Gut Stasis in Chickens Infected with *Eimeria*[1]

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ABSTRACT The duration and locations of gut stasis were examined in chickens infected with either *Eimeria acervulina* or *E. maxima*. Gut passage time (GPT) was used to determine gut stasis. The location of feed retention was determined qualitatively and quantitatively. Infections with both species were associated with increased GPT from Days 5 to 13 postinoculation. Feed appeared to be retained in the crop and gizzard of infected birds when judged visually. However, measurements of total dry matter retained in various regions of the gastrointestinal tract did not differ significantly from each other.
(*Key words:* coccidiosis, *Eimeria*, gut motility, gut stasis, chicken)

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INTRODUCTION

Marked systemic and local physiological alterations are associated with coccidial infections. Systemic responses to coccidiosis include a substantial increase in blood glucose (Pratt, 1940), a concomitant decrease in muscle glycogen (Pratt, 1941; Freeman, 1970), and reduction in the ability of skeletal muscle to do work (Levine and Herrick, 1954). The physiology of the intestine *per se* is also greatly affected during acute coccidiosis (Turk, 1978; Ruff, 1986). Schildt and Herrick (1955) first reported decreased gut motility in chickens heavily infected with *Eimeria tenella*. They also found that feed was retained in the crop. Aylott *et al.* (1968) compared the effects of *E. necatrix, E. maxima,* and *E. tenella* on gut motility as measured by gut passage time. They found a marked increase in gut passage time (GPT) from Days 6 through 12 of infection. *Eimeria necatrix* infections resulted in the severest loss of motility, whereas the effect of *E. maxima* infections on GPT was less marked.

The objectives of this study were to further delineate the effect of intestinal coccidia on gut motility *in vivo* and to determine possible areas of the gastrointestinal tract affected by decreased gut motility.

MATERIALS AND METHODS

Birds. One-day-old male broiler chicks were maintained coccidia-free in suspended wire-floored cages until the beginning of each experiment. Birds were fed an unmedicated corn-soy ration (Table 1) throughout all experiments and had constant access to water.

Gut Passage Time. Gut passage time was calculated as the period of time immediately after the dye ingestion period to the first presence of dye in the feces. Clean brown paper was placed under the cages during the observation period to facilitate detection of dye. Observation times were scheduled for the same time of day throughout the experimental period. Two protocols were used in the GPT experiments.

In a preliminary GPT experiment, 48 broiler chicks were randomized by weight at 17 days of age. Each bird was placed in a Petersime brooder battery at a stocking ratio of one bird per cage so that individual GPT could be observed. After the birds had acclimated for 7 days, two groups of 16 chickens each were orally inoculated with either 1×10^4 or 5×10^5 oocysts of *E. acervulina*. A third group that was not inoculated served as a control.

On Days 3, 5, 7, and 10 postinoculation (PI), chicks were fasted for 1.5 to 2 hr and then allowed to feed on a ration containing 1% Day-Glo yellow (Day-Glo Color Corp., Cleveland, OH) for 5 to 10 min. After this period, birds were returned to nonpigmented feed. Cumulative

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TABLE 1. *Composition of the diet*

Ingredients	(%)
Ground yellow corn	57.75
Soybean oil meal (dehulled)	31.00
Poultry by-product meal	5.00
Poultry fat	3.00
Ground limestone	.65
Defluorinated phosphate	1.75
Salt	.40
DL-Methionine	.15
Trace mineral mix[1]	.05
Vitamin mix[2]	.25

[1] Trace mineral mix provides (ppm of diet): Mn, 60; Zn, 50; Fe, 30; Cu, 5; I, 1.05; Ca, 75 (min) and 90 (max).

[2] Vitamin mix provides (per kilogram/diet): vitamin A, 11,000 IU; vitamin D_3, 1100 ICU; vitamin E, 11 IU; riboflavin, 4.4 mg; Ca pantothenate, 12 mg; nicotinic acid, 44 mg; choline Cl, 220 mg; vitamin B_{12}, 6.6 μg; vitamin B_6, 2.2 mg; menadione, 1.1 mg (as menadione sodium bisulfite complex); folic acid, .55 mg; d-biotin, .11 mg; thiamine, 2.2 mg (as thiamine mononitrate); ethoxyquin, 125 mg.

weight gain was calculated for all birds during the period of infection.

In the second GPT experiment, 36 weight-randomized broiler chickens were placed in individual cages at 28 days of age (Guill and Washburn, 1972) and allowed to acclimate for 7 days. Two groups of 12 birds were then orally inoculated with either 5×10^5 oocysts of *E. acervulina* or 5×10^4 oocysts of *E. maxima*. A third group served as uninfected controls.

In the second experiment all birds were fasted for 1 hr and subsequently force-fed approximately 10 g of feed containing .25% Day-Glo orange. Chromic oxide (1%) was substituted as a marker on alternate days to ensure an accurate measure of GPT. After force feeding, birds were returned to the regular diet. Gut passage time was determined through Day 9 PI. Weight gain and feed consumption were determined daily.

Location of Gut Stasis. After GPT was determined on Day 10 PI in the preliminary experiment, all birds were kept an additional 18 hr. Eight chickens from each group were killed with sodium pentobarbitol injection and the entire gastrointestinal tract was excised, opened longitudinally, and examined for the location of a pigmented bolus.

In a second set of experiments, timed, paired necropsies were performed to determine the location of gut stasis during the acute phase of infection. In the first gut stasis location experiment, two groups of 35 28-day-old broiler chickens were inoculated with either 5×10^5 oocysts of *E. acervulina* or 5×10^4 oocysts of *E. maxima*. A third group remained uninoculated and served as a control. On the 7th day PI all birds were intubated with a glucose solution containing .5% Day-Glo orange. The first five birds intubated from each group were killed 1 hr later. Five chickens from each group were then killed every 30 min for five periods. All birds were frozen immediately after death to minimize problems of gut content movement and to aid in organ separation. The next day the chickens were partially defrosted and the crop, proventriculus/gizzard, small intestine (region from the gizzard sphincter to the yolk sac diverticulum), large intestine (region from the yolk sac diverticulum to the cloaca), and ceca were removed. All gut contents were removed and placed in individual containers for qualitative evaluation of the dye present.

Quantitative data on the location of feed retention was collected in a second gut stasis location experiment using the protocol just described. Gut contents from the crop, proventriculus/gizzard, duodenum (region from the gizzard sphincter to 1 cm past the pancreatic loop), jejunum (region past the duodenum to the yolk sac diverticulum), ileum (region from the yolk sac diverticulum to the cloaca), and ceca were dried in a forced draft oven and weighed.

Statistics. All results were analyzed using the general linear model (Statistical Analysis System, 1982) and means were analyzed using Duncan's multiple range test ($P<.05$).

RESULTS

Gut Passage Time. Data from the preliminary GPT experiment show that *E. acervulina* infections increase GPT (Table 2). Mean cumulative weight gains from both infected groups were significantly less than the control groups: birds infected with 1×10^4 oocysts gained 402 g, birds infected with 5×10^5 oocysts gained 377 g, whereas control birds gained 466 g.

Gut passage times were increased from Days 5 to 9 PI in birds infected with *E. acervulina* (Fig. 1) in the second GPT experiment. Significant changes in gut motility of birds infected with *E. maxima* occurred on Days 6 to 9 PI. Increases in GPT were greatest on Day 7 of the *E. acervulina* infection and on Day 8 of the *E. maxima* infection. An increase in GPT occurred

TABLE 2. *Gut passage time (means and standard errors) of uninfected chickens and chickens inoculated with Eimeria acervulina (preliminary experiment)*

Day postinoculation	Treatment		
	Uninfected	1×10^4	5×10^5
	min		
3	220 ± 8.0[1]	218 ± 3.2	231 ± 4.4*
5	191 ± 4.3	197 ± 3.2	195 ± 3.4
7	180 ± 5.6	190 ± 6.3*	>12 h*
10	174 ± 6.3	186 ± 6.3*	196 ± 7.6*

*Significantly different from controls (P<.05).

in all groups, including controls, on Day 5 PI.

Chickens infected with *E. acervulina* did not have reduced weight gains compared with controls (Fig. 2). However, weight gain was reduced significantly in chickens infected with *E. maxima* during the acute phase of the disease (days 4 to 8). Feed consumption was decreased from Days 5 to 9 PI in birds infected with *E. maxima*. However, feed consumption of birds infected with *E. acervulina* did not differ from controls (Fig. 3).

Location of Gut Stasis. In the preliminary GPT experiment, no dye was observed in the gastrointestinal tract of uninfected chickens. However, half of the chickens infected with 1×10^4 oocysts had a bolus containing dye in the crop or gizzard. All chickens infected with 5×10^5 oocysts had feed containing dye in the crop and gizzard.

Qualitative information about the movement of dye in the intestine is shown in Table 3. Dye

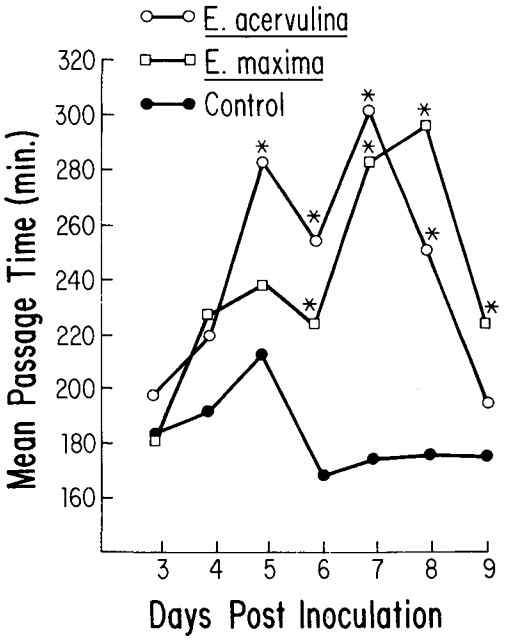

FIG. 1. Mean gut passage time of chickens infected with *Eimeria acervulina* or *E. maxima*. *Significantly different from control (P<.05).

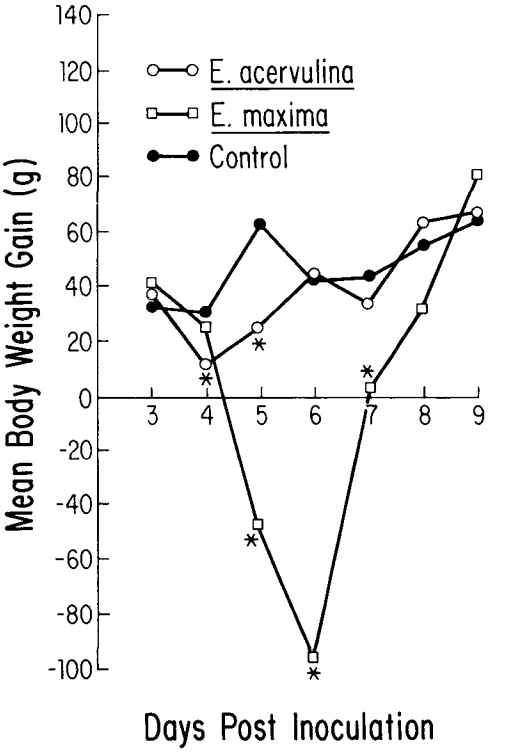

FIG. 2. Mean daily body weight gain of chickens infected with *Eimeria acervulina* or *E. maxima*. *Significantly different from controls (P<.05).

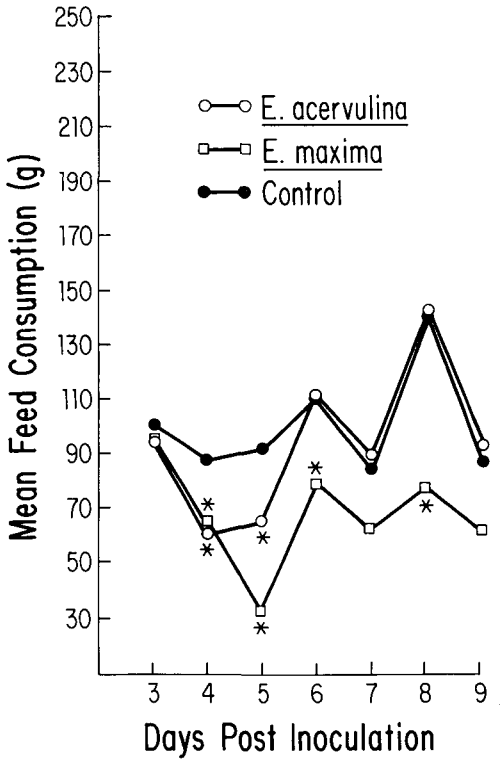

FIG. 3. Mean daily feed consumption of chickens infected with *Eimeria acervulina* or *E. maxima*. *Significantly different from controls (P<.05).

was observed in the small intestine of all groups within 30 min after intubation (data not shown). Dye was no longer present in the crop of uninfected birds by 210 min postintubation. Two control birds had defecated dye by 210 min. Both groups of infected birds had dye in all parts of the gastrointestinal tract at 210 min postintubation, but none had defecated dye.

Mean amounts of dry matter measured in organs of the gut are given in Table 4. Abnormal retention of feed in particular regions of the gut was not generally observed. However, significantly more dry matter was present in the ileum, and significantly less in the duodenum, of birds infected with *E. acervulina*, compared with controls. Significantly less dry matter was present in the crops of birds infected with *E. maxima* compared with controls.

DISCUSSION

In birds with heavy infections of *E. acervulina* and *E. maxima* GPT increased, although increased GPT was not consistently significant in birds infected with 1×10^4 oocysts of *E. acervulina* (Table 2, Fig. 1). Sturkie (1976) reported that diseased birds or birds that simply consume less feed have an increased GPT because of decreased food bulk in the gut. This is a possible explanation for the increased GPT observed in birds infected with *E. maxima*, because these birds did consume significantly less feed than controls (Fig. 3). However, the increase in GPT seen in birds infected with *E. acervulina* was not associated with decreased feed consumption. The increased GPT in control birds early in Experiment 2 may be explained

TABLE 3. *Qualitative evaluation of the location of dye in the digestive tract of chickens infected with Eimeria acervulina or E. maxima on day 6 postinoculation*

Time post-intubation	Treatment	Location[1]				
		Crop	Gizzard	Small intestine	Large intestine	Ceca
60 min	*E. acervulina*	++	++	++	–	–
	E. maxima	++	+–	+–	–	–
	Control	++	++	++	–	–
150 min	*E. acervulina*	+–	++	++	–	–
	E. maxima	++	++	++	+–	–
	Control	+–	++	++	–	–
210 min	*E. acervulina*	++	+–	++	+–	–
	E. maxima	++	+–	++	++	+–
	Control	–	+–	+–	++	+–

[1] ++ = Dye present in great amounts; +– = some dye present; – = no dye present.

TABLE 4. *Mean dry matter weight in grams and standard error of the mean in various areas of the gastrointestinal tract of birds during the 6th day postinoculation of Eimeria acervulina or E. maxima infections*

Treatment	n	Location					
		Crop	Gizzard	Duodenum	Jejunum	Ileum	Ceca
Uninfected	30	1.4 ± .23[a]	3.3 ± .19[a]	1.1 ± .06[a]	1.7 ± .09[a]	1.4 ± .12[b]	.53 ± .05[a]
E. acervulina	27	1.9 ± .34[a]	3.5 ± .30[a]	.93 ± .05[b]	1.9 ± .12[a]	2.1 ± .12[a]	.46 ± .03[a]
E. maxima	28	.56 ± .18[b]	2.8 ± .25[a]	1.1 ± .05[a]	1.5 ± .14[a]	1.6 ± .11[b]	.58 ± .05[a]

[a,b]Values within columns with different superscripts are significantly different ($P<.05$).

by the stress associated with force-feeding. However, after the third day of force-feeding control birds had a consistent GPT.

The location of feed retention sites in infected birds appears to be generalized (Table 4). However, based on qualitative assessment, it seems that feed is retained in the crop and gizzard for prolonged periods in infected birds compared with controls (Table 3). The crop has been noted as the site of feed retention in several studies. Schildt and Herrick (1955) reported that feed was retained in the crop of birds infected with *E. tenella* based on the location of dye in the gut. However, they also observed a general delay of feed passage in birds if the feed was not localized in the crop. Preston-Mafham and Sykes (1970) found that crops of birds infected with *E. acervulina* always contained feed or liquid. Sykes and Walters (1971) observed a reduced rate of gastric emptying in birds infected with *E. acervulina*, but intestinal transit of feed was not significantly affected.

Gastric motility is known to decrease when the pH of the duodenum is decreased (Duke and Evanson, 1972). A significant decrease in pH of the duodenum of birds infected with *E. acervulina*, and to a lesser extent with *E. maxima*, is well-documented (Ruff *et al.*, 1974; Ruff and Reid, 1975). The decreased pH of the region of the coccidial infection thus may influence the observed decreased rate of gastric emptying. The regulatory mechanisms of gastric and intestinal motility influenced by pH changes in the gut are primarily hormonal (Duke, 1984). However, the influence of gut hormones on the pathogenic aspects of coccidiosis has not been examined.

Several workers have tried to determine possible neuromuscular mechanisms, which might explain decreased motility of the gut when infected with coccidia (Oikawa and Kawaguchi, 1975; Witlock and Fetterer, 1983). Although Oikawa and Kawaguchi (1975) found somewhat generalized effects of acetylcholine on gut contractility for *E. acervulina* and *E. tenella*, Witlock and Fetterer (1983) observed that the contractility of infected cecal tissue was increased and there was no effect on contractility of intestinal tissues infected with *E. necatrix*. The latter workers postulated that changes in gut motility or related aberrations associated with coccidiosis may be due to changes in innervation of the intestine or blockage of neurotransmitters.

Effects of coccidial infections upon GPT are probably not directly related to effects of coc-

cidiosis on nutrient absorption (Aylott *et al.*, 1968). However, decreased motility of the gut may predispose some chickens to other infections, especially necrotic enteritis. Parish (1961) was only able to reproduce necrotic enteritis in chickens given a solution of opium and bicarbonate over a prolonged period, which theoretically decreased gut motility and allowed the *Clostridium* to develop toxins. Davis (1973) reported a correlation of coccidial infection and stress on the development of ulcerative enteritis in chickens, although this work has been difficult to reproduce.

The possibility that increased GPT may be influenced by a toxin produced by the parasite or by the host in response to the parasite has not been investigated, although lethal factors are associated with all coccidial species infecting the chicken (McKenzie *et al.*, 1985).

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