DIET-INDUCED CHANGES IN INTESTINAL VISCOSITY AND RESPONSE TO LOW DOSE EIMERIA MAXIMA INFECTION IN BROILER CHICKENS

C. W. WONG$^1$, J. DUAN$^2$, M. CHOCT$^1$ and S. W. WALKDEN-BROWN$^1$

Summary

This study investigated whether diet-induced changes in intestinal viscosity could influence the response of broiler chickens to Eimeria maxima challenge at a single low dose (50 oocysts/bird). Immediately before the challenge, birds fed a low-ME wheat diet showed a higher level of intestinal viscosity than those fed either a control maize diet or a xylanase-supplemented low-ME wheat diet. The birds fed the low-ME wheat diet also had a significantly lower total faecal oocyst output than those fed the control maize and xylanase-supplemented low-ME wheat diets that elicited relatively lower intestinal viscosity. The results suggest that intestinal viscosity may influence the host resistance to low-dose Eimeria infection in broilers. The possible impact of this on the efficacy of low-dose live vaccines should not be overlooked.

I. INTRODUCTION

The interaction between specific dietary nutrients and the pathogenicity of coccidiosis has been widely studied in chickens, but little is known about the role of gut environment in the development of coccidiosis. Although reduced intestinal viscosity has been shown to improve nutrient utilisation and growth performance in both healthy broilers and broilers affected by coccidiosis (Bedford and Classen, 1992; Morgan and Bedford, 1995), its direct impact on the pathogenicity of coccidiosis is unclear. With growing interest in using live Eimeria oocysts for “low-dose” vaccination (Joyner and Norton, 1976; Pierson et al., 1997; Williams, 1998; Danforth, 1998), it is important to determine whether intestinal viscosity affects the response of birds to a low-dose Eimeria infection. In this study, we tested the hypothesis that diet-induced change in intestinal viscosity can affect the host response to a low-dose infection with E. maxima in broilers.

II. MATERIALS AND METHODS

(a) Chickens and dietary treatments

A total of ninety Cobb broilers (Baiada Poultry Pty Ltd, Australia), 25 days of age, were divided into three treatment groups (I, II, III). Each treatment group had six replicates of five birds. Treatment I received a control maize-based diet during the whole experiment. Treatment II and III received a low-metabolisable energy (ME) wheat-based diet without and with a xylanase (Biofeed Wheat) at 250g per kg of diet respectively during the pre-infection and pre-patent period of coccidiosis followed by the control maize-based diet for the patent period.

$^1$ Animal Science, School of Rural Science and Natural Resources, University of New England, Armidale NSW 2351.

$^2$ Beijing Agricultural College, Beijing 102206, P.R. China.
(b) Coccidial infection and oocyst output

_E. maxima_ oocysts used in this study were kindly supplied by Dr W.K. Jorgensen, Animal Research Institute, DPI, Queensland. Birds, at day 29 of age (four days after being on experimental diets), were each dosed orally with 50 oocysts in 1ml saline. Daily total faecal outputs were collected between day 6 and 12 post-infection and total oocyst output determined.

(c) Collection of intestinal digesta for viscosity test

At day 29 day of age (immediately before _Eimeria_ infection), 5 birds per diet group were euthanased by intraperitoneal injections of pentobarbitone sodium and the contents of duodenum, jejunum and ileum collected and centrifuged at 12,000g for 15 min. The supernatant was stored at -20 °C until viscosity was measured using a Brookfield viscometer at 25°C with a CP40 cone and shear rate of 5-500 s⁻¹.

(d) Growth performance measurement

Bird weights and feed intake were recorded during the pre-infection, pre-patent and patent periods. Liveweight gain and feed conversion rate (FCR) were calculated.

(e) Statistical analysis

The data were subjected to two-way and one-way analysis of variance and multiple comparisons using Student-Newman-Keuls method. All values are expressed as mean ± SEM.

III RESULTS

Regardless of diet treatments the pre-patent and patent periods had higher levels of feed intake and FCR than the pre-infection period (P<0.05). For Treatment II, the pre-patent period had a significantly higher daily weight gain than the pre-infection period (P<0.05). During the pre-infection period, Treatment III had a significantly higher (P<0.05) daily liveweight gain than Treatment II, but the latter had a significantly higher (P<0.05) FCR than both Treatments I and III (Table 1).

Figure 1 shows that birds on Treatment II (low-ME wheat diet) had higher jejunal and ileal viscosity than those on Treatments I (control maize) and III (low-ME wheat diet with xylanase) (P<0.05). Duodenal viscosity was found to be higher in the birds on Treatment II than those on Treatment I (P<0.05). Birds on Treatment III also tended towards (P=0.054) a lower level of duodenal viscosity than those on Treatment II. In response to _Eimeria_ infection, birds on Treatment II had a significantly lower (P<0.05) total faecal oocyst output than those on Treatment I (Figure 2). Birds on Treatment II also tended towards (P=0.054) a lower level of faecal oocyst output than those on Treatment III.
Table 1. Effects of diets on liveweight gain, feed intake and FCR of broilers.

<table>
<thead>
<tr>
<th>Period (Day)</th>
<th>Treatment (Diet used)</th>
<th>Liveweight gain (g/bird/day)</th>
<th>Feed intake (g/bird/day)</th>
<th>FCR (g/g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-infection</td>
<td>I (Maize control)</td>
<td>56.2 ± 1.4&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>108.8 ± 1.1</td>
<td>1.94 ± 0.06&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>II (Low-ME wheat)</td>
<td>47.1 ± 5.0&lt;sup&gt;a&lt;/sup&gt;</td>
<td>105.1 ± 9.4</td>
<td>2.28 ± 0.11&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>III (Low-ME wheat + xylanase)</td>
<td>66.5 ± 2.8&lt;sup&gt;b&lt;/sup&gt;</td>
<td>108.6 ± 3.2</td>
<td>1.64 ± 0.03&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Pre-patent</td>
<td>I (Maize control)</td>
<td>55.4 ± 4.8</td>
<td>166.7 ± 8.0</td>
<td>3.23 ± 0.51</td>
</tr>
<tr>
<td></td>
<td>II (Low-ME wheat)</td>
<td>69.6 ± 3.7</td>
<td>183.2 ± 9.9</td>
<td>2.70 ± 0.27</td>
</tr>
<tr>
<td></td>
<td>III (Low-ME wheat + xylanase)</td>
<td>63.2 ± 5.9</td>
<td>173.6 ± 1.4</td>
<td>2.95 ± 0.42</td>
</tr>
<tr>
<td>Patent</td>
<td>I (Maize control)</td>
<td>63.5 ± 5.4</td>
<td>177.8 ± 5.4</td>
<td>2.86 ± 0.15</td>
</tr>
<tr>
<td></td>
<td>II (Maize control)</td>
<td>59.4 ± 2.5</td>
<td>173.1 ± 3.7</td>
<td>2.93 ± 0.11</td>
</tr>
<tr>
<td></td>
<td>III (Maize control)</td>
<td>60.1 ± 7.7</td>
<td>181.8 ± 2.2</td>
<td>3.27 ± 0.42</td>
</tr>
</tbody>
</table>

<sup>ab</sup> Values with different superscripts differ significantly within the same column and period at P<0.05. <sup>1</sup> For effect of period, refer to the text.

Figure 1. Effect of diet on the viscosity of digesta collected from various intestinal sections in birds on day 29 immediately prior to E. maxima infection. <sup>ab</sup>Values with different superscripts differ significantly within the same intestinal section at P<0.05

Figure 2. Effect of treatment on the total faecal oocyst output between day 6 and 12 post-infection with E. maxima. <sup>ab</sup>Values with different superscripts differ significantly at P<0.05

IV. DISCUSSION

In response to a low-dose infection with E. maxima, broilers fed a low-ME wheat-based diet that led to a higher level of intestinal viscosity during the pre-infection period showed a markedly reduced level of faecal oocyst output, compared to those fed the other diets with lower intestinal viscosity. We speculate that the high intestinal viscosity might physically prevent sporozoites from invading the gut wall and hence reduce their infectivity. This notion is supported by a recent study showing that increasing dietary fibre level reduced total faecal oocyst output in chickens infected with a high-dose of more pathogenic Eimeria
strains (Muir and Bryden, 1998). However, we cannot exclude effects due to differences in the nutritive value of the experimental diets. This might also influence resistance to *E. maxima* during its life cycle within the gut tissue. However, since there was no difference in the growth performance among the three treatment groups during the pre-patent and patent periods, the impact of differences in the nutritive values, if any, between the experimental diets appears to be minimal.

The supplementation of low-ME wheat diet with xylanase was found to significantly improve weight gain and feed conversion of the birds prior to *E. maxima* infection, which was consistent with previous findings (Choct, 1998). The reason why this difference did not continue into the pre-patent period is not clear. Dosing birds with a low number of *Eimeria* oocysts should not have any significant impact on their growth performance (Conway et al., 1993; Richard, 1998). However, coccidiosis itself may reduce intestinal viscosity in broilers (Morgan and Bedford, 1995). Whether *E. maxima* had any impact on the intestinal viscosity in the birds fed the low-ME wheat-based diet and consequently modulated nutrient utilisation, which cancelled out pre-infection differences in the growth performance, remains to be solved. In this regard, our data showing that the birds fed the low-ME wheat diet had an improved liveweight gain following the infection in the pre-patent period, has supported the above explanation.

In conclusion, our findings support the hypothesis that diet-induced change in intestinal viscosity can affect the host response to a low-dose *E. maxima* infection in broilers. The *E. maxima* dose used in this study was similar to those commonly used for low-dose vaccination. Whether the current finding of variation in oocyst output is due to differences in digesta viscosity and/or differences in host immunity remains to be confirmed. As the use of low-dose live vaccines has become an important control measure for coccidiosis in chickens, we should not overlook the possible role of diet formulation and feed enzymes in modulating their efficacy.

REFERENCES


