU and NE occurrence during the entire rearing period.

GEU explains more of the variation in C. perfringens than NE, correcting for the effect of the pen within the experiment. This finding is due to the fact that the number of birds with GEU lesions was substantially higher than the number of birds with NE. It was decided to correct for NE and the pen within the experiment, because of the correlation existing between C. perfringens and NE, in order to eliminate the effect from these two variables—resulting in GEU explaining 31.8% of the variation of C. perfringens. As demonstrated, age and GEU are also correlated but it was not possible to correct for age in our analysis because the dataset was not large enough. Instead it was decided to add the effect of age to the model. GEU and age explained together 51.3% of the variation of C. perfringens.

C. perfringens increased with age, reaching its maximum at 27 to 29 days of age, when it stabilized and even decreased. This development has been observed in other studies (Lovland et al., 2004).

None of the birds in these experiments had access to antibiotics, neither for therapeutic use nor as growth promoters. In four of the experiments the anticoccidial Amprol+ was added to the feed instead of the ionophore narasin in order to avoid the antibacterial effect of ionophores (Berg & Hamill, 1978; Brennan et al., 2001). It is assumed that Amprol+ does not suppress C. perfringens proliferation, and the high concentrations of C. perfringens observed in this study verify this assumption. In the other two experiments, the rations did not contain any antimicrobial feed additives. Further work has been started to compare the GEU frequencies in broiler flocks offered feed with and without ionophores.

The cause(s) of GEU in our material remain unknown. GEU has also been found recently in commercial broiler flocks in Norway (unpublished data). Previous studies have linked GEU occurrence with several nutritional and infectious factors (Poupoulis & Jensen, 1976; Okazaki et al., 1983; Fossum et al., 1988; Hedman et al., 1995; Kaya et al., 2002; Hetland et al., 2003; Ono et al., 2003a). C. perfringens has been isolated from chickens with gizzard lesions, but the aetiological role of C. perfringens in this condition is uncertain. Further work is required to determine the cause of GEU in Norway. It would be useful to examine affected gizzards histologically to see whether C. perfringens organisms are found in association with the lesions. Furthermore, it would be useful to examine C. perfringens isolates by a molecular technique such as pulsed

Figure 3. Logarithmic transformed concentration of C. perfringens expressed as a function of age with 95% confidence interval.

<table>
<thead>
<tr>
<th>Gizzard score</th>
<th>Experiment 1</th>
<th>Experiment 2</th>
<th>Experiment 3</th>
<th>Experiment 4</th>
<th>Experiment 5</th>
<th>Experiment 6</th>
<th>All experiments</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0/0 (–)</td>
<td>1/6 (17)</td>
<td>0/105 (&lt;1)</td>
<td>14/66 (21)</td>
<td>3/10 (30)</td>
<td>0/9 (&lt;1)</td>
<td>18/196 (9)</td>
</tr>
<tr>
<td>1</td>
<td>9/39 (23)</td>
<td>53/145 (37)</td>
<td>0/122 (&lt;1)</td>
<td>2/22 (9)</td>
<td>24/182 (13)</td>
<td>1/54 (2)</td>
<td>89/564 (16)</td>
</tr>
<tr>
<td>2</td>
<td>7/33 (21)</td>
<td>5/27 (19)</td>
<td>2/130 (2)</td>
<td>1/7 (14)</td>
<td>5/23 (22)</td>
<td>3/237 (1)</td>
<td>23/457 (5)</td>
</tr>
</tbody>
</table>

Data presented as the number of animals presenting subclinical NE/total number of animals examined per lesion category and experiment (%).
field gel electrophoresis to investigate relationships and compare isolates between diseased and healthy birds. If the findings of this study are confirmed by future investigations, they will have significant implications regarding efforts to mitigate C. perfringens-associated health and productivity problems in chickens. In that case, preventive measures against gizzard erosion and ulceration would become an integrated part of such efforts.

Acknowledgements

The authors are grateful to all the people involved in sampling and attending the chickens. They also wish to thank the staff from the Section of Microbiology at the Norwegian School of Veterinary Science, Kerstin Nord-Olsson and other staff from the Section of Pathology at the National Veterinary Institute in Oslo involved in the bacteriologic examinations, and staff from the Section of Veterinary Microbiology at the Norwegian School of Veterinary Science, Kerstin Nord-Olsson, for invaluable contributions in the studies. This study was funded by grants from the Norwegian Research Council, project numbers 136326/110, 133447/110, 137443/110 and 155867/110.

References


Non-English Abstracts

Association between gizzard lesions and increased caecal Clostridium perfringens counts in broiler chickens

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Le but de cette étude a été d’investiguer la relation entre les lésions de la muqueuse du gésier et le nombre de Clostridium perfringens (CP) dans les cæca. Les lésions macroscopiques pathologiques au niveau du gésier et de l’intestin grêle ainsi que le nombre des CP dans les cæca de 1217 poulets de chair ont été enregistrés au cours de six expérimentations et ont été analysés statistiquement. Le nombre de CP a augmenté significativement (p < 0,001) avec la gravité des lésions de la muqueuse du gésier. Ces dernières étaient plus prévalentes que l’entérite nécrotique. Après correction des résultats de chaque expérience pour prendre en compte les effets “parquets” et “entérite nécrotique”, les lésions de la muqueuse du gésier expliquaient 31,8% de la variation du nombre de CP. Les lésions de la muqueuse du gésier et l’âge expliquaient, ensemble, 59,1% de la variation du nombre de CP. Les moyennes d’âge des animaux présentant des lésions modérées et sévères de la muqueuse du gésier étaient respectivement de 1,7 et 0,8 jours inférieures à celles des animaux présentant une entérite nécrotique. L’association entre les lésions de la muqueuse du gésier et le nombre élevé de CP peut être important lors d’essai d’amélioration de la productivité, de la santé et du bien être des poulets.

Ziel dieser Studie war es, die Beziehung zwischen Schleimhautläsionen im Drüsenmagen und der Anzahl von Clostridium perfringens (CP) im Zäkum zu untersuchen. Im Verlauf von sechs Experimenten wurden bei 1217 Hühnern des Masttyps die pathologisch-anatomischen Befunde in Drüsenmagen und Dünndarm sowie die zäkale CP-Anzahl ermittelt. Die CP-Anzahlen stiegen mit zunehmender Schwere der mukosalen Drüsenmagenläsionen signifikant an (p < 0,001). Diese Schleimhautläsionen im Dünndarm traten häufiger auf als eine nekrotisierende Enteritis. Korrigiert um die Faktoren Haltung und nekrotisierende Enteritis erklärten die Schleimhautveränderungen im Dünndarm 31,8% der Variationen bei den CP-Zahlen. Schleimhautläsionen und Alter zusammengenommen standen mit 59,1% der CP-Zahlerhöhung im Zusammenhang. Die Tiere mit mittel- bis hochgradigen Schleimhautläsionen im Dünndarm waren 1,7 bzw. 0,8 Tage jünger als die Broiler mit nekrotisierender Enteritis. Die Verbindung zwischen mukosalen Drüsenmagenveränderungen und hohen CP-Zahlen kann wichtig sein bei dem Versuch die Produktionssefektivität, die Gesundheit und den Tierschutz bei Hühnern zu verbessern.

El objetivo de este trabajo fue estudiar la relación entre las lesiones en la mucosa de la molleja y los recuentos de Clostridium perfringens (CP) en el ciego. Se registraron y se analizaron estadisticamente los cambios macroscópicos en la molleja e intestino delgado, y los recuentos de CP en el ciego de 1217 pollos de engorde durante el transcurso de seis pruebas experimentales. Los recuentos de CP aumentaron significativamente (p < 0,001) con la gravedad de las lesiones en la mucosa de la molleja. Las lesiones en la mucosa de la molleja tuvieron una mayor prevalencia que la enteritis necrótica. Las lesiones en la mucosa de la molleja explicaron el 31.8% de la variación en los recuentos de CP cuando se corrigieron los efectos corral y enteritis necrótica en la prueba. Las lesiones en la mucosa de la molleja y la edad explicaron el 59.1% de la variación de los recuentos de CP. La media de edad de las aves con lesiones moderadas y graves en la mucosa de la molleja fue 1.7 y 0.8 días menor, respectivamente, que la media de edad de las aves con enteritis necrótica. La relación entre las lesiones en la mucosa de la molleja y los recuentos altos de CP podría ser importante cuando se intenta mejorar la productividad, salud y bienestar de los pollos.

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ISSN 0307-9457 (print)/ISSN 1465-3338 (online)/S0001-01 © 2006 Houghton Trust Ltd
DOI: 10.1080/03079450600924150