Effect of Dietary Cadmium Levels on Nutrient Digestibility and Retention of Iron, Copper and Zinc in Tissues of Growing Pigs

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ABSTRACT : This experiment was conducted to investigate the effect of cadmium levels on weight gain, nutrient digestibility and the retention of iron, copper and zinc in tissues of growing pigs. A total of one hundred and ninety-two crossbred pigs (barrows, Duroc×Landrace×Yorkshine, 27.67±1.33 kg of average initial body weight) were randomly allotted to four treatments. Each treatment had three replicates with 16 pigs per pen. The corn-soybean basal diets were supplemented with 0, 0.5, 5.0, 10.0 mg/kg cadmium respectively, and the feeding experiment lasted for eight-three days. Cadmium chloride was used as cadmium source. The results showed that pigs fed the diet containing 10.0 mg/kg cadmium had lower ADG and FCR than any other treatments ($p<0.05$). Apparent digestibility of protein in 10.0 mg/kg cadmium-treated group was lower than that of other groups ($p<0.05$). There was lower iron retention in some tissues of 5.0 mg/kg and 10.0 mg/kg cadmium treatments ($p<0.05$). However, pigs fed the diet 10.0 mg/kg cadmium had higher copper content in most tissues than that of any other groups ($p<0.05$). There was a significantly increase of zinc retention in kidney of 10.0 mg/kg cadmium additional group ($p<0.05$) and zinc concentrations in lymphaden, pancreas and heart of 10.0 mg/kg cadmium treatment were lower than those of the control ($p<0.05$). This study indicated that relatively high cadmium level (10.0 mg/kg) could decrease pig growth performance and change the retention of iron, copper and zinc in most tissues during extended cadmium exposure period. ***(Asian-Aust. J. Anim. Sci. 2004. Vol 17, No. 7 : 1007-1013)***

Key Words : Growing Pigs, Cadmium, Growth Performance, Nutrient Digestibility, Iron, Copper, Zinc

INTRODUCTION

Cadmium (Cd) is a trace element that has no known metabolic function (Crowe and Morgan, 1997). It is taken by mammals through the food chain and creates a health risk for both animals and humans. Furthermore, cadmium is an accumulative contaminant because the body has no homeostatic mechanism to keep cadmium at a constant safe level (Miller, 1971). Hence, the adverse effect of cadmium exposure comes from its high persistence in the body since the mean half-life of this poisonous heavy metal in the human body has been estimated to be 16-38 years (Cotzias et al., 1961; Fox, 1987). Cadmium has been shown to accumulate in almost all tissues of animals especially in liver and kidney (Matsubara-Khan and Machida, 1975; Kanwar et al., 1980; Eybl et al., 1998). The accumulation of cadmium in animal body would result in various disorders of the organs and threaten health of animal and human. Approximately, 1/3rd of cadmium dietary intake is attributed to the ingestion of animal products in human (Nasreddine and Parent-Massin, 2002).

In domestic animals, administration of a relatively high dosage of cadmium could decrease feed intake and weight gain and affect fertilizing function (Pond et al., 1966; Cousins et al., 1973; Czarnecki and Baker, 1982; Bafundo et al., 1984; Mohan et al., 1992). In general, these experiments about adverse effect of cadmium have been investigated with relatively high dosage and abbreviated exposure periods. Such experiments may not reflect the tolerance or condition of animals receiving more realistic exposures (Ammerman et al., 1977). Actually, a relatively low cadmium levels in animal feed may be occurred since more and more industrial processes related to cadmium have been developing. Very few experiments were conducted to approach the effect of relatively low cadmium levels on performance in domestic animals.

The interaction among essential and toxic metals is a significant aspect of trace metal metabolism. Many studies showed that cadmium could interfere with the transport and metabolism of many essential metals, such as iron, zinc and copper (Davies and Campbell, 1977; Webster, 1979; Coppen-Jaeger and Wilhelm, 1989; Kozlowska et al., 1993; Oishi et al., 2000). There is some evidence that several essential metals can inhibit cadmium absorption in the intestine (Foulkes, 1985). However, most studies have focused on a single metal-metal interaction, and the mechanisms of both cadmium absorption and the way in which other metals interfere with this process are still unclear. Generally, ample daily dietary intakes of iron, copper and zinc would be in domestic animal feeding at present. Very little work has been done in examining the interactions between iron, copper and zinc loading and cadmium exposure, and the effect of cadmium levels on nutrient digestibility in domestic animals.

The aim of this experiment was to study the effects of

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Table 1. Ingredients of the basal diet and their nutrient levels

Treatment	Body weight 27-50 kg	Body weight 50-80 kg	Body weight 80 kg or over
Ingredients (%)			
Corn	64.65	66.85	68.10
Soybean meal	20.0	19.0	13.0
Wheat bran	8.0	10.0	15.0
Yeast	3.0		
Fish meal	0.25	0.25	
$CaHPO_4$	1.50	1.25	1.10
Limestone	1.0	1.1	1.2
Salt	0.4	0.4	0.4
Lysine	0.2	0.15	0.2
Mineral/vitamin premix[a]	1.0	1.0	1.0
Estimated values[b]			
Digestible energy,MJ/kg[c]	13.86	13.73	13.65
Dry matter, %	87.8	86.7	86.6
Crude protein, %	18.3	15.9	13.8
Ether extract, %	1.9	2.0	2.0
Calcium, %	0.96	0.92	0.98
Total phosphorus, %	0.54	0.53	0.57

[a] Mineral/vitamin premix supplied per kilogram of complete diet: 20-40 kg: vitamin A, 2,500 IU; vitamin D_3, 300 IU; vitamin E, 20 IU; riboflavin, 4.6 mg; nicotinic acid, 20 mg; d-pantothenic acid, 15 mg; vitamin B_{12}, 0.02 mg; biotin, 0.1 mg; choline, 0.56 mg, $FeSO_4 \cdot 7H_2O$ 400 mg, $CuSO_4 \cdot 5H_2O$ 800 mg, $ZnSO_4 \cdot 7H_2O$ 360 mg, $MnSO_4 \cdot H_2O$ 72 mg. 40-80 kg: vitamin A, 3,500 IU; vitamin D_3, 400 IU; vitamin E, 30 IU; riboflavin, 5.2 mg; nicotinic acid, 20 mg; d-pantothenic acid, 18 mg; vitamin B_{12}, 0.02 mg; biotin, 0.15 mg; choline, 0.77 mg, $FeSO_4 \cdot 7H_2O$ 400 mg, $CuSO_4 \cdot 5H_2O$ 800 mg, $ZnSO_4 \cdot 7H_2O$ 300 mg, $MnSO_4 \cdot H_2O$ 72 mg. 80-120 kg: vitamin A, 4,000 IU; vitamin D_3, 500 IU; vitamin E, 40 IU; riboflavin, 6.2 mg; nicotinic acid, 22 mg; d-pantothenic acid, 22 mg; vitamin B_{12}, 0.02 mg; biotin, 0.15 mg; choline, 0.92 mg, $FeSO_4 \cdot 7H_2O$ 350 g, $CuSO_4 \cdot 5H_2O$ 400 mg, $ZnSO_4 \cdot 7H_2O$ 280 mg, $MnSO_4 \cdot H_2O$ 60 mg.

[b] Measured value ([c] Calculated value).

relatively low cadmium levels on weight gain, nutrient digestibility and retention of iron, copper and zinc in tissues of growing pigs during longer cadmium exposure period.

MATERIALS AND METHODS

Experiment 1

A feeding experiment was conducted using crossbred growing pigs. One hundred and ninety-two barrows (Duroc×Landrace×Yorkshire), weighing an average of 27.67±1.33 kg, were randomly assigned to four different treatments. Each of these groups consisted of three replications (i.e., pens) with sixteen pigs per replicate. The treatments received the same basal diet and supplemented with 0, 0.5, 5.0, 10.0 mg/kg cadmium (as $CdCl_2$) respectively, and three corn-soybean basal diets were used during experimental period (pig body weight from 27 kg to 90 kg). The content of cadmium in basal diets was 0.11 mg/kg and cadmium in drinking water was not found. Experimental diets were formulated to meet or exceed the nutrient requirement for growing and finishing pigs recommended by the NRC (1998). All diets were fed as mash and the composition of diets are presented in Table 1.

The feeding trial was conducted in Zhejiang China and lasted for eighty-three days after seven days of adaptation period. All pigs were housed in an open-front pig barn with concrete floor and the size of the pens used was 350 cm×350 cm. A Dry/wet feeders with two waterers was allocated in each pen for growing pigs and the pigs were allowed *ad libitum* access to feed and water.

Experiment 2

For the determination of nutrient digestibility, the total fecal collection method was used. The 24 barrows weighing about 86 kg were kept in individual pens, and given their respective experimental diets, six pigs per diet. Feed and water were available *ad libitum*. Collection of fecal material was undertaken through three consecutive days, after an adaptation period of 1 week. The amount of feed consumed and feces were recorded daily. Feces were collected at least four times daily and stored in closed plastic containers at 4°C during the collection period. At the end of this period, collected excreta were mixed thoroughly and a 500 g sample was taken from respective homogenized fecal sample and were dried in a drying oven at 70°C and ground in a Wiley Mill to pass through a 1 mm screen prior to chemical analysis.

Sampling for the feeding study

Feed intake was recorded per pen for the feeding study. At the start and the end of the study, body weight of each animal was recorded to determine average daily gain (ADG) and feed consumption for each pen.

At the end of the feeding trial, four pigs from each pen were randomly selected based on similar body weight and slaughtered. Blood samples were centrifuged at 3,000

Table 2. Growth performance of growing pigs (Experiment 1)[1]

Item	Cadmium/(mg·kg^{-1})				SEM[2]
	0	0.5	5.0	10.0	
Initial wt (kg)	27.76	27.67	27.63	27.63	0.19
Final wt (kg)	89.35^{a}	89.17^{a}	87.50^{a}	77.75^{b}	1.16
ADG (g)	741.97^{a}	740.96^{a}	721.39^{a}	603.92^{b}	13.35
ADFI (kg)	2.23^{a}	2.22^{a}	2.20^{a}	2.14^{b}	0.02
F/G	3.03^{b}	3.03^{b}	3.06^{b}	3.57^{a}	0.01

[1] Means within a row with different superscripts differ significantly ($p<0.05$). [2] Standard error of mean.

Table 3. Apparent digestibility of nutrients (Experiment 2)[1] (%)

Item	Cadmium/(mg·kg^{-1})				SEM[2]
	0	0.5	5.0	10.0	
Dry matter	87.98^{a}	87.88^{a}	85.98^{a}	77.98^{b}	0.70
Crude protein	85.85^{a}	85.77^{a}	83.52^{a}	75.25^{b}	0.80
Ether extract	72.40	72.38	71.77	69.95	0.84
Calcium	63.08	63.05	62.17	60.67	0.83
Total phosphorus	65.45	65.42	64.33	62.98	0.84

[1] Means within a row with different superscripts differ significantly ($p<0.05$). [2] Standard error of mean.

rpm/min for 10 min, and serum was separated and packed in Eppendorf tubes respectively. The samples of renal cortex, renal medulla, liver, heart, pancreas, bile, spleen and lymphaden in intestine mesentery were collected and packed in plastic bags. All samples were stored at -70°C until required for analysis.

Chemical analysis of diets and feces

The proximate analyses of the composition of the experimental diets and excreta were carried out according to the methods of AOAC (1990). Calcium was determined by method of titration with 0.1 N EDTA. Total phosphorus was determined colorimetrically using a molybdovandate reagent with a UV-visible spectrophotometer (Ultrospec. 2000, Sweden) (AOAC, 1990). The cadmium analyses of diets and water were performed by atomic absorption spectrophotometry (AAS) with graphite furnace technique. The iron, copper and zinc analyses in tissues were determined by AAS with flame technique (Shimadzu AA 6501, Japan)(Wang, 1994).

Statistical analysis

All data measured in the study were analyzed by comparing means according to least significant difference test, using the general linear model procedure of SAS (version 6.12). In Exp 1, pens were considered the experimental unit for each analysis. In Exp 2, nutrients digestibility was analyzed with the individual pig as the experimental unit, as well as serum and tissue minerals. A significant level of 0.05 was used.

RESULT AND DISCUSSION

Growth performance

Pig growth performance is shown in Table 2. Compared to the control, the addition of 10.0 mg/kg cadmium to the diet resulted in 18.6% ($p<0.05$) decrease in ADG, and there was no significant difference among the pigs fed diets containing 0, 0.5, 5.0 mg/kg cadmium.

ADFI of 10.0 mg/kg cadmium treatment was lower than that of other groups ($p<0.05$), and there was no effect of 0.5 and 5.0 mg/kg cadmium on ADFI compared with the control ($p>0.05$).

F/G, namely the efficiency of feed utilization, was significantly affected by relatively high cadmium levels. F/G for pigs fed 10.0 mg/kg cadmium-treated diet increased by 17.8% in comparison with the control ($p<0.05$). There was no significant effect of 0.5 and 5.0 mg/kg cadmium on the feed conversion ratio compared to the control.

Previous studies demonstrated that high cadmium levels in pig diet decreased their feed intake and weight gain after 4 week or 6 week of feeding (Pond et al., 1966; Cousins et al., 1973; Pond and Walker, 1973; Hansen and Hinsley, 1979; Anke et al., 1989). The cadmium levels supplemented in pig diet were 50, 150, 154, 450, 1,350 mg/kg, and pig growth was arrested at the 1,350 mg/kg cadmium level. Raszy et al. (1992) found that the pigs from a farm located in a lignite mining area and near solid fuel power plant also showed higher feed consumption per 1 kg weight gain and lower average daily weight gain. However, there was no adverse effect of a diet containing 0.24 mg/kg cadmium, which was from cadmium-polluted corn, on growth performance during growing and finishing period (Lisk et al., 1982). It appears that pig growth has relation to dietary cadmium level. In the present study, we found that relatively lower cadmium content in the diet also had adverse effect on porcine weight gain after 3 month experimental period. This effect is similar to that of high cadmium dosage in pig diet in abbreviated exposure periods. Cadmium toxicity in animals is a function of dose and

Table 4. Iron retention in tissues of growing pigs[1]

Item	Cadmium/(mg·kg^{-1})				SEM[2]
	0	0.5	5.0	10.0	
Liver/μg·g^{-1}	70.33^{a}	68.24^{ab}	61.62^{b}	39.93^{c}	2.49
Renal cortex/μg·g^{-1}	48.97^{a}	47.44^{a}	42.79^{b}	29.85^{c}	1.62
Heart/μg·g^{-1}	32.82	32.78	32.26	31.98	0.46
Pancreas/μg·g^{-1}	10.94^{a}	10.32^{ab}	9.59^{b}	6.83^{c}	0.29
Bile/μg·g^{-1}	8.08^{a}	7.96^{a}	7.54^{a}	5.14^{b}	0.26
Spleen/μg·g^{-1}	103.53^{a}	103.53^{a}	92.41^{b}	67.98^{c}	3.08
Lymphaden/μg·g^{-1}	13.50	13.41	13.34	13.04	0.45
Serum/(μg·ml^{-1})	35.87^{a}	34.23^{a}	32.31^{b}	29.18^{c}	0.60

[1] Means within a row with different superscripts differ significantly ($p<0.05$). [2] Standard error of mean.

duration of exposure. Tolerances will be highly dependant on total accumulated body burden and on hepatic and renal function during exposure (Ammerman et al., 1977). Our results also indicated the accumulative toxicity at relatively low dosage during longer cadmium exposure.

Apparent digestibility of nutrient

The apparent digestibility of nutrients is shown in Table 3. The apparent digestibility of dry matter and crude protein were significantly lower in 10.0 mg/kg cadmium added group than that of other groups ($p<0.05$), and there was no differences in the apparent digestibility among pigs fed diets containing 0, 0.5 and 5.0 mg/kg Cd ($p>0.05$). The apparent digestibility of crude fat, calcium and total phosphorus were similar among the groups ($p>0.05$).

Little is known about the effect of cadmium on nutrient digestibility in animal. Studies indicated that cadmium could inhibit the sulfhydryl enzyme systems necessary for cellular metabolism and has a high affinity to biological structures such as proteins and enzymes containing-SH groups, the affinity of cadmium to S-ligands as well as to N-donors is greater than that of zinc (Jacobson and Turner, 1980; Jones and Cherian, 1990). Thus, the activities of digestive enzymes in gastrointestinal content may be affected by extended cadmium exposure, which may lead to change of nutrient metabolism and digestibility. Further research may be needed to study mechanism about the effects of low dosage cadmium exposure during extended period on nutrient digestibility of animals.

This result suggests that the decrease of apparent digestibility of nutrients may be one of the reasons that resulted in decrease of growth performance in 10.0 mg/kg Cd-treated group.

Iron, copper and zinc retention in tissues

Tissue iron levels of 5.0 mg/kg and 10.0 mg/kg Cd-treated groups were significantly lower than that of the control ($p<0.05$), and there was no significant difference among the groups in lymphaden and heart. No differences of tissues iron levels were found between 0.5 mg/kg cadmium-treated group and the control ($p>0.05$)(Table 4).

It has been known that cadmium can decrease the intestinal absorption of iron, with many studies showing the reduction of iron retention in different tissues, when animals are subjected to dietary cadmium loading (Pond et al., 1973; Hamilton and Valberg, 1974; Kozlowska et al., 1993; Elsenhans et al., 1994). They also provided evidence that cadmium interferes directly with iron absorption through the intestine, possibly by competing with iron in the absorptive process. It was found that dietary iron loading could protect animals from cadmium-induced anemia (Banis et al., 1969; Pond et al., 1973). However, Crowe and Morgan (1997) suggested that cadmium inhibited iron absorption only at low to normal levels of dietary iron and that at high levels of intake iron and cadmium are largely absorbed by other, noncompetitive mechanisms in rats.

However, in the present study, 10.0 mg/kg cadmium could lower iron concentrations in tissues including serum, liver, kidney, pancreas, bile and spleen in adequate dietary iron conditions. The mechanism behind the competition of cadmium and iron is still not understood. Schafer and Forth (1984) postulated that cadmium competes with the iron transfer system mainly by binding to mucosal transferrin, an important determinant in iron uptake in the intestine. Other studies pointed out that it is possible for the uptake of small amounts of cadmium to involve binding to intestinal ferritin, although the level of cadmium binding to ferritin is low compared with the binding of iron (Fox et al., 1980; Joshi and Zimmerman,1988). Reduction of iron uptake could be the result of toxic effects of cadmium on the cell membrane or energy metabolism (Davies et al., 1977; Miccadei et al., 1993; Koizumi et al., 1994).

Pigs that received the diet containing10.0 mg/kg cadmium had higher copper concentration in liver, kidney, spleen, bile and heart ($p<0.05$) and there was no difference among other pigs fed the diets supplemented 0, 0.5, 5.0 mg/kg cadmium respectively ($p>0.05$). There was no effect of cadmium on pancreas and lymphaden copper level in the present experiment ($p>0.05$) (Table 5).

There are disagreements about the interaction between copper and cadmium. Adverse effects of cadmium on iron.

Table 5. Copper retention in tissues of growing pigs[1]

Item	Cadmium/(mg·kg^{-1})				SEM[2]
	0	0.5	5.0	10.0	
Liver/μg·g^{-1}	7.90^{b}	7.95^{b}	8.53^{b}	17.93^{a}	0.31
Renal cortex/μg·g^{-1}	7.68^{b}	7.73^{b}	7.93^{b}	17.59^{a}	0.30
Heart/μg·g^{-1}	1.60^{b}	1.63^{b}	1.73^{b}	2.21^{a}	0.06
Pancreas/μg·g^{-1}	2.59	2.64	2.69	2.77	0.07
Bile/μg·g^{-1}	3.36^{b}	3.41^{b}	3.53^{b}	4.35^{a}	0.11
Spleen/μg·g^{-1}	1.67^{b}	1.71^{b}	1.80^{b}	2.54^{a}	0.05
Lymphaden/μg·g^{-1}	0.87	0.88	0.91	0.93	0.02
Serum/(μg·ml^{-1})	8.96^{b}	9.02^{b}	9.57^{b}	12.27^{a}	0.28

[1] Means within a row with different superscripts differ significantly ($p<0.05$). [2] Standard error of mean.

Table 6. Cadmium retention in tissues of growing pigs[1]

Item	Cadmium/(mg·kg^{-1})				SEM[2]
	0	0.5	5.0	10.0	
Liver/μg·g^{-1}	0.13^{c}	0.42^{c}	2.57^{b}	6.94^{a}	0.15
Renal cortex/μg·g^{-1}	0.45^{c}	1.64^{c}	15.86^{b}	33.01^{a}	0.46
Renal medulla/μg·g^{-1}	0.19^{c}	0.68^{c}	6.09^{b}	13.87^{a}	0.24
Spleen/μg·g^{-1}	0.05^{c}	0.11^{c}	0.63^{b}	1.62^{a}	0.03
Lymphaden/μg·g^{-1}	0.08^{c}	0.19^{c}	1.66^{b}	3.64^{a}	0.10

[1] Means within a row with different superscripts differ significantly ($p<0.05$). [2] Standard error of mean.

Table 7. Zinc retention in tissues of growing pigs[1]

Item	Cadmium/(mg·kg^{-1})				SEM[2]
	0	0.5	5.0	10.0	
Liver/μg·g^{-1}	46.68	47.09	47.92	48.67	0.96
Renal cortex/μg·g^{-1}	27.89^{b}	28.87^{b}	29.91^{b}	33.67^{a}	0.79
Heart/μg·g^{-1}	18.12^{a}	18.10^{a}	18.02^{a}	16.62^{b}	0.30
Pancreas/μg·g^{-1}	34.91^{a}	34.41^{a}	32.89^{a}	27. 20^{b}	0.73
Spleen/μg·g^{-1}	22.96	22.87	22.42	22.26	0.38
Lymphaden/μg·g^{-1}	13.26^{a}	13.22^{a}	13.13^{a}	12.36^{b}	0.25
Serum/(μg·ml^{-1})	10.82	10.79	10.63	10.18	0.26

[1] Means within a row with different superscripts differ significantly ($p<0.05$). [2] Standard error of mean.

copper and zinc metabolism had been reported (Ammerman et al., 1977). Reduced copper content in fetal liver, placenta and fetal intestine were also found in rats after gestational exposure to oral cadmium (50 μg/ml Cd in drinking water) (Steibert et al., 1984; Sowa et al., 1985). However, in our study, cadmium induced increase of copper content in tissues. Similar results were observed in the study of Chmielnicka et al. (1996). On the other hand, studies indicated that increased copper supplemented in feed could promote retention of cadmium in tissues of swine (Rambeck et al., 1991; Rothe et al., 1994). In their experiment, weanling pigs received for 3 months a diet containing 1 mg/kg cadmium, as well as 0, 50, 100 or 200 mg/kg copper. When 200 mg/kg copper were added, cadmium rose in the river from 770 to 1,720 μg/kg and in the kidney from 4,620 to 9,320 μg/kg. Tissue cadmium levels of pigs fed diets supplemented with cadmium showed higher and rose with increased cadmium added in feed at the present study (Table 6). These results show that synergistic effect between copper and cadmium may occur in certain conditions. Increased copper content in liver and kidney may be ascribed to increased metallothionein level induced by cadmium, but which cannot be used to explain increased copper level in other tissues. Further researches are needed on interaction between copper and cadmium, since high copper levels have been used in commercial pig fattening to improve the weight gain of the pigs and cadmium is a potential and accumulative contaminant to threaten the health of animal.

There was a significant increase of zinc concentration in renal cortex in 10.0 mg/kg cadmium added group compared to the control ($p<0.05$). Zinc levels in serum, liver and spleen were similar among the treatments ($p>0.05$). However, zinc contents in lymphaden, pancreas and heart of 10.0 mg/kg cadmium treatment were lower than that of the control ($p<0.05$). Zinc levels in tissues among other groups have no difference ($p>0.05$)(Table 7).

The cadmium-induced changes in zinc homeostasis resulted in an increased retention of zinc in the liver and/or kidney of rats or mice, which was reviewed by Brzóska and Moniuszko-Jakouiuk (2001). The cadmium-induced

retention of zinc in the liver and/or kidney is due to cadmium accumulation and metallothionein induction in these organs (Sharma et al., 1991; Brzóska et al., 2000). The molecular ratio of cadmium, zinc and copper in renal metallothionein depends on cadmium and metallothionein concentrations. At low cadmium levels in the kidneys, zinc is the dominating metal in metallothionein, while at high cadmium concentrations cadmium is the dominating metal (Elinder et al., 1987). Thus, deposition of zinc in liver and/or kidney increases, as seen from present study. Changes of zinc content in other soft tissues were also found, as reviewed by Brzóska and Moniuszko-Jakouiuk (2001). However, they are not as evident as in the liver and kidney since cadmium does not induce production of measurable quantities of metallothionein (Sharma et al., 1991; Brzóska et al., 2000).

In the present study, there was high zinc level supplemented in experimental diets, but zinc retention in tissues was still affected by 10.0 mg/kg cadmium exposure. This result shows that it is vital to be aware of cadmium content in animal feed, since zinc plays an important role in growth, development and functioning of all living cells.

Most investigations have focused on a single metal-metal interaction and little is known about the retention of iron, copper and zinc, in the presence of adequate dietary iron, copper and zinc loaded conditions, induced by relatively low cadmium level during extended exposure in domestic animal. Indeed, interactions between cadmium and other essential minerals are complicate and different for each animal species. More research is required to determine interactions of cadmium and other minerals since cadmium is a serious and accumulative environmental contaminant.

IMPLICATION

This study indicated that 10.0 mg/kg cadmium had significant effect on ADG and FCR of growing pigs, which may be resulted from decreased apparent digestibility of nutrients caused by cadmium. Iron and zinc levels in most tissues decreased while tissue copper content increased after extended cadmium exposure period.

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