

Effects of Dietary Zinc Levels on Zinc, Iron and Copper Content in Tissues of the White Leghorn Cockerels

Eltohamy, Magda Mohammed

Laboratory of Animal Husbandry II, Faculty of Agriculture, Kyushu University

Takahara, Hitoshi

Laboratory of Animal Husbandry II, Faculty of Agriculture, Kyushu University

Okamoto, Masao

Laboratory of Animal Husbandry II, Faculty of Agriculture, Kyushu University

<https://doi.org/10.5109/23696>

出版情報：九州大学大学院農学研究院紀要. 24 (2/3), pp.65-74, 1979-11. Kyushu University
バージョン：
権利関係：

Effects of Dietary Zinc Levels on Zinc, Iron and Copper Content in Tissues of the White Leghorn Cockerels

**Magda Mohammed Eltohamy, Hitoshi Takahara
and Masao Okamoto**

Laboratory of Animal Husbandry II, Faculty of Agriculture,
Kyushu University 46-06, Fukuoka 812

(Received May 17, 1979)

An experiment was conducted with one day-old White Leghorn male chicks to study the effects of different levels of dietary Zn on the concentration of Zn, Fe and Cu in different organs. Regression analysis revealed positive correlation between dietary Zn concentration and Zn concentration in the liver and pancreas until 50 days of age and in the testes and kidneys until 40 days. There was negative correlation between dietary Zn concentration and tissue Zn at duration of feeding in the testes with all dietary Zn levels and in the pancreas with the two higher levels of Zn. Zn concentration in the heart, brain, breast muscle and leg muscle was not influenced by addition of Zn to the diet. The antagonistic effect of Zn with Fe contents was observed at 40, 50 and 85 days of age in the liver. The same effect was detected at 50 and 85 days in the testes and kidneys. In the spleen and heart, accompanying the increase in the dietary Zn, decreases in Fe concentration were evident at 30 and 50 days. No significant changes were found in Fe concentration in the brain and muscles throughout the experimental period. The lack of a loss of Fe concentration in these organs suggests that a critical level of Zn was not attained to affect Fe concentration. The Cu concentration in the liver, kidneys, heart and brain was not affected by addition of Zn to the diet and by duration of feeding.

INTRODUCTION

Adaptation to environmental alteration and the capacity of self regulation are fundamental characteristics of living systems which result in the maintenance of a dynamic steady state called homeostasis. A basic requirement in maintaining homeostasis is the ability to alter the rate of metabolic reactions which underlie the various physiological processes. Zn homeostasis has been established in rats and ruminants. (Stake *et al.*, 1973, 1975; Ansari *et al.*, 1976). Information relating to regulation may be obtained by investigating the role of different levels of Zn in the maintenance of homeostasis (Miller *et al.*, 1970; Stake *et al.*, 1975). In ruminants, Zn homeostasis is effective with low dietary Zn but less effective with the high Zn level.

The interactions of Zn with other dietary constituents are important especially in the study of Zn. It appears that no particular symptom may be specifically attributed to Zn alone, but some changes are observed as a result of several other nutrients. Smith and Larson (1946) indicated that the micro-

cytic anemia caused by feeding a relatively high concentration of Zn in rats was actually the result of impaired utilization of Fe for hemoglobin formation and this condition was corrected by addition of Cu to the diet. Later, Settlemire and Matrone (1967) suggested that Zn affected Fe metabolism by; a) impairing the incorporation of Fe into or release from ferritin, which would influence Fe absorption and storage, and b) shortening the life span of erythrocytes, which would cause a faster turnover of Fe. Van Campen and Scalfe (1967) postulated that Zn alters Cu metabolism by impairing its absorption as mediated primarily via the direct effect of Zn either in or on the intestine, when rats received Zn either intraduodenally or intraperitoneally.

Excess dietary Zn has been documented to produce an antagonism to Fe, Cu and Ca in rats (Cox and Harris, 1960). But this effect is not completely understood in the fowl.

The present study was therefore undertaken to investigate the effects of dietary Zn levels on the concentration of Zn, Fe and Cu in different organs.

MATERIALS AND METHODS

The experiment was conducted with one day-old White Leghorn cockerels randomly divided into 4 groups, one control and three experimental groups, of 30 chicks each. The control group was fed the basal diet described previously (Eltohamy *et al.*, 1979) containing 32 ppm Zn, 85 ppm Fe and 5 ppm Cu. Three groups were given the experimental diets which 50, 250 and 1000 ppm Zn (as Zn ammonium sulphate) were added to the basal diet respectively. The experimental period was 85 days. The diet and distilled water were offered *ad libitum*. Five chicks from each group were killed at 10, 20, 30, 40, 50 and 85 days of age. The liver, testes, pancreas, kidneys, heart, spleen, brain, breast muscle and leg muscle were removed rapidly, and the individual organs from each chick were stored separately at -20°C until required for analysis.

The tissues were wet-digested with a 3 : 1 (v/v) mixture of nitric and perchloric acids and then assayed by atomic absorption spectrophotometry for Zn, Fe and Cu determination. Zn, Fe and Cu content were calculated as Zn, Fe and Cu concentration (ppm) on fresh weight basis. The results were analysed statistically by analysis of variance and linear regression correlation (Snedecor, 1956). The standard error of the mean was given.

RESULTS

Data showing the effects of different levels of dietary Zn on the concentration of Zn, Fe and Cu in selected organs are presented in Tables 1, 2 and 3. At 10 days of age, the concentration of Zn in the liver, pancreas, testes, kidneys and spleen of cockerels fed 1000 ppm dietary Zn was significantly higher than those fed the basal control diet. There was a significant increase in the concentration of Zn in the liver and spleen of cockerels fed 250 ppm dietary Zn compared with those from the control group. But the apparent increase

Table 1. Effect of dietary Zn levels on Zn concentration of selected organs.

Tissue	Zn added to the diet (ppm)	Tissue Zn concentration at the experimental period					
		10 days (ppm)	20 days (ppm)	30 days (ppm)	40 days (ppm)	50 days (ppm)	85 days (ppm)
Liver	0	24.0±2.2*	22.1±1.9*	21.1±12.1*	21.6±4.2"	20.7±1.3*	27.3±4.5"
	50	30.1±3.9	27.4±7.51'	27.2±1.0"	26.8±1.82'	24.4±4.6"	32.2±4.3
	250	37.0±7.1 ¹⁾	29.6±2.5	29.0±2.4"	24.5±2.3	22.4±3.42'	28.1±9.9
	1000	64.2±28.4 ¹⁾	36.0±10.9 ¹⁾	51.8±3.4 ¹⁾	44.71-24.21'	28.1±4.3 ³⁾	36.4±5.3
Pancreas	0	28.8±9.4	33.1-c 4.9	24.7±8.5	19.2±1.3	15.9, 2.6	24.9±5.4
	50	47.1±9.2	44.9±9.2	39.1±5.5	34.8±3.82'	23.6±6.92'	40.6±0.8 ¹⁾
	250	53.2±6.1	50.2±2.0	47.6±3.0 ²⁾	29.8±3.9 ²⁾	25.4±2.8	15.3±1.0"
	1000	110.7±34.1 ¹⁾	79.9±7.9	78.6±2.72'	49.9±12.0 ²⁾	43.4±11.4 ¹⁾	21.8±4.5
Testes	0	83.3±29.9	77.1±26.6	77.3±47.6	35.8±7.4	24.2±7.6	13.4±1.6
	50	144.7i27.4	141.3i11.71'	93.1±60.6	32.3±5.7	33.5±9.4	13.6±0.5
	250	210.4±79.0	159.8±78.0 ²⁾	87.1i37.2	35.1±12.5	38.3±17.1	6.5±0.2 ¹⁾
	1000	306.6±35.3 ¹⁾	231.5±69.9 ¹⁾	100.8±47.4	45.1±18.5 ³⁾	42.6±25.9 ¹⁾	6.2±0.6 ²⁾
Kidneys	0	27.9±8.6	18.4±1.3	19.4±3.4	18.7±5.7	17.1±2.4	19.4±1.2
	50	26.4±6.5	19.6±2.3	21.2±1.22'	22.9±0.5 ¹⁾	20.3±1.8	23.9±1.9
	250	29.1±6.5	20.6±2.0	22.7±1.5"	22.6±2.5	19.1±1.5	23.1±2.2
	1000	34.4±2.82'	25.3±1.7	28.4±4.8	28.7±4.6	21.7±2.4	23.5±1.1
Spleen	0	28.5±4.5	35.9±9.3	25.1±3.9	24.7±3.9	20.4±0.9	22.7±0.7
	50	25.5±4.5	36.2±6.2	25.9±2.2	29.6±4.3	25.2±2.32'	22.5±0.7
	250	42.8±18.1 ¹⁾	42.0i112.5	28.8±2.9	30.5±0.1	21.9±1.5	22.4±0.7
	1000	46.9±12.9 ²⁾	48.7±15.0	28.6±3.4	32.6±6.0	22.5±0.9	20.7±0.8
Heart	0	40.8±10.9	25.8±3.5	24.5±2.2	24.2±1.7	24.1±1.8	25.9±0.8
	50	37.2±13.2	26.2±3.4	24.5±1.5	25.6±1.2	24.8±0.9	25.0, 3.8
	250	34.3±5.3	27.2±2.5	26.4±1.8	24.2±5.8	23.8±2.3	25.0±1.5
	1000	36.3±8.8	26.4i 4.1	27.3±1.5	24.1±0.9	24.2±2.0	24.4±1.1
Brain	0	11.1±1.4	11.7±0.6	11.3±0.5	12.6±2.0	8.8±1.4	10.1±1.2
	50	11.9±1.3	14.5±1.7	11.5±0.8	12.0±2.0	10.3±0.6	10.4±0.6
	250	12. a+ 3.0	13.2±0.4	11.3±1.1	13.9±2.8	9.8±0.7	12.0±1.2
	1000	13.0±1.6	13.2±2.7	12.9±0.6	12.3±0.8	10.5±0.7	11.6±2.6
Breast muscle	0	11.7±3.4	7.6±0.9	6.2±0.7	4.6±0.2	4.8, 0.5	4.8±0.2
	50	11.4±0.8	7.8±0.9	5.7±0.4	4.9±0.6	5.5±0.9	5.8±0.9
	250	16.0±4.2	7.8±0.9	6.1±0.3	5.3±0.6	5.8±2.1	5.3±0.6
	1000	18.5±3.5	8.4i 0.7	5.9±0.6	5.4±1.2	4.8±0.4	5.6±1.6
Leg muscle	0	21.3±3.2	18.0-t 1.8	14.8±1.1	13.1±0.6	13.6±2.0	14.2±3.5
	50	24.0±6.8	18.6±2.8	15.1±2.8	13.7±1.6	21.7±4.5	10.9±0.8
	250	24.8±9.7	19.4±2.4	16.8±2.5	14.8±2.9	12.8±2.9	9.9±1.4
	1000	25.4±3.5	20.4±3.5	16.4±1.1	14.7, 1.4	11.1±1.8	15.9±3.1

* Mean value & standard error. ¹⁾ Significantly different ($p < 0.01$) from control group by analysis of variance. ²⁾ Significantly different ($p < 0.05$) from control group by analysis of variance.

in the concentration of Zn in the other organs, when 250 and 50 ppm dietary Zn was fed, was not statistically significant. Though Zn concentration was reduced in the heart with the dietary Zn levels, the reduction was not statistically significant.

The data indicated that increasing levels of dietary Zn were associated

Table 2. Effect of dietary Zn levels on Fe concentration of selected organs.

Tissue	Zn added to the diet (ppm)	Tissue Fe concentration at the experimental period					
		10 days (ppm)	20 days (ppm)	30 days (ppm)	40 days (ppm)	50 days (ppm)	85 days (ppm)
Liver	0	56.2±10.5*	43.2±8.1*	50.1±13.9*	66.2±17.0*	97.3±25.7*	171.8±23.3*
	50	73.21 6.1	43.0± 6.4	61.4±13.7	60.0f 10.1	90.3±11.1 ²⁾	163.0±33.1
	250	60.3±19.4	43.2± 4.2	74.5±16.5	53.7±20.2	67.1±2.8 ²⁾	152.1±21.0
	1000	79.91-19.2	52.4± 7.6	74.8t10.6	46.4±10.8	49.2, 4.21'	147.2±39. a
Pancreas	0	14.1±1.6	14.3±1.7	12.31 1.8	10.4± 2.1	11.7± 2.1	10.1± 1.1
	50	16.1±3.2	13.3±1.9	11.6± 1.7	10.7-c 0.9	13.0±0.6	9.5± 2.0
	250	17.3± 3.5	13.6±1.2	10.3± 2.1	11.5± 3.3	11.9± 1.9	8.6± 1.1
	1000	30.3±13.9 ²⁾	12.0± 2.8	10.9± 1.7	10.6± 1.3	10.3± 1.1	9.1± 0.8
Testes	0	114.9±45.3	15.7±6.3	14.0± 0.5	14.1± 4.1	11.5± 1.9	6.9± 0.5
	50	125.9±58.4	34.7111.7	20.0± 0.4	19.1± 4.3	9.8± 1.4	4.2± 1.0
	250	123.8t42.8	38.6± 5.9	22.4± 0.8	19.0f 2.7	8.4± 0.6 ²⁾	4.7± 0.8
	1000	120.3±40.6	43.7± 9.9	23.4± 1.3	19.0± 5.8	7.7± 0.87'	5.7± 0.4
Kidneys	0	28.5± 5.0	39.8± 5.3	48.0± 8.4	41.1±8.9	47.1± 5.1	73.2t10.5
	50	23.3± 7.4	34.9± 7.8	47.3± 3.7	46.2± 5.3	56.3± 8.9	79.7± 7.7
	250	21.3± 8.4	36.0± 3.7	42.7± 5.9	46.9± 6.0	53.0± 5.1	70.7± 8.0
	1000	40.4± 5.0	32.3± 4.2	45.0± 6.2	45.3±11.3	42.01 3.9	67.0t13.8
Spleen	0	52.8± 8.4	66.1111.9	88.5i11.6	71.7± 8.7	114.6±10.7	144.3f20.5
	50	71.9-t 6.6	69.5± 5.1	67.9-t 14.7	88.1±15.1	109.3±12.7	146.7±22.5
	250	80.1±18.2	67.9± 9.9	67.9±11.1	68.3± 9.7	103.6f23.6	114.8k12.9
	1000	101.3±20.9 ²⁾	71.2±11.6	72.4±12.6	72.3k15.1	102.4±24.1	150.3t15.8
Heart	0	32.1± 3.1	34.9± 3.1	37.7± 2.1	31.4± 2.4	45.8± 6.6	39.1± 2.3
	50	35.0f 2.2	34.9± 3.0	33.0± 4.6	35.3± 2.5	41.5± 6.8	39.3± 4.1
	250	36.2± 5.2	34.2± 2.7	38.2± 4.0	31.8± 3.6	37.9, 3.0	38.3± 4.2
	1000	38.8± 6.8	34.5± 2.7	31.2± 2.9	32.1± 3.2	34.9± 2.0 ²⁾	38.5± 2.8
Brain	0	11.3± 0.9	13.0, 0.9	10.4± 2.3	10.4± 1.0	13.3± 0.1	14.1± 0.7
	50	11.3± 1.8	13.0± 0.8	11.2± 1.5	10.0± 0.6	15.8± 0.1	13.8± 0.5
	250	11.6± 1.4	13.0± 0.8	14.0± 1.2	10.9± 0.9	14.0± 1.9	14.1± 1.2
	1000	14.2± 1.6	11.7± 1.8	13.6± 1.5	14.9± 0.6	14.9± 0.6	13.8± 0.8
Breast muscle	0	9.7± 3.9	6.0± 0.9	5.7± 1.1	4.9± 0.3	5.9± 0.5	3.8± 0.7
	50	11.21 2.6	5.2± 0.4	5.1± 1.0	4.2± 0.1	5.2± 0.3	4.6-f 0.9
	250	12.3± 2.8	5.4, 0.5	4.7± 1.0	5.0± 0.3	6.5± 0.4	3.9± 0.9
	1000	15.0± 6.7	6.0± 0.8	4.7± 0.5	4.6± 0.2	5.8± 0.9	3.9± 0.9
Leg muscle	0	12.2± 2.1	12.5± 1.2	9.9± 0.8	7.8± 0.8	8.5± 0.3	9.1± 0.9
	50	12.6± 4.3	11.5± 1.2	9.2± 0.8	13.6, 1.7	8.5, 0.2	7.9, 0.7
	250	12.6± 2.1	10.8± 1.4	9.4± 1.5	8.7± 0.5	7.7± 0.2	6.4± 0.5
	1000	17.3± 3.5	12.3± 1.0	10.7± 0.5	9.8± 1.6	8.9± 0.6	8.8± 1.7

* Mean value±standard error. ¹⁾ Significantly different ($p < 0.01$) from control group by analysis of variance. ²⁾ Significantly different ($p < 0.05$) from control group by analysis of variance.

with increases in the concentration of Zn in the liver, pancreas, testes, kidneys and spleen at 10, 20, 30, 40 and 50 days of age (Table 1). At 85 days of age, no significant differences in the concentration of Zn in the liver were found between groups. In the pancreas, Zn concentration was declined with the levels of 1000 and 250 ppm dietary Zn at the same age. The reduction of

Table 3. Effect of dietary Zn levels on Cu concentration in liver, kidneys, heart and brain.

Tissue	Zn added to the diet (ppm)	Tissue Cu concentration at the experimental period					
		10 days (ppm)	20 days (ppm)	30 days (ppm)	40 days (ppm)	50 days (ppm)	85 days (ppm)
Liver	0	4.3±0.5*	3.6±1.3*	3.7±1.0*	3.9±0.2*	3.3±0.2*	4.3±1.5*
	50	5.0±0.9	4.1±0.1	4.3±0.6	4.2±0.8	3.4±0.5	2.3±0.1
	250	4.8±0.5	3.9±0.5	4.8±0.6	4.1±0.4	3.4±0.3	4.2±0.4
	1000	4.7±0.9	4.0±0.4	5.6±0.9	4.4±0.3	3.8±0.4	4.7±0.4
Kidneys	0	Traces	1.9±0.3	2.2±0.2	2.4±0.2	2.4±0.0	2.6±0.2
	50	Traces	2.0±0.1	2.4±0.1	2.8±0.1	2.7±0.3	2.6±0.2
	250	Traces	2.1±0.1	2.7±0.5	2.5±0.2	2.5±0.3	2.8±0.2
	1000	Traces	2.4±0.5	2.4±0.4	2.8±0.1	2.6±0.4	3.2±0.6
Heart	0	3.3±0.3	3.2±0.1	3.5±0.1	3.1±0.1	2.8±0.4	3.3±0.3
	50	4.0±0.0	3.1±0.1	3.6±0.2	3.4±0.2	2.7±0.1	2.8±0.8
	250	3.5±0.6	3.1±0.2	3.5±0.1	3.3±0.1	3.1±0.2	3.2±0.8
	1000	3.7±1.5	3.2±0.1	3.9±0.2	3.4±0.3	3.3±0.2	3.2±0.2
Brain	0	1.8±0.9		2.2±0.4	1.9±0.2	1.5±0.4	1.8±0.2
	50	2.0±0.2	2.1±0.2	2.0±0.3	1.8±0.3	1.7±0.1	1.7±0.1
	250		1.9±0.3				
	1000	1.6±0.2±0.24	2.0±0.34	2.2±0.30±0.4	3.3±0.17±0.43	1.8±0.21±0.2	1.4±0.41±0.3

* Mean value±standard error. All variances showed no significance from the control values by statistical analysis.

Zn concentration was significant only with the level of 250 ppm Zn. In contrast, the concentration of Zn in the pancreas with 50 ppm dietary Zn was increased significantly by 62% over those of the basal control group. The concentration of Zn in the testes confirmed the same trend, but the magnitude was larger than in the pancreas. It is noteworthy that the testes contained the highest concentration of Zn among all organs tested at 10 days of life and contained the lowest concentration at 85 days. In the kidneys, Zn concentration was the same for the three groups receiving dietary Zn at 85 days. The three groups did not differ significantly from the control group. Zn concentration in the heart was not influenced by the dietary Zn levels at 20, 30, 40, 50 and 85 days of age. The levels of Zn in the heart of cockerels receiving dietary Zn was essentially the same as for cockerels fed the basal diet. No significant changes were noted in the concentration of Zn in the other tissues by increasing dietary Zn levels.

As the levels of dietary Zn were increased, a corresponding increase in Fe concentration in the liver was observed at 10 days of age but the differences between groups were insignificant. The dietary Zn levels appeared to be antagonistic to Fe concentration in the liver. The antagonistic effect was observed at 40, 50 and 85 days of age but the data were statistically significant only at 50 days.

In the pancreas, there was an increase in the concentration of Fe with the increase in the dietary Zn levels at 10 days of age. Accompanying the increase in the dietary Zn, smaller decreases were evident at 20, 30, 40, 50

and 85 days.

A slight increase in the Fe concentration was found in the testes by increasing the levels of dietary Zn at 10, 20, 30 and 40 days, while at 50 and 85 days, Fe concentration decreased as the levels of dietary Zn was increased. The decrease in Fe concentration was statistically significant with the levels of 1000 and 250 ppm dietary Zn at 50 days.

At 10 days of age, variations between groups occurred in the Fe concentration in the kidneys. The cockerels fed 1000 ppm dietary Zn contained more Fe concentration in the kidneys than the cockerels fed the control diet, whereas the levels of 50 and 250 ppm Zn resulted in reduction in the Fe concentration as compared with the control group. These changes were not statistically significant. The antagonistic effect of dietary Zn in the kidneys was occurred from 20 days of age. At 50 and 85 days, the antagonistic effect was observed only with the level of 1000 ppm Zn, while the levels of 250 and 50 ppm Zn at 50 days and the level of 50 ppm Zn at 85 days caused an increase over those of the control group, though statistical significance was not attained.

In the spleen and heart, there were an increase in Fe concentration with the increase in the dietary Zn levels at 10 days of age, but the only significant elevation was noted with the level of 1000 ppm Zn. A trend of lower concentration of Fe was observed in these organs as the levels of dietary Zn was increased at 30 and 50 days, but Fe concentration in the heart was significantly decreased with the levels of 1000 ppm Zn at 50 days of life.

None of the dietary treatments of Zn had any significant effect on Fe concentration of the brain, breast muscle and leg muscle during all the experimental periods.

The Cu concentrations of the liver, kidneys, heart and brain were not affected by the addition of Zn to the diet and by duration of feeding. Whereas, the Cu concentration of the testes, pancreas, spleen, breast muscle and leg muscle was traces and there were undetectable changes with dietary Zn levels,

In order to prove more sufficiently the relationship between concentration of Zn or Fe in tissues and dietary Zn, regression analyses on the Zn or Fe values in tissues versus the dietary Zn level and age were carried out. The present finding, where statistical significant relationships or trend were existed, are given in Tables 4, 5 and 6.

Analysis revealed highly significant ($p < 0.01$) positive linear regression of dietary Zn level on Zn concentration in the liver at 10, 30 and 40 days of age and significant ($p < 0.05$) at 20 and 50 days. But, it should be noted that the Zn concentration in the liver for the group receiving 50 ppm dietary Zn was higher than the corresponding values for the group receiving 250 ppm dietary Zn at 40 and 50 days (Table 1).

The linear regressions of dietary Zn level on Zn concentration in the pancreas were highly significant ($p < 0.01$) at 10, 20, 30 and 50 days of age and significant ($p < 0.05$) at 40 days. With the two higher levels of dietary Zn, there was a significant ($p < 0.01$) negative linear regression of Zn concentration in the pancreas and duration of feeding (Table 5).

Table 4. Correlation coefficients between dietary Zn concentration and Zn concentration in selected organs.

Period (days)	Correlation coefficients								
	Liver	Pancreas	Testes	Kidneys	Spleen	Heart	Brain	Breast muscle	Leg muscle
10	0.994 ¹⁾	0.986 ¹⁾	0.937 ²⁾	0.963 ¹⁾	0.83 ⁵	-0.422	0.768	0.9113 ⁷	0.717
20	0.929 ³⁾	0.979 ¹⁾	0.973 ³⁾	0.992 ¹⁾	0.968 ¹⁾	0.241	0.745	0.359	0.9333 ⁷
30	0.988 ¹⁾	0.971 ¹⁾	0.973 ³⁾	0.986 ¹⁾	0.725	0.887 ³⁾	0.954 ¹⁾	0.113	0.644
40	0.963 ³⁾	0.8813 ⁷	0.944 ²⁾	0.928 ³⁾	0.763	0.449	0.293	0.787	0.701
50	0.8793 ⁷	0.973 ⁷	0.802	0.769	-0.127	-0.117	0.641	-0.411	-0.587
85	0.813	0.381	0.764	0.436	-0.995 ⁷	-0.8 ² 24	0.614	0.346	0.608

^{1), 2), 3)} Significant at 0.01, 0.02 and 0.05 level, respectively.

Table 5. Correlation coefficients between dietary Zn concentration and tissue Zn concentration during the experimental period.

Zn added to the diet (ppm)	Correlation coefficients																	
	Liver	Pancreas	Testes	Kidneys	Spleen	Heart	Brain	Breast muscle	Leg muscle									
0																		
50	-0.539	-0.301	-0.446	-0.402	-0.9051 ¹⁾	-0.878 ¹⁾	-0.439	-0.177	-0.642	-0.539	-0.506	-0.574	-0.456	-0.645	-0.138	-0.632	-0.688	-0.677
250	-0.543	-0.945 ¹⁾	-0.872 ¹⁾	-0.371	-0.845 ²⁾	-0.657	-0.313	-0.658	-0.736									
1000	-0.596	-0.941 ⁷	-0.845 ²⁾	-0.686	-0.844 ²⁾	-0.667	-0.689	-0.688	-0.638									

^{1), 2)} Significant at 0.01 and 0.02 level, respectively.

Table 6. Correlation coefficients between dietary Zn concentration and Fe concentration in selected organs.

Period (days)	Correlation coefficients								
	Liver	Pancreas	Testes	Kidneys	Spleen	Heart	Brain	Breast muscle	Leg muscle
10									
20	0.718	0.987 ⁷	-0.232	0.841	0.912 ³⁾	0.8833 ¹⁾	0.992 ¹⁾	0.957 ²⁾	0.982 ¹⁾
	0.974 ¹⁾	-0.9143 ⁷	0.704	-0.795	-0.788	-0.504	0.9681	0.394	0.271
30	0.713	-0.490	0.698	-0.463	-0.269	-0.649	0.675	-0.658	0.839
40	-0.909 ³⁾	-0.305	0.456	-0.254	-0.350	-0.312	0.990 ⁷	-0.282	-0.920 ³⁾
50	-0.9243 ⁷	-0.861	-0.788	-0.723	-0.677	-0.856	0.243	0.135	0.475
85	-0.830	-0.445	0.515	-0.779	-0.246	-0.614	-0.349	-0.263	0.185

^{1), 2), 3)} Significant at 0.01, 0.02 and 0.05 level, respectively.

Zn concentration in the testes showed positive linear relationship ($p < 0.02$) with the level of dietary Zn at 10 and 40 days of age and significant ($p < 0.05$) at 20 days. It showed negative relationship between the duration of feeding and all dietary Zn levels (Table 5). Zn concentration in the kidneys increased with increasing dietary Zn levels. The response was highly significant ($p < 0.01$) at 10, 20 and 30 days and significant ($p < 0.05$) at 40 days of age. A positive linear regression between dietary Zn level and Zn concentration in the spleen was found to be highly significant ($p < 0.01$) at 20 days.

Significant ($p < 0.01$) linear increase in Fe concentration of the liver at 20 days and in the pancreas at 10 days occurred due to increasing Zn in the diet (Table 6). Significant ($p < 0.05$) linear decrease in Fe concentration in the liv-

er occurred at 40 and 50 days of age when the level of Zn in the diet increased. The same trend occurred at 85 days but was not significant. In the pancreas, the only significant ($p < 0.05$) negative relationship between the levels of dietary Zn and the concentration of Fe was noted at 20 days of age.

DISCUSSION

In the present study, the apparent linearity of the relationship between Zn concentration in the liver, pancreas, testes and kidneys and the levels of dietary Zn supplementation indicated that additional Zn would have accumulated with increasing the levels of Zn in the diet. Whereas, the lack of significant linearity in all tissues at 85 days of life suggested that after long term feeding, the homeostatic mechanism can prevent the tissue content from remaining at the initially increased level. Stake *et al.* (1975) have concluded, however, that no homeostatic adaptation was detected in the liver, pancreas and kidneys of calves during 21 days period by 600 ppm dietary Zn. The same diet did not materially affect the Zn content of the liver and kidneys in rats suggesting that the homeostatic mechanism of Zn are much more effective in rats (Ansari *et al.*, 1976). From the result of the present study it is evident that fowl is similar to rats rather than to calves in their response to dietary Zn Supplementation.

There are several factors which may have contributed to the contradictory results in the literature. One of these factors to be considered is age of animals. In this context the finding of Miller and Cragle (1965) that Zn absorption decreases as cattle get mature and the accumulation of Zn is probably related to absorption. In the present study, examination of the relationship between dietary Zn and tissue Zn at the duration of feeding suggested that the pancreas and testes showed negative correlation. This indicated that there was no tendency for the cockerel to store Zn in the pancreas with the two highest levels and in the testes with all dietary Zn levels. These findings are in agreement with the study of Kincaid *et al.* (1976), who reported Zn homeostatic control mechanism protected the tissue, for at least limited periods of time, against increased Zn in tissues from dietary Zn up to 1200 ppm in the chick. In contrast, Zn concentration was unaffected in certain other tissues especially heart, brain and muscle indicating complete homeostasis (Stake *et al.*, 1975).

The concentration of Fe in the liver, testes, kidneys, spleen and heart also tended to be influenced by Zn intake. In the fact, Fe concentration in these organs tended to be lowest when the dietary Zn was highest, suggesting noteworthy relationship between Zn and Fe. The decrease in Fe concentration in the present study with high dietary Zn may seem at variance with the result of Ott *et al.* (1966) who found an increase in Fe of the liver of cattle fed high levels of dietary Zn for 10 weeks periods. In rats, Cox and Harris (1960) reported a high inverse correlation between Fe concentration in the liver and kidneys and dietary Zn levels of rat fed high Zn diet. This relationship was detected after 7 days, whereas in the present study the antag-

onistic effect was detected from the 40th day of age. The effect on fowl would appear to be rather similar to that in rat than that in cattle but fowl appear to be somewhat more resistant to the antagonistic effect. From the result of this study, it is evident that the 50 ppm Zn added to the diet induced an increase in Zn concentration in the liver, kidneys, pancreas and spleen, but could not induce any reduction in Fe concentration. This result is in agreement with the proposal that Zn level in these organs must attain a critical level before interfering with the Fe absorption. In view of this proposal, the Zn level in the liver, spleen and heart with high Zn intake did not attain a critical level to affect Fe content until 40 to 50 days in the present study.

Cox and Harris (1960) have reported that rats fed high levels of dietary Zn exhibited an early loss of the liver Fe, and after a relatively long period of time also exhibited a reduction in liver Cu. The data suggested that the reduction of liver Cu was a result of reduced liver Fe rather than an effect of liver Zn. In contrast, in the present study, the results showed that Cu concentration in the liver and heart was not changed in the fowl by any dietary treatment throughout the experimental period. In support of the present finding Johnson *et al.* (1962) reported in the fowl that the concentration of Cu in the liver was not affected by several graded level of dietary Zn, up to 4000 ppm Zn. Another possible explanation to account *is* that the level of Fe was not reduced to the critical level to affect Cu metabolism (Cox and Harris, 1962).

ACKNOWLEDGMENT

The authors wish to express appreciation to prof. K. Wada, Laboratory of Soil Science, Kyushu University for the use of atomic absorption spectrophotometer.

REFERENCES

- Ansari, M. S., W. J. Miller, M. W. Neathery, J. W. Lassiter, R. P. Gentry and R. L. Kincaid 1976 Zinc metabolism and homeostasis in rats fed a wide range of high dietary Zn levels. *Proc. Soc. Exp. Biol. Med.*, 152: 192-194
- Cox, D. H. and D. L. Harris 1960 Effect of excess dietary zinc on iron and copper in the rat. *J. Nutr.*, 70: 514-521,
- Cox, D. H. and D. L. Harris 1962 Reduction of liver xanthine oxidase activity and iron storage proteins in rats fed excess zinc. *J. Nutr.*, 78: 415-419
- Eltohamy, M. M., H. Takahara and M. Okamoto 1979 Effects of dietary Zn levels on the histological changes produced in White Leghorn cockerels. *J. Fac. Agr., Kyushu Univ.*, 24: 11-20
- Johnson, D. J. R., A. L. Mehring, Jr., F. X. Savino and H. W. Titus 1962 The tolerance of growing chickens for dietary zinc. *Poult. Sci.*, 41: 311-317
- Kincaid, R. L., W. J. Miller, L. S. Jensen, D. L. Hampton, M. W. Neathery and R. P. Gentry 1976 Effect of high amounts of dietary zinc and age upon tissue zinc in young chicks. *Poult. Sci.* 55: 1954-1957
- Miller, W. J., D. M. Blackmon, R. P. Gentry and F. M. Pate 1970 Effects of high but non-

- toxic levels of zinc in practical diets on ^{65}Zn and zinc metabolism in Holstein calves. *J. Nutr.*, 100 : 893-902
- Miller, J. K. and R. G. Cragle 1965 Gastrointestinal sites of absorption and endogenous secretion of Zn in dairy cattle. *J. Dairy Sci.*, 48: 370-374
- Ott, E. A., W. H. Smith, R. B. Harrington, H. E. Parker and W. M. Beeson 1966 Zinc toxicity in ruminants 3. Physiological changes in tissues of beef cattle. *J. Anim. Sci.*, 25: 419-423
- Settlemyre, C. T. and G. Matrone 1976 In vivo interference of zinc with ferritin iron in the rat. *J. Nutr.*, 92: 153-158
- Smith, S. E. and E. J. Larson 1946 Zinc toxicity in rats. Antagonistic effects of copper and liver. *J. Biol. Chem.*, 163: 29-31
- Snedecor, G. W. 1956 *Statistical Methods*. 5th ed. Iowa State Univ. Press, Ames., Iowa.
- Stake, P. E., W. J. Miller and R. P. Gentry 1973 Zinc metabolism and homeostasis in ruminants as affected by dietary energy intake and growth rate. *Proc. Soc. Exp. Biol. Med.*, 142: 494-496
- Stake, P.E., W. J. Miller, R. P. Gentry and M. W. Neathery 1975 Zinc metabolism adaptation in calves fed a high but nontoxic zinc level for varying time periods. *J. Anim. Sci.*, 40: 132-137
- Van Campen, D. R. and R. U. Scalfe 1967 Zinc interference with copper absorption in rats. *J. Nutr.*, 91: 473-476