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M. C. Fox

D. R. Brown

L. L. Southern

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Effect of Dietary Buffer Additions on Gain, Efficiency, Duodenal pH, and Copper Concentration in Liver of *Eimeria acervulina*-Infected Chicks

M. C. FOX, D. R. BROWN,[1] and L. LEE SOUTHERN

Animal Science Department, Louisiana Agricultural Experiment Station, Louisiana State University Agricultural Center, Baton Rouge, Louisiana 70803

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ABSTRACT Three experiments were conducted with broiler chicks to investigate the effect of dietary additions of $NaHCO_3$(1%), $Al(OH)_3$(.5%), kaolin(1%), $Al(OH)_2NaCO_3$(.23%), $CaCO_3$(.37%), and MgO(1%) on gain, efficiency, duodenal pH, and liver Cu concentration of *Eimeria acervulina*-infected chicks. Experimental coccidial infection consistently reduced chick gain, efficiency, and duodenal pH, but it increased liver Cu concentration of chicks fed excess Cu. Sodium bicarbonate addition improved chick gain and efficiency slightly, whereas the MgO addition reduced these performance criteria. Sodium bicarbonate improved gain more in coccidiosis-infected chicks than in uninfected chicks, but it failed to alleviate, to any extent, the coccidiosis-induced liver Cu increase or the duodenal pH decrease.
(*Key words:* gain, efficiency, liver Cu, coccidiosis, dietary buffers)

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INTRODUCTION

Southern and Baker (1982a,b; 1983a,b) and Brown and Southern (1985a) have consistently shown that *Eimeria acervulina*-infected chicks were more susceptible to trace element toxicities than uninfected chicks. The exact mechanism of this trace mineral by coccidiosis interaction is not known. The coccidial infection, however, produces a myriad of adverse pathological symptoms (Stephens *et al.*, 1974; Ruff and Reid, 1975; Allen and Danforth, 1984), one of which is a reduction in duodenal pH. Availability of trace metals for absorption and utilization is enhanced by reduced duodenal pH (Hungerford and Linder, 1983; Menard *et al.*, 1983).

Use of buffers in livestock feeds has increased during the past 25 years. Buffers, or alkalizing agents, have improved gain and efficiency in dairy cattle, purportedly by an increase in digestive tract pH (Thomas and Emery, 1984). In trials with ruminants, alterations in digestive tract pH were extrapolated from fecal pH measurements. Although no correlation may exist between duodenal pH and fecal pH, it would be logical to assume that dietary buffers could influence duodenal pH in a similar manner.

We hypothesized that by reduction of the coccidiosis-induced drop in duodenal pH with dietary buffers, we might partially reduce the morbidity associated with coccidiosis. Thus, the purpose of this investigation was to assess the effect of dietary buffers on gain, efficiency, duodenal pH, and liver Cu concentration of uninfected and *Eimeria acervulina*-infected chicks. Gain and efficiency of gain are sensitive response criteria for evaluating *E. acervulina* infections (Hill *et al.*, 1985), and liver Cu concentration increases dramatically in *E. acervulina*-infected chicks fed high Cu diets (Southern and Baker, 1983a).

MATERIALS AND METHODS

Male Cobb (Experiment 1) or unsexed Arbor Acre (Experiments 2 and 3) chicks were used in this investigation. From hatching to 4 days posthatching, chicks received a corn-soybean meal diet (Table 1). After an overnight fast of both feed and water, chicks were inspected for fecal pasting and navel infection and culled if either condition existed. Chicks were then weighed, windbanded, and randomly assigned to treatment groups. They were provided continuous light and housed in heated, thermostatically controlled starter batteries (mean temperature of 35 C) with raised wire floors. Three replicates of five (Experiment 1) or six (Experiments 2 and 3) chicks were assigned to each treatment. The experimental periods were 5 to 13 (Experiments 1 and 2) or 5 to 15 days (Ex-

[1]Present address: Department of Animal Sciences, University of Arizona, Tucson, AZ 85721.

TABLE 1. *Composition of the basal diet*[1]

Ingredient	(%)
Dextrose	to 100.00
Corn	45.94
Soybean meal (44% CP)	42.50
Corn oil	5.00
Alfalfa leaf meal	2.00
Defluorinated rock phosphate	2.10
Oyster shell flour	.40
NaCl	.40
Vitamin mix[2]	.25
DL-Methionine	.15
$MnSO_4 \cdot H_2O$ (32.51% Mn)	.05
$ZnCO_3$	.01

[1] Calculated composition of the diet: crude protein (CP), 23%; lysine, 1.37%; methionine, .52%; cystine, .37%; Ca, .99%; total P, .79%; metabolizable energy, 3,000 kcal/kg.

[2] Roche Chemical Division, Nutley, NJ. Provided the following per kilogram of diet: retinyl acetate, 6,614 IU; cholecalciferol, 1,653 IU; dl-alpha-tocopheryl acetate, 7 IU; vitamin B_{12}, 11 μg; riboflavin, 6.6 mg; niacin, 33.1 mg; d-pantothenic acid, 11.0 mg; choline, 551 mg; menadione, 1.5 mg; folic acid, .7 mg; pyridoxine, 1.1 mg; thiamin, 1.1 mg; d-biotin, 55 μg.

periment 3) posthatching, and chicks were allowed *ad libitum* access to experimental diets and tap water. Average initial weight of the chicks was 68, 77, and 80 g for Experiments 1 to 3, respectively.

The basal diet (Table 1) was a conventional corn-soybean meal diet formulated to meet or exceed the nutrient requirements of growing chicks (National Research Council, 1984). Dietary additions were made to the basal diet at the expense of dextrose, and Cu was supplied by $CuSO_4 \cdot 5H_2O$.

Coccidial infections were established by crop intubation of 1 ml of an aqueous inoculum containing 1×10^6 (Experiments 1 and 2) or 4×10^5 (Experiment 3) sporulated *Eimeria acervulina* oocysts on Day 2 of Experiments 1 and 2 or on Days 0, 3, and 6 of Experiment 3. Uninfected control chicks received sham inoculations of tap water. The single inoculation with 1×10^6 sporulated oocysts (Experiments 1 and 2) was given to induce acute coccidiosis and subsequently to assess changes in duodenal pH during acute infection. Serial inoculation of chicks in Experiment 3 (Days 0, 3, and 6 of the experiment) with the lower level of sporulated oocysts allowed an extended experimental period necessary for optimum accumulation of liver Cu. This inoculation scheme also more closely parallels the natural infection encountered in practical poultry production systems. Coccidial infections were verified by recovery of viable oocysts in the feces of infected chicks.

In Experiment 1, the basal diet (B), B + 1% $NaHCO_3$, B + .5% $Al(OH)_3$, or B + 1% kaolin were fed to control or to coccidiosis-infected chicks. A similar treatment arrangement was employed in Experiment 2, but additions to the

TABLE 2. *Gain, efficiency, and duodenal pH of control (–) and Eimeria acervulina-infected (+) chicks fed $NaHCO_3$, $Al(OH)_3$, and kaolin (Experiment 1)*[1,2]

Dietary additions	Gain, g[3,4]		Gain:feed[3,4]		Duodenal pH[3]	
	–	+	–	+	–	+
Basal (B)	128	105	.801	.724	6.63	4.72
B + 1% $NaHCO_3$	134	116	.815	.761	6.51	4.35
B + .5% $Al(OH)_3$	128	112	.782	.754	6.46	4.69
B + 1% Kaolin	128	103	.778	.725	6.50	4.36
Pooled SEM	3.7		.014		.217	

[1] Gain and gain:feed data represent means of three replicates of five chicks each on Day 6 of the Experiment (Day 4 PI). The pH data represent means of three replicates of one chick each on Day 5 PI. Average initial chick weight was 68 g.

[2] Experimental coccidial infection was established by crop intubation of 1×10^6 sporulated *E. acervulina* oocysts on Day 2 of the Experiment.

[3] Coccidiosis effect ($P<.01$).

[4] Sodium bicarbonate effect; gain ($P<.06$), gain:feed ($P<.09$).

basal diet were B + .23% $Al(OH)_2NaCO_3$,[2] B + .37% $CaCO_3$,[2] or B + 1% MgO. In Experiment 3, 0 or 500 ppm Cu and 0 or 1% $NaHCO_3$ fed to control or coccidiosis-infected chicks, constitute a 2 × 2 × 2 factorial arrangement of treatments.

On Days 6, 7, and 8 of Experiments 1 and 2 (Days 4, 5, and 6 postinoculation), one chick was randomly selected from each replicate and killed by cervical dislocation. The duodenum was excised and pH of the lumen contents measured as previously described (Brown and Southern, 1985b). Only the duodenal pH data for Day 5 postinoculation (PI) are presented because the most severe effect of coccidiosis is detected at this time, and the Day 5 PI data are generally representative of Days 4 and 6 PI. Weight gain and feed consumption are presented for Day 6 (Day 4 PI) of Experiments 1 and 2 because the entire complement of chicks within a replication was represented on this day.

At the termination of Experiment 3 (Day 10 of the experiment), three chicks within a replicate were selected by uniform weight and killed by cervical dislocation. The duodenum was excised and pH of contents measured. The mean of the pH values from chicks within a replicate was taken as the observation for that replicate. Liver samples were obtained from these chicks and pooled within replicates. They were dried for 24 hr at 100 C, weighed, and then subjected to wet ashing with HNO_3 and H_2O_2. Copper analyses were made by atomic absorption spectroscopy (Association of Official Analytical Chemists, 1980).

Data were analyzed by analysis of variance procedures (Steel and Torrie, 1980) appropriate for factorially arranged treatments. Orthogonal or meaningful nonorthogonal single degree-of-freedom comparisons were used to detect treatment differences. Treatment variances for liver Cu data in Experiment 3 were proportional to the liver Cu means and were heterogeneous; therefore, one was added to each observation and the data log-transformed (natural) for statistical analysis.

[2] $Al(OH)_2NaCO_3$ and $CaCO_3$ were from commercially available antacids containing 23% $Al(OH)_2NaCO_3$ and 37% $CaCO_3$, which were added to the diet at the rate of 1%, respectively.

RESULTS AND DISCUSSION

The results of Experiment 1 are presented in Table 2. Experimental coccidial infection reduced ($P<.01$) gain, efficiency, and duodenal pH. Sodium bicarbonate additions tended to improve ($P<.09$) rate and efficiency of gain in both uninfected and in coccidiosis-infected chicks. The improvement in gain and efficiency, however, was greater in coccidiosis-infected chicks than in uninfected chicks. Improvement in gain was 5% in uninfected chicks but 10% in coccidiosis-infected chicks. Similarly, efficiency of gain was improved 2% in control chicks and 5% in coccidiosis-infected chicks. The coccidiosis × $NaHCO_3$ interactions were not significant ($P>.10$), however. Aluminum hydroxide and kaolin did not affect ($P>.10$) gain or efficiency of healthy or infected chicks. Duodenal pH was not affected ($P>.10$) by dietary buffer addition regardless of the infective state of the chicks.

In Experiment 2 (Table 3), experimental coccidial infection again reduced chick gain and efficiency ($P<.05$) as well as duodenal pH ($P<.01$). Calcium carbonate increased ($P<.06$) efficiency of feed utilization of both healthy and infected chicks. The effect was more pronounced, however, in coccidiosis-infected chicks. Magnesium oxide reduced ($P<.01$) rate and efficiency of gain of both uninfected and coccidiosis-infected chicks. Coccidiosis × $CaCO_3$ and coccidiosis × MgO interactions were observed in duodenal pH. Rather than ameliorating the coccidiosis-induced decrease in duodenal pH, however, both compounds exacerbated the coccidiosis-induced decline in duodenal pH. The $Al(OH)_2NaCO_3$ did not affect ($P>.10$) chick performance criteria or duodenal pH.

In Experiment 3 (Table 4), the coccidial infection, induced by serial inoculation of sporulated oocysts, reduced ($P<.01$) chick gain and feed efficiency. Copper addition had no effect ($P>.10$) on rate or efficiency of gain of healthy chicks but reduced both criteria in infected chicks (coccidiosis × Cu interaction, $P<.01$). Sodium bicarbonate did not affect gain or efficiency of uninfected chicks but improved these performance criteria in coccidiosis-infected chicks [coccidiosis × $NaHCO_3$ interaction in gain ($P<.02$) and gain:feed ($P<.07$)]. This improved performance was evident regardless of whether 500 ppm Cu was included in the diet. Thus, $NaHCO_3$ partially ameliorated the adverse

TABLE 3. *Gain, efficiency, and duodenal pH of control (−) and Eimeria acervulina-infected (+) chicks fed $Al(OH)_2NaCO_3$, $CaCO_3$, and MgO (Experiment 2)*[1,2]

Dietary additions	Gain, g[3,5]		Gain:feed[3,4,5]		Duodenal pH[3,6]	
	−	+	−	+	−	+
Basal (B)	139	126	.767	.727	6.33	6.19
B + .23% $Al(OH)_2NaCO_3$	147	130	.752	.728	6.47	5.95
B + .37% $CaCO_3$	134	131	.775	.767	6.58	5.36
B + 1% MgO	113	98	.687	.686	6.60	5.77
Pooled SEM	6.5		.012		.186	

[1] Gain and gain:feed data represent means of three replicates of six chicks each on Day 6 of the Experiment (Day 4 PI). The pH data represent means of three replicates of two chicks each on Day 5 PI. Average initial chick weight was 77 g.

[2] Experimental coccidial infection was established by crop intubation of 1×10^6 sporulated *E. acervulina* oocysts on Day 2 of the Experiment.

[3] Coccidiosis effect; gain and gain:feed ($P<.05$), pH ($P<.01$).

[4] Calcium carbonate effect ($P<.06$).

[5] Magnesium oxide effect ($P<.01$).

[6] Coccidiosis × $CaCO_3$ interaction ($P<.01$) and coccidiosis × MgO interaction ($P<.10$).

effects of duodenal coccidiosis as assessed by gain and gain:feed responses.

Duodenal pH was reduced ($P<.01$) by the coccidial infection and by the 500 ppm Cu addition; in combination, the two treatments further reduced duodenal pH [coccidiosis × Cu interaction ($P<.01$)]. Coccidiosis and dietary Cu also acted synergistically ($P<.01$) in affecting liver Cu concentration. Dietary Cu increased liver Cu concentration in uninfected chicks, and liver Cu content was further increased three- to fourfold in coccidiosis-infected chicks.

Duodenal coccidiosis clearly reduced chick gain, gain:feed, and duodenal pH regardless of whether chicks were inoculated once with a high dose of sporulated oocysts (Experiments 1 and

TABLE 4. *Gain, efficiency, duodenal pH, and liver copper concentration of control (−) and Eimeria acervulina-infected (+) chicks fed 500 ppm Cu, 1% $NaHCO_3$, or both (Experiment 3)*[1,2]

Dietary additions	Gain, g[3,4]		Gain:feed[3,4]		Duodenal pH[3]		Liver Cu, ppm[3,5]	
	−	+	−	+	−	+	−	+
Basal (B)	282	159	.790	.631	6.44	5.86	14	16
B + 1% $NaHCO_3$	279	168	.775	.666	6.48	5.80	14	10
B + 500 ppm Cu	285	93	.800	.493	6.29	4.23	216	637
B + Cu + $NaHCO_3$	272	110	.789	.531	6.03	4.00	199	824
Pooled SEM	5.5		.018		.167		71	

[1] Gain and gain:feed data represent means of three replicates of six chicks each on Day 10 of the Experiment. The pH and liver Cu data represent means of three replicates of three chicks each on Day 10 of the Experiment. Average initial chick weight was 80 g.

[2] Experimental coccidial infection was established by crop intubation of 4×10^5 sporulated *E. acervulina* oocysts on Days 0, 3, and 6 of the Experiment.

[3] Coccidiosis and Cu effects and coccidiosis × Cu interaction were significant ($P<.01$).

[4] Coccidiosis × $NaHCO_3$ interaction; gain ($P<.02$), gain:feed ($P<.07$).

[5] Treatment variances were heteroscedastic; therefore, data were log-transformed for statistical analysis. Pooled standard error of the means of transformed data was .154.

2) or serially inoculated with moderate doses of sporulated oocysts (Experiment 3). Use of dietary buffers was hypothesized to ameliorate, to some extent, the coccidiosis-induced reduction in duodenal pH, which subsequently would partially alleviate the coccidiosis-induced increase in liver Cu content. However, this hypothesis must be rejected since little or no effect on duodenal pH or on liver Cu content was observed from any of the buffers used in this investigation. In fact, $NaHCO_3$ in the presence of duodenal coccidiosis amd excess dietary Cu tended to increase liver Cu concentration. Sodium bicarbonate, however, tended to improve chick gain and efficiency in both uninfected and infected chicks, but the improvement was greater in coccidiosis-infected chicks than in controls. The beneficial effect of $NaHCO_3$ on chick performance was not mediated via changes in duodenal pH but probably by improving the electrolyte status of the diet and thus by partially alleviating a coccidiosis-induced acidosis.

The results of the duodenal pH determinations are in good agreement with previously reported values from our laboratory (Brown and Southern, 1985b, 1986) but are slightly lower than values reported by Riley and Austic (1984). The difference, as indicated by Riley and Austic (1984), is probably in the method of pH determination of the duodenal contents. Riley and Austic (1984) inserted a needle electrode directly into the duodenum, whereas we rinsed the duodenal contents into test tubes and measured the pH of this solution. Riley and Austic (1984), however, concluded that dietary electrolytes (K and Cl) did not affect intestinal pH. Similarly, we conclude from our investigation that dietary buffers do not affect duodenal pH.

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