

# Distribution Pattern of Cadmium in Liver and Kidney of Broiler Chicken: An Experimental Study

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## Abstract

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**Background:** Cadmium (Cd) is not considered as essential element for living organisms, therefore its presence in tissues is considered as toxic element. From food hazards control point of view, information on the distribution of these toxic elements in the animal tissues is very important. The present study was designed to evaluate Cd distribution pattern in kidney and liver of broiler chicken.

**Methods:** Forty eight day-old broiler chickens were randomly and equally divided into different groups including control, I, II and III. During 42 days Cd was added to their diet in amount of 0, 25, 50 and 100 ppm, respectively. From each group, four chickens were sacrificed at 14, 28, 42 days of age, and amount of Cd in their liver and kidney were measured by atomic absorbent spectrophotometer. Analysis of data was performed by two-way ANOVA using SPSS software version 16.0.

**Results:** Interaction effect of the time duration of Cd diet consumption and the accumulation of Cd contents from either liver or kidney in different groups was significant ( $p < 0.05$ ). Increasing dietary Cd levels and exposure days resulted in higher Cd accumulation in kidney and liver. In all groups and in all different ages, kidney Cd levels were higher than those of liver.

**Conclusion:** Cd content in chicken organs is attributable to its dietary level and the duration of exposure. The mean concentration of Cd accumulated in chicken kidney tissue was higher in comparison to it in liver tissue.

## Introduction

Cadmium (Cd) as a toxic heavy metal has been distributed widely and uniformly and with small amounts throughout the earth's crust. Cd is not considered as essential element for living organisms, therefore its presence in organism tissues is considered as contamination (Kramarova et al., 2005; Rehman et al., 2012). Human activities such as production of dyes, plastics, dry batteries, porcelain and etc. have increased Cd contamination in the environment (Akan et al., 2010).

This element is known as one of the most important environmental and industrial toxic agents and affects many

target tissues such as appetite and pain centers, brain, heart and blood vessel, kidney and lungs (Beneddouche et al., 2014; Hyder et al., 2013). High levels of Cd create acute disorders and long time exposure with small amounts create chronic adverse effect such as renal disorders and dysfunction, enzyme reduction of alanine amino transferase, lactate dehydrogenase, aspartate amino transferase in liver and skeletal changes (Maxwell and Iwegbue, 2008).

Intake of Cd resulted in its accumulation in different organs which its amount depends on the interval of exposure, the quantity ingestion, the production and reproduction phases of the animals, as well as their age and breed (Baykov et al., 1996). This element has a very long

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biological half-life and may reach up to 20 years, depending on type of animal (De la Fuente et al., 2002).

Distribution of Cd driven from feed among animal target organs is a key for estimating health risk from this exposure and determining the safety of animal origin foods; however, the bioaccumulation of Cd from feed is modified by many dietary components (Akan et al., 2010).

Several researchers studied the distribution of Cd in liver and kidney of animals (Baykov et al., 1996; Doganoc and Gacnik, 1995; Sharma et al., 1979; Swaileh et al., 2009; White and Finley, 1978). The aim of this study was to investigate distribution pattern of dietary Cd in liver and kidney tissues of broiler chicken and its relation with Cd intake dosage and duration.

## Materials and methods

### *Chemicals and experimental animals*

All chemicals used for determination of Cd were of analytical grade and purchased from Merck Company (Darmstadt, Germany). To achieve this study, 48 day-old broiler chickens having a body weight of  $40 \pm 2$  g were purchased from Zarbal Company (Amol, Iran).

### *Ethical approval*

Protocol and experimental design of study was approved by Department of Animal Ethics Committee, Faculty of Veterinary Medicine, Tehran University, Tehran, Iran (Protocol No. 75070096/6/18).

### *Experimental design*

The design of study was based on Bharavi et al. (2011) with a little modification. Forty eight day-old broiler chickens (Ross) were randomly and equally divided into 4 different groups and each group consisted of 12 chickens. Breeding stages were done in metal cages at room temperature and light levels were adjusted based on standard conditions at various stages. During the breeding, all the chickens had freely access to food and water. Chicken base diet is given in Table 1. During 42 days, Cd was added to base diet in amount of 0, 25, 50 and 100 ppm. In each stage of sample preparation, added Cd amount accuracy was determined by atomic absorbent spectrophotometer.

Control group chickens were fed from base feed including starter diet from day 1 to 14, growth diet from day 14 to 28 and finisher diet from day 28 to 42. The formula of these diets in addition to their effective chemical component has been given in Table 1. The chickens pertaining to group I, II and III were fed with base feed with additional

Cd levels of 25, 50 and 100 ppm, respectively. From each group at 14, 28 and 42 days of age, four chickens were randomly chosen to measure the amount of Cd accumulated in liver and kidney tissue. Chickens were euthanized and their liver and kidney removed. The tissue samples were kept at  $-18$  °C until analysis.

### *Determination of Cd*

For measuring Cd amounts, liver and kidney samples were grounded and homogenized. Five ml of perchloric acid and 20 ml of nitric acid were added to 2 g of homogenate sample and heated at  $130$  °C for 2 h. Chloric acid 10% (10 ml) was added to ash and adjusted by distilled water to 50 ml. The undissolved particles were filtered off. Final digested samples were analyzed for the presence of Cd using a flame atomic absorption spectrophotometer (Melbourne, Australia) under conditions recommended by the instrument manufacturer.

### *Statistical analysis*

Analysis of data was performed using SPSS software version 16.0. To investigate the interaction effect of diet uptake time and diet Cd dose on Cd accumulated in liver and kidney, two-way ANOVA was used. *P* value less than 0.05 was considered to be statistically significant.

## Results

The relations of Cd intake doses and exposure time interaction with Cd average accumulated in the liver were significant ( $p < 0.05$ ). Table 2 shows Cd amounts accumulated in the liver at different ages and groups. In groups I, II and III, liver mean concentration of Cd was higher than that in control group. With the increasing levels of diet Cd, concentrations of Cd in liver increased. In group I, the liver mean Cd amounts in 14, 28 and 42 days of ages showed significant difference compared with control group. There were significant differences between mean of group I (0.64 mg/kg) and group II (5.53 mg/kg) with group III (14.65 mg/kg). The mean accumulated Cd in liver during all days of ages in group II and III are significantly more than group I and control group.

Table 3 shows Cd accumulation in kidney in different ages and groups. Diet Cd dosages and exposure time interaction effect on the mean of accumulated Cd in the kidney was significant ( $p < 0.05$ ). Feeding a diet containing approximately 100 ppm Cd for a period of 42 days was caused 46.65 mg/kg accumulation of this metal in the kidney. There was a significant difference ( $p < 0.05$ ) in the Cd accumulated mean in the kidney of treatment and control groups.

**Table 1:** Composition of base diets in studied broiler chickens

Ingredient	Starter diet	Growth diet	Finisher diet
	(1 to 14 days of age)	(15 to 28 days of age)	(29 to 42 days of age)
	(%)	(%)	(%)
Corn	55.07	58.01	64.46
Soy	38.17	35.45	28
Fatty acid	2.44	3.43	3.5
Dicalcium phosphate	2	1.76	1.62
Calcium carbonate	1.1	1.1	1.1
Salt	0.34	0.34	0.34
Methionine	0.24	0.23	0.2
Lysine	0.19	0.18	0.18
Vitamin supplement	0.20	0.25	0.25
Mineral supplement	0.15	0.25	0.25
Vitamin E supplement	0.1	0.1	0.1
<b>Diet contents by analysis</b>			
Protein	22.1	20.55	18.05
Argenin	1.41	1.302	1.124
Lysine	1.32	1.218	1.061
Methionine	0.572	0.543	0.486
Methonine+Cysteine	0.924	0.875	0.787
Threonine	0.818	0.761	0.669
Tryptophan	0.316	0.29	0.246
Calcium	1.02	0.92	0.91
Bioavailable phosphor	0.51	0.42	0.43
Bioavailable sodium	0.16	0.15	0.15
Bioavailable raw fiber	3.9	3.69	3.38
Bioavailable raw fat	2.42	2.48	2.67

**Table 2:** The mean Cd concentration (mg/kg) accumulated in liver of broiler chicken given different levels of dietary Cd for 42 days

Age (day)	Control	Group I	Group II	Group III
14	ND <sup>Ca*</sup>	0.64±0.07 <sup>Cb**</sup>	5.53±1.07 <sup>Bb</sup>	14.65±6.9 <sup>Ab</sup>
28	ND <sup>Da</sup>	2.43±0.98 <sup>Cb</sup>	13.22±2.99 <sup>Ba</sup>	21.57±9.58 <sup>Aa</sup>
42	0.02±0.003 <sup>Da</sup>	5.36±1.77 <sup>Ca</sup>	15.27±4.9 <sup>Ba</sup>	26.01±3.92 <sup>Aa</sup>

\*ND: Not Detected

\*\*Values were presented as mean value±SD (n=4). Different superscript capital letters within a row indicate statistically significant differences ( $p<0.05$ ) among values. Different superscript small letters within a column indicate statistically significant differences ( $p<0.05$ ) among values**Table 3:** The mean Cd concentration (mg/kg) accumulated in kidney of broiler chicken given different levels of dietary Cd for 42 days

Age (day)	Control	Group I	Group II	Group III
14	ND <sup>Ca*</sup>	1.64±0.07 <sup>Cb**</sup>	17.34±6.06 <sup>Bb</sup>	33.26±3.29 <sup>Ac</sup>
28	ND <sup>Ba</sup>	2.97±1.39 <sup>Cb</sup>	18.76±2.78 <sup>Bb</sup>	38.43±17.82 <sup>Ab</sup>
42	0.03±0.002 <sup>Da</sup>	12.39±4.58 <sup>Ca</sup>	26.33±7.46 <sup>Ba</sup>	46.65±4.03 <sup>Aa</sup>

\*ND: Not Detected

\*\*Values were presented as mean value±SD (n=4). Different superscript capital letters within a row indicate statistically significant differences ( $p<0.05$ ) among values. Different superscript small letters within a column indicate statistically significant differences ( $p<0.05$ ) among values

## Discussion

Cd was absorbed via the gastrointestinal tract and lungs, transported by blood and accumulated in various tissues and organs in the body, mainly kidney and liver because its rate of elimination from these organs is relatively low (Koréneková et al., 2002).

In poultry, feed composition can influence the retention of trace elements. It seems absorption and accumulation of Cd in tissues depends on a wide range of factors including nutritional substance and vitamin status, age and sex of the animal (Massanyi et al., 2003). The ability of liver and kidneys to accumulate high concentration of Cd is a common feature to chickens and many other animals. The accumulation of Cd in liver and kidney tissue is partly attributable to the increased levels of metallothionein in these tissues (Sharma et al., 1979).

Our results showed that dietary Cd greatly increased its content in the liver and kidney of broiler chicken; therefore, Cd accumulation in these organs depended on dietary Cd and exposure time. The rate of accumulation increased along with exposure time and Cd content of diet. We found Cd accumulation in kidney was more than it in liver. These results are in consistence with previous report which showed highest accumulation of Cd occurred in kidney than in liver and the accumulation increase in these organs is a consequence to Cd doses increase in chicken diet (Barregard et al., 2010; Baykov et al., 1996; Sharma et al., 1979; Villar et al., 2005; White and Finley, 1978). Also, the present finding was comparable with those of Kumar et al. (2007) that reported the higher ability of kidneys to accumulate Cd compared liver and muscle in freshwater fish and chicken (Kumar et al., 2007). It is also in consistent with the results of Doganoc and Gačnik (1995) that found Cd accumulation is more in kidney than liver when chicken age was increased. They looked for correlation between the animals' age and Cd content in kidney from *Ilirska Bistrica*. The positive correlation between the concentrations of Cd in kidney of game from *Ilirska Bistrica* and the age was evident. Age-dependent increase in Cd content is due to long biological half-time of it in the liver and kidney (Doganoc and Gačnik, 1995).

## Conclusions

In summary, adding Cd to a chicken diet effects the accumulation of this element in kidney and liver. Cd content in these organs is attributable to its dietary level and the duration of exposure. In this study, it was concluded the mean concentration of Cd accumulated in chicken kidney tissue was higher in comparison to it in liver tissue.

## Conflicts of interest

The authors declare that they have no conflict of interest in this study.

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## References

- Akan J.C., Abdulrahman F.I., Sodipo O.A., Chiroma Y.A. (2010). Distribution of heavy metals in the liver, kidney and meat of beef, mutton, caprine and chicken from Kasuwan Shanu market in Maiduguri Metropolis, Borno state, Nigeria. *Research Journal of Applied Sciences, Engineering and Technology*. 2: 743-748.
- Barregard L., Fabricius-Lagging E., Lundh T., Molne J., Wallin M., Olausson M., Modigh C., Sallsten G. (2010). Cadmium, mercury, and lead in kidney cortex of living kidney donors: impact of different exposure sources. *Environmental Research*. 110: 47-54.
- Baykov B.D., Stoyanov M.P., Gugova M.L. (1996). Cadmium and lead bioaccumulation in male chickens for high food concentrations. *Environmental Toxicology and Chemistry*. 54: 155-159.
- Bendeddouche B., Rachid Z., Esmā B. (2014). Levels of selected heavy metals in fresh meat from cattle, sheep, chicken and camel produced in Algeria. *Annual Review and Research in Biology*. 4: 1260-1267.
- Bharavi K., Reddy A.G., Rao G.S., Kumar P.R., Kumar D.S., Prasadini P.P. (2011). Prevention of cadmium bioaccumulation by herbal adaptogens. *Indian Journal of Pharmacology*. 43: 45-49.
- De la Fuente H., Portales-Perez D., Baranda L., Diaz-Barriga F., Saavedra-Alanis E., Gonzalez-Amaro R. (2002). Effect of arsenic, cadmium and lead on the induction of apoptosis of normal human mononuclear cells. *Clinical and Experimental Immunology*. 129: 69-77.
- Doganoc D.Z., Gačnik K.Š. (1995). Lead and cadmium in meat and organs of game in Slovenia. *Bulletin of Environmental Contamination and Toxicology*. 54: 166-170.
- Hyder O., Chung M., Cosgrove D., Herman J.M., Li Z., Firoozmand A., Gurakar A., Koteish A., Pawliket T.M. (2013). Cadmium exposure and liver disease among US adults. *Journal of Gastrointestinal Surgery*. 17: 1265-1273.
- Koréneková B., Skalická M., Nař P. (2002). Concentration of some heavy metals in cattle reared in the vicinity of a metallurgic industry. *Veterinarski Arhiv*. 72: 259-267.
- Kramarova M., Massanyi P., Slamecka J., Tataruch F., Jancova A., Gasparik J., Fabis M., Kovacic J., Toman R., Galova J., Jurcik R. (2005). Distribution of cadmium and lead in liver and kidney of some wild animals in Slovakia. *Journal of Environmental Science and Health. Part A, Toxic/Hazardous Substances and Environmental Engineering*. 40: 593-600.
- Kumar P., Prasad Y., Patra A.K., Swarup D. (2007). Levels of cadmium and lead in tissues of freshwater fish (*Clarias batrachus* L.) and chicken in Western UP (India). *Bulletin of Environmental Contamination and Toxicology*. 79: 396-400.
- Massanyi P., Tataruch F., Slamecka J., Toman R., Jurik R. (2003). Accumulation of lead, cadmium, and mercury in liver and kidney of the brown hare (*Lepus europaeus*) in relation to the season, age, and sex in the west Slovakian lowland. *Journal of Environmental Science and Health. Part A, Toxic/Hazardous Substances and Environmental Engineering*. 38: 1299-1309.
- Maxwell C., Iwegbue A. (2008). Heavy metal composition of livers and kidneys of cattle from southern Nigeria. *Veterinarski Arhiv*. 78: 401-410.
- Rehman K.U., Andalib S., Ansar M., Bukhar S., Naeem N.M., Yousaf K. (2012). Assessment of heavy metal in different tissues of and domestic layers. *Global Veterinaria*. 9: 32-37.
- Sharma R.P., Street J.C., Verma M.P., Shupe J.L. (1979). Cadmium uptake from feed and its distribution to food prod-

- ucts of livestock. *Environmental Health Perspectives*. 28: 59-66.
- Swailah K.M., Abdulkhaliq A., Hussein R.M., Matani M. (2009). Distribution of toxic metals in organs of local cattle, sheep, goat and poultry from the west Bank, Palestinian authority. *Bulletin of Environmental Contamination and Toxicology*. 83: 265–268.
- Villar T.D.C., Elaine J.P.K., Flavier M.E. (2005). Lead and cadmium levels in edible internal organs and blood of poultry chicken. *Journal of Applied Sciences*. 5: 1250–1253.
- White D.H., Finley M.T. (1978). Uptake and retention of dietary cadmium in mallard ducks. *Environmental Research*. 17: 53-59.