METABOLISM AND NUTRITION

Effects of Cysteamine on Growth Performance, Digestive Enzyme Activities, and Metabolic Hormones in Broilers

C. M. Yang, A. G. Chen,[1] Q. H. Hong, J. X. Liu, and J. S. Liu

College of Animal Sciences, Zhejiang University, Huajiachi Campus, Hangzhou 310029, P.R. China

ABSTRACT A total of 600 avian male broilers at the age of 1 d were used to investigate the effects of cysteamine (CSH) on growth performance, digestive enzyme activities, and concentrations of serum hormones. The broilers received the same basal diets, with CSH added at 0 (control), 60, 90, 120, or 150 mg/kg. The feeding program consisted of a starter diet until 21 d and a grower diet until 42 d. The broilers with addition of CSH at 60 or 90 mg/kg had significantly higher growth rates during d 1 to 21 or d 21 to 42 compared with the control, respectively. However, adding 150 mg of CSH/kg significantly suppressed the growth of broilers. Adding 60 mg of CSH/kg significantly increased the activities of protease, amylase, and lipase in the pancreas and small intestinal contents during d 1 to 21, and the activities of protease and amylase in the small intestinal contents during d 21 to 42. Adding 90 mg of CSH/kg significantly increased the activities of lipase during d 1 to 21 and protease, amylase, and lipase during d 21 to 42 in small intestines. The activities of digestive enzymes during the whole period were suppressed by adding 150 mg of CSH/kg. The concentration of serum thyroxine was higher in the CSH-added birds during the whole period, whereas serum triiodothyronine was higher only during d 1 to 21 compared with the control. These findings indicate that low doses of dietary CSH may improve the growth performance and the activities of the digestive enzyme, but high doses of CSH appear to be detrimental to growth and digestion.

Key words: cysteamine, growth performance, digestive enzyme, metabolic hormone, broiler

2006 Poultry Science 85:1912–1916

INTRODUCTION

The sulfhydryl compound cysteamine (**CSH**) is biologically derived from the metabolism of cysteine. Within the body of mammals and chickens CSH may increase the release of growth hormone (**GH**) and improve growth rates (Hall et al., 1986; Gallavan et al., 1988; McLeod et al., 1995). The increased GH release is presumably due to the inhibitory effect of CSH on somatostatin (**SS**) from the hypothalamus, though the exact mechanism by which the CSH acts on the exhaustion of SS remains unclear. The most noted role of SS is as an inhibitor of pituitary secretion of GH (Brown et al., 1983; Dubreuil et al., 1989; Millard, 1989). However, SS has also been shown to exert inhibitory effects on numerous physiological processes in tissues, including neuroendocrine and exocrine secretions from the pancreas and gastrointestinal tract, cell proliferation, nutrient absorption, and splanchnic blood flow (Schusdziarra, 1985; Francis et al., 1990; Layer and Ohe, 1991).

Despite the extensive research that has been conducted on the regulation of CSH on GH and SS secretion, there has not been sufficient information on the effects of CSH on the activities of digestive enzymes and metabolic hormones, especially in broiler chickens. Furthermore, most of the current research on CSH is conducted by injection or infusion once per 6 or 7 d, which proves to be useful but not practical in depleting SS. Dietary administration of CSH would be much more practical than administering it by injection, especially for broiler breeders.

Therefore, an experiment was carried out to investigate the effects of the dietary addition of CSH on growth performance, digestive enzyme activities, and the concentration of metabolic hormones in broilers.

MATERIALS AND METHODS

Birds and Diets

All procedures were approved by the Zhejiang University Institutional Animal Care and Use Committee. Six hundred commercial male broiler chickens at 1 d of age were randomly divided into 5 equal groups of 120 birds each, and each of these groups was then assigned to 1 of 5 treatments. Birds in each group were kept in 4 pens (30 broilers each). All birds were offered the same basal diets with the addition of CSH (Walcom Bio-Chemicals Industrial Ltd., Shanghai, P.R. China; the antioxidant envelope was prepared by this company to prevent the oxidation of CSH) at levels of 0 (control), 60, 90, 120, and 150 mg/

Received November 22, 2005.
Accepted March 17, 2006.
[1]Corresponding author: agchen@zju.edu.cn

Table 1. Ingredients and nutrient composition of diets[1]

Ingredient, %	Starter (0 to 21 d)	Finisher (21 to 42 d)
Corn	52.6	57.4
Soybean meal	31.1	27
Wheat bran	2.0	4
Fish meal[2]	6.0	3
Rapeseed oil[3]	5.0	5
NaCl	0.3	0.3
Calcium phosphate	1.0	1.5
Limestone	1.2	1.2
DL-Methionine	0.3	0.1
Vitamin-mineral premix[4]	0.5	0.5
Nutrition composition		
Calculated		
ME, Mcal/kg	3.10	3.14
CP, %	22.52	19.74
Lys, %	1.19	1.08
Met + cysteine, %	0.93	0.71
Ca, %	1.00	0.90
Total P, %	0.80	0.76
Available P, %	0.47	0.39
Analyzed		
CP, %	22.44	19.66
Ca, %	1.03	0.94
Total P, %	0.84	0.79

[1]Nutrient level of the diets was based on NRC recommendations.

[2]Crude protein content is 62.5% and ME is 2.79 Mcal/kg.

[3]Metabolizable energy is 8.8 Mcal/kg.

[4]Supplied per kilogram of diet: vitamin A (retinyl acetate), 1,500 IU; cholecalciferol, 200 IU; vitamin E (DL-α-tocopheryl acetate), 10 IU; riboflavin, 3.5 mg; pantothenic acid, 10 mg; niacin, 30 mg; cobalamin, 10 μg; choline chloride, 1,000 mg; biotin, 0.15 mg; folic acid, 0.5 mg; thiamine, 1.5 mg; pyridoxine, 3.0 mg; Fe, 80 mg; Zn, 40 mg; Mn, 60 mg; I, 0.18 mg; Cu, 8 mg; Se, 0.15 mg.

kg. In the current study, antioxidized CSH contained 30% of CSH, and the dosages of CSH were the actual amount of CSH.

Diets were fed from d 1 to 42 and included starter (d 1 to 21) and finisher (d 21 to 42) phases. Nutrient levels of the diets (Table 1) were based on the NRC (1994) recommendations. Feeds were analyzed for CP, Ca, and total P, according to the methods of AOAC (1990). All chickens had free access to feed and drinking water. Temperature was maintained at 32°C for the first 5 d and then gradually reduced until a temperature of 22°C was achieved according to broiler management practices. Continuous lighting was maintained. Chickens were weighed individually at the age of 1, 21, and 42 d to determine average daily weight gain (**ADG**). Feed consumption on a pen basis was recorded daily, the uneaten feed was discarded, and the feeders were replenished with fresh feed. Average daily feed intake and feed-to-gain ratio were calculated. On d 21 and 42 of the feeding trial, 12 chickens per treatment (3 per pen) were slaughtered to obtain the small intestinal digesta and pancreatic tissue samples.

Digestive Enzyme Activities in Pancreatic Tissue and Small Intestinal Contents

The sampling of small intestinal digesta and the pancreas tissue was conducted according to the procedures described by Jin et al. (2000). Amylase activity (α-1, 4-glucan 4-glucanohydrolase, EC 3.2.1.1) was determined using a kit (no. 700, Sigma Chemical Co., St. Louis, MO) by the method of Somogyi (1960). Lipase activity (triacylglycerol lipase, EC 3.1.1.3.) was assayed using a kit (no. 800, Sigma) according to the method by Tietz and Fiereck (1966). Protease activity was analyzed using the modified method of Lynn and Clevette-Radford (1984), and all chemicals were purchased in a kit (no. 690, Sigma).

Blood Sample Collection and Analysis

On d 21 and 42, blood samples were obtained at 0, 25, 50, and 75 min after the morning feeding (0830 h) by jugular venipuncture into evacuated tubes, and then the samples were pooled to analyze the concentrations of thyroxine ($\mathbf{T_4}$), triiodothyronine ($\mathbf{T_3}$), insulin (**INS**), and gastrin (**GAS**). Blood samples were allowed to clot at 4°C and were centrifuged at 1,520 × g for 20 min before harvesting serum. Serum samples were stored at –20°C until assayed. Concentration of T_4, T_3, INS, and GAS were determined using the commercial RIA kit (Beifang Biotechnology Corp., Beijing, P.R. China).

Statistical Analysis

One-way ANOVA was performed using the GLM procedure of SAS software (SAS Institute Inc., 1989). Differences among dietary treatment means were compared using Duncan's multiple range tests (Steel and Torrie, 1980). A significance level of $P < 0.05$ was used.

RESULTS

Growth Performance

Growth performance of chickens is presented in Table 2. The ADG during d 1 to 21 was significantly higher in the birds fed 60 mg of CSH/kg, but lower in those with 120 or 150 mg of CSH/kg compared with the control; the ADG during d 21 to 42 was significantly higher with 90 mg of CSH/kg, but lower with 150 mg of CSH/kg. Over the experiment, chickens fed 90 mg of CSH/kg had higher ADG, whereas those on 120 or 150 mg of CSH/kg had lower ADG than the control. Chickens fed 150 mg of CSH/kg had lower ADG than those in other groups. Chickens fed 120 or 150 mg of CSH/kg had lower average daily feed intake during d 1 to 21, and those fed with 150 mg of CSH/kg had lower feed conversions than others during the whole period.

Digestive Enzymes

The activities of digestive enzymes in the pancreas and the small intestinal contents of chickens are shown in Table 3. The activities of protease, amylase, and lipase were higher in broilers fed 60 mg of CSH/kg than those fed 0 (control), 120, and 150 mg of CSH/kg during d 1 to 21. No significant differences were observed in protease and amylase between broilers fed with 60 and 90 mg of

Table 2. Effect of cysteamine (CSH) on growth performance of broilers[1]

	CSH, mg/kg						
	0	60	90	120	150	SEM	*P*-value
BW, g							
1 d	47	47	47	47	47	0.25	NS[2]
21 d	806[b]	870[a]	811[b]	653[c]	568[d]	9.37	<0.001
42 d	2,148[b]	2,229[a]	2,284[a]	2,038[c]	1,754[d]	42.40	<0.001
Average daily gain, g/d							
1–21 d	36[b]	39[a]	36[b]	29[c]	25[d]	0.45	<0.001
22–42 d	64[b]	65[b]	70[a]	66[b]	56[c]	1.99	<0.001
1–42 d	50[b]	52[ab]	53[a]	47[c]	41[d]	1.01	<0.001
Average daily feed intake, g/d							
1–21 d	50[b]	54[a]	50[b]	41[c]	38[d]	1.02	<0.001
22–42 d	123[b]	123[b]	137[a]	132[ab]	125[b]	5.23	0.052
1–42 d	87[bc]	88[ab]	94[a]	86[bc]	81[c]	2.65	0.060
Feed to gain, g/g							
1–21 d	1.39[b]	1.38[b]	1.38[b]	1.41[b]	1.52[a]	0.02	<0.001
22–42 d	1.91[c]	1.90[c]	1.96[bc]	2.01[b]	2.22[a]	0.04	<0.001
1–42 d	1.73[c]	1.70[c]	1.76[bc]	1.82[b]	2.00[a]	0.03	<0.001

[a–d]Means within a row with different letters differ significantly ($P < 0.05$).

[1]Data in each group represented mean values of 4 replicates, 1 replicate indicating the average of 30 chickens in 1 cage.

[2]$P > 0.10$.

CSH/kg or in amylase and lipase between broilers fed with 120 and 150 mg of CSH/kg during d 1 to 21. Chickens fed with 90 mg of CSH/kg had higher activities of protease in the pancreas as well as protease, amylase, and lipase in the small intestinal contents, compared with the control during d 21 to 42. Birds fed 150 mg of CSH/kg had lower activities of protease than other groups during d 21 to 42.

Metabolic Hormone

The serum concentrations of T_4, T_3, INS, and GAS of chickens are shown in Table 4. During d 1 to 21, adding 60, 90, 120, and 150 mg of CSH/kg significantly increased the serum concentrations of T_4 and T_3, whereas adding 120 and 150 mg of CSH/kg significantly increased the serum concentrations of GAS. No significant differences

Table 3. Effects of cysteamine (CSH) on the digestive enzyme activities in the pancreas and the small intestinal contents of broilers[1]

	CSH, mg/kg						
	0	60	90	120	150	SEM	*P*-value
Pancreas, 1–21 d							
Protease, unit[2]	137.72[b]	162.82[a]	150.08[ab]	112.57[c]	100.08[c]	7.54	<0.001
Amylase, Somogyi unit[3]	31.77[b]	43.60[a]	37.72[ab]	27.61[bc]	22.84[c]	2.20	<0.001
Lipase, Sigma-Tietz unit[4]	36.09[b]	43.43[a]	34.60[b]	31.51[bc]	25.83[c]	2.34	<0.001
Pancreas, 21–42 d							
Protease, unit	166.13[bc]	176.25[b]	198.72[a]	157.95[c]	125.96[d]	7.76	<0.001
Amylase, Somogyi unit	42.86[ab]	45.25[ab]	47.79[a]	39.45[b]	39.06[b]	3.32	<0.001
Lipase, Sigma-Tietz unit	37.30	39.07	40.95	36.99	35.73	2.90	NS[5]
Small intestine, 1–21 d							
Protease, unit	66.72[b]	77.86[a]	70.92[ab]	66.43[b]	53.36[c]	3.29	<0.001
Amylase, Somogyi unit	12.19[b]	13.89[a]	12.65[ab]	11.23[b]	11.19[b]	0.76	0.040
Lipase, Sigma-Tietz unit	11.14[b]	15.53[a]	17.03[a]	9.92[b]	9.52[b]	1.12	<0.001
Small intestine, 21–42 d							
Protease, unit	86.02[b]	99.05[a]	100.33[a]	85.55[b]	71.62[c]	6.26	<0.001
Amylase, Somogyi unit	13.41[b]	18.79[a]	21.07[a]	11.83[b]	11.76[b]	1.17	<0.001
Lipase, Sigma-Tietz unit	22.67[b]	24.27[ab]	27.25[a]	22.17[b]	21.82[b]	1.65	0.010

[a–d]Means within a row with different letters differ significantly ($P < 0.05$).

[1]Means represent 12 chickens at the age of 21 and 42 d (3 chickens from each of 4 pens) per treatment.

[2]Protease activity unit was defined as milligrams of azocasein degraded during 2 h of incubation at 38°C per milligram of intestinal digesta protein or pancreas.

[3]Amylase activity unit (1 Somogyi unit) was defined as the amount of amylase that will cause formation of reducing power equivalent to 1 mg of glucose in 30 min at 38°C per milligram of intestinal digesta protein or pancreas.

[4]Lipase activity unit (1 Sigma-Tietz unit) was equal to the volume (milliliter) of 0.05 *M* NaOH required to neutralize the fatty acid liberated during 6 h incubation with 3 mL of lipase substrate at 38°C per milligram of intestinal digesta protein or pancreas.

[5]$P > 0.10$.

Table 4. Effects of cysteamine (CSH) on serum concentrations of thyroxine (T_4), triiodothyronine (T_3), insulin (INS), and gastrin (GAS) in broilers[1]

	CSH, mg/kg						
	0	60	90	120	150	SEM	*P*-value
1–21 d							
T_4, μg/L	121.13[b]	164.02[a]	175.65[a]	177.02[a]	173.52[a]	10.80	<0.001
T_3, μg/L	3.31[b]	5.40[a]	6.03[a]	5.79[a]	5.80[a]	0.27	<0.001
INS, μU/mL	37.66	38.73	40.20	42.80	41.35	2.57	NS[2]
GAS, ng/mL	70.71[b]	75.96[ab]	77.29[ab]	83.16[a]	83.90[a]	4.23	0.017
21–42 d							
T_4, μg/L	109.07[b]	116.05[b]	140.39[a]	141.41[a]	157.06[a]	9.71	<0.001
T_3, ng/dL	2.70	2.73	2.99	2.92	3.02	0.17	NS
INS, μU/mL	21.18	22.25	23.46	21.28	23.37	1.33	NS
GAS, ng/mL	60.60[b]	63.16[ab]	63.27[ab]	65.41[ab]	67.34[a]	3.03	NS

[a,b]Means within a row with different letters differ significantly ($P < 0.05$).

[1]Means represent 12 chickens per treatment of a total of 4 blood samples per chicken at the age of 21 and 42 d (3 chickens from each of 4 pens). The blood sample was taken at 0, 25, 50, and 75 min after feeding, respectively, and analyzed as pooled.

[2]$P > 0.10$.

were observed in T_4 and T_3 among broilers fed with 60, 90, 120, and 150 mg of CSH/kg. During d 21 to 42, adding 90, 120, and 150 mg of CSH/kg significantly increased the serum concentration of T_4, whereas the serum concentration of GAS was significantly increased with addition of 150 mg of CSH/kg.

DISCUSSION

Growth hormone plays the most important role in animal growth, whereas CSH has a great effect on GH secretion by depleting SS (Szabo and Reichlin, 1981; Millard et al., 1985; McLeod et al., 1995). Therefore, the CSH added in diets may affect the growth performance of broiler chickens.

The growth performance of broilers was improved with the lower dose of CSH (60 and 90 mg of CSH/kg), whereas the addition at a high dose (150 mg of CSH/kg) had an adverse effect on growth performance. Little published information is available on the effect of dietary CSH on growth performance. Using growing chicks at 3 to 10 d of age and 17 to 24 d of age, Zavy and Lindsey (1988) observed that feed efficiency was significantly improved by adding 1,200 and 1,800 mg of CSH/kg, attributable to a significant decrease in feed intake. There was no difference in weight gains between chickens in the control group and those receiving 1,200 mg of CSH/kg, but a significant decrease in weight gain was observed in chickens fed 1,800 mg of CSH/kg. The dose of CSH used in this study was much lower than that by Zavy and Lindsey (1988), because our CSH was enveloped by the antioxidant. It was observed that the addition of 100 mg of CSH/kg of BW per week to the diet of broiler fowls from 21 or 32 d of age increased weight gain and food conversion but decreased abdominal fat pad weight (Han and Lin, 1992). Low doses of CSH appear to have a specific effect on SS-mediated inhibition of GH secretion, whereas high doses of CSH might disrupt GH secretion by an alternative mechanism (Millard et al., 1983; McLeod et al., 1995). In addition, high doses of CSH might result in gastric ulcers and gastrointestinal lining damage (Szabo and Reichlin, 1985; Nassar et al., 1987; Yang et al., 2002).

The activities of protease, amylase, and lipase in the pancreas and small intestinal contents of broilers were increased with the addition of CSH at 60 and 90 mg of CSH/kg, whereas they were decreased at 150 mg of CSH/kg. It is known that SS inhibits secretion of digestive enzymes from the pancreas and gastrointestine, whereas CSH can relieve the inhibition. Ai and Han (2002) reported that CSH added in the diet of geese at dosages of 100 mg/kg of BW per week increased pancreatic secretion. Amylase activity was higher in CSH-added birds than in the control birds. It might be due to the depletion of SS resulting from the addition of CSH, because depletion of SS could enhance the movement of gastrointestine, increase the enzyme secretion of the pancreas and gastrointestine, and, finally, improve the activities of digestive enzymes.

In mammals, CSH has been shown to promote the release of INS from pancreatic islets; however, greater levels of CSH are required to alter INS release than GH secretion (Patel et al., 1985; Hashimoto et al., 1987). Conversely, purified INS has been demonstrated to have similar sensitivity to SS when incubated in the presence of CSH (Hashimoto et al., 1987). In in vitro studies, CSH has been shown to promote basal and glucose-stimulated releases of INS from pancreatic islets in rats and mice (Patel et al., 1985; Petersson and Hellerstrom, 1985). Rideau et al. (1990) studied the effects of subcutaneous injection of CSH (300 mg/kg) in 5- to 6-wk-old chickens. Cysteamine increased plasma glucose and INS in the short term (1 h), but INS was not modified in a longer term (17 to 24 h). In a study with geese, Ai et al. (2004) observed that CSH decreased the plasma SS levels and increased the levels of T_4 and glucagon, resulting in benefits in growth. Xiao and Lin (2003) reported that the action of CSH in stimulating GH release in vitro appeared to be mediated through hypothalamic pathways. Dietary delivery of CSH directly or indirectly stimulated endoge-

nous GH, T_3, and T_4 secretion, leading to an increase in the growth rate in grass carp.

In the present experiment, adding CSH improved serum concentrations of T_4 and T_3 but did not affect INS. In addition, high doses of CSH significantly increased serum concentrations of GAS. The enhanced concentration of T_4, T_3, and INS might promote the growth of chickens, but high GAS content might result in gastric ulcers and, consequently, decrease growth performance.

In conclusion, lower doses of dietary CSH increased growth performance and enhanced activities of digestive enzymes in broilers, but higher doses of CSH appeared to decrease activities of digestive enzymes and, hence, inhibit growth performance. Adding CSH exerted effects on the concentrations of metabolic hormones.

ACKNOWLEDGMENTS

This work was partially supported by the National Basic Research Program of China (project no. 2004CB117506). We gratefully acknowledge X. F. Zheng and H. Yang for their skilled technical assistance. Special thanks to H. H. Wang and L. Y. Wu for experimental arrangements.

REFERENCES

Ai, X. J., and Z. K. Han. 2002. Effect of cysteamine on the pancreatic secretion and lipase activity of geese. J. Vet. Sci. 22:624–626. (in Chinese)

Ai, X. J., Y. L. Zheng, G. T. Liu, W. H. Chen, and Z. K. Han. 2004. Effects of cysteamine on the hormone contents and cholesterol in plasma of adult geese. J. Vet. Sci. 24:366–368. (in Chinese)

AOAC. 1990. Official Methods of Analysis. 15th ed. Association of Official Analytical Chemists, Arlington, VA.

Brown, M. R., L. A. Fisher, P. E. Sawchenko, L. W. Swanson, and W. W. Vale. 1983. Biological effects of cysteamine: Relationship to somatostatin depletion. Regul. Pept. 5:163–179.

Dubreuil, P., G. Pelletier, D. Lapierre, H. Petitclerc, P. Gaudreau, and P. Brazeau. 1989. Effects of active immunization against somatostatin on serum growth hormone concentration in growing pigs: Influence of fasting and repetitive somatocrinin injections. Endocrinology 125:1378–1384.

Francis, B. H., D. G. Baskin, D. R. Saunders, and J. W. Habener. 1990. Distribution of somatostatin-14 and somatostatin-28 in gastrointestinal-pancreatic cells of rats and humans. Gastroenterology 99:1283–1287.

Gallavan, R. H. Jr., M. T. Zavy, and D. L. VonTungeln. 1988. Effects of peroral cysteamine (2-aminoethanethiol) on ovine somatostatin (SS), feed intake (FI), thyroxine (T_4) and triiodothyronine (T_3). J. Anim. Sci. 66(Suppl. 1):277. (Abstr.)

Hall, T. R., A. Cheung, S. Harvey, S. K. Lam, and G. G. Spencer. 1986. Somatostatin immunoneutralization affects plasma metabolite concentrations in the domestic fowl. Comp. Biochem. Physiol. A. 85:489–497.

Han, Z. K., and L. Lin. 1992. Effect of a somatostatin inhibitor, cysteamine, on growth in broilers. Acta Vet. Zoot. Sin. 23:314–318. (in Chinese)

Hashimoto, N., A. Kanatsuka, H. Makino, M. Sakurada, and S. Yoshida. 1987. Mechanisms involved in the depleting effect of cysteamine on pancreatic somatostatin. Horm. Metab. Res. 19:11–14.

Jin, J., Y. W. Ho, N. Abdullah, and S. Jalaludin. 2000. Digestive and bacterial enzyme activities in broilers fed diets supplemented with lactobacillus cultures. Poult. Sci. 79:886–891.

Layer, P., and M. Ohe. 1991. Effects of somatostatin on the exocrine pancreas. Scand. J. Gastroenterol. 26:129–136.

Lynn, K. R., and N. A. Clevette-Radford. 1984. Purification and characterization of hevin, a serin protease from *Hevea brazilliensis.* Biochem. J. 23:963–964.

McLeod, K. R., D. L. Harmon, K. K. Schillo, S. M. Hileman, and G. E. Mitchell. 1995. Effects of cysteamine on pulsatile growth hormone release and plasma insulin concentrations in sheep. Comp. Biochem. Physiol. B. Biochem. Mol. Biol. 112:523–533.

Millard, W. J. 1989. Central regulation of growth hormone secretion. Pages 237–255 in Animal Growth Regulation. D. R. Campion, G. J. Hausman, and R. J. Martin, ed. Plenum Press, New York, NY.

Millard, W. J., S. M. Sagar, T. M. Badger, and J. B. Martin. 1983. Cysteamine effects on growth hormone secretion in the male rat. Endocrinology 112:509–517.

Millard, W. J., S. M. Sagar, and J. B. Martin. 1985. Cysteamine-induced depletion of somatostatin and prolactin. Fed. Proc. 44:2546–2550.

Nassar, C. F., O. H. Halabi, A. R. Jurjus, and Z. M. Habbal. 1987. Cysteamine-induced duodenal ulcer: Effects on calcium absorption across rat duodenum. Comp. Biochem. Physiol. A. 86:103–107.

National Research Council. 1994. Nutrient Requirements for Poultry. 9th rev. ed. Natl. Acad. Press, Washington, DC.

Patel, Y. C., I. Pierzchala, M. Amherdt, and L. Orci. 1985. Effects of cysteamine and antibody to somatostatin on islet cell function in vitro: Evidence that intracellular somatostatin deficiency augments insulin and glucagon secretion. J. Clin. Invest. 75:1249–1255.

Petersson, B., and C. Hellerstrom. 1985. Rapid depletion of somatostatin in isolated mouse pancreatic islets after treatment with cysteamine. Acta Endocrinol. (Copenh.) 110:227–231.

Rideau, N., H. Karmann, and J. Simon. 1990. Effects of cysteamine administration on plasma concentration of metabolites, pancreatic glucagon and insulin in the chicken. Comp. Biochem. Physiol. A. 96:327–331.

SAS Institute Inc. 1989. SAS/STAT User's Guide. Version 6. SAS Inst. Inc., Cary, NC.

Schusdziarra, V. 1985. Role of somatostatin in nutrient regulation. Adv. Exp. Med. Biol. 188:425–427.

Somogyi, M. 1960. Modification of two methods for the assay of amylase. Clin. Chem. 6:23–27.

Steel, R. G. D., and J. H. Torrie. 1980. Principles and Procedures of Statistics: A Biometrical Approach. 2nd ed. McGraw-Hill, New York, NY.

Szabo, S., and S. Reichlin. 1981. Somatostatin in rat tissues is depleted by cysteamine administration. Endocrinology 109:2255–2257.

Szabo, S., and S. Reichlin. 1985. Somatostatin depletion by cysteamine: Mechanism and implication for duodenal ulceration. Fed. Proc. 44:2540–2548.

Tietz, N. W., and E. A. Fiereck. 1966. A specific method for serum lipase determination. Clin. Chim. Acta 13:352–355.

Xiao, D., and H. R. Lin. 2003. Cysteamine - a somatostatin-inhibiting agent - induced growth hormone secretion and growth acceleration in juvenile grass carp (*Ctenopharyngodon idellus*). Gen. Comp. Endocrinol. 134:285–295.

Yang, C. M., A. G. Chen, and J. S. Liu. 2002. Effects of cysteamine on growth performance and peptic in broilers. Chin. J. Anim. Sci. 38:16–17. (in Chinese)

Zavy, M. T., and T. O. Lindsey. 1988. Effect of cysteamine administration on growth and efficiency of food utilization in chicks. Br. Poult. Sci. 29:409–417.