Estimation of Some Heavy Metals in Polluted Well Water and Mercury Accumulation in Broiler Organs

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ABSTRACT

The aim of this study was to investigate the relationship between the concentrations of heavy metals in well water and bioaccumulation of the most abundant metals in chicken tissues in some areas in the province of Mecca Almokaramah, Saudi Arabia. Among the heavy metals (Cd, Zn, Cr, Mn, Cu Hg, Pb and Ni) studied, mercury (Hg) revealed highest in concentration in well waters. The concentration of mercury in the ground water, beside in liver, kidney, muscle and blood samples of ten chickens from each of four poultry- production farms were estimated using atomic absorption spectrophotometer. The results showed that the kidney followed by liver had the highest bioaccumulation of mercury in all farm samples. The level of mercury in the ground water was 7.06µg/L. The relationship between mercury accumulation levels in the kidney and those in the liver tissues were proportionally correlated and altered with elevation in the antioxidant enzyme activities such as AST and ALT. These elevated enzymatic activities were induced by the level of toxicity. There was a significant elevation in the level of liver and kidney malondialdhyde (MDA), while the activities of antioxidant enzymes superoxide dismutase and catalase (SOD and CAT) were significantly decreased. Biochemical observations were supplemented by histopathological examination of liver and kidney sections.

INTRODUCTION

Water pollution is the contamination of water bodies (e.g. lakes, rivers, oceans and groundwater). Water pollution occurs when pollutants are discharged directly or indirectly into the water bodies without adequate treatment to remove the harmful compounds. Water pollution affects the animals and other organisms living in these bodies of water; and, in almost all cases the effect is damaging not only to individual species and populations, but also to the natural biological communities (Mapanda et al. 2005; Anne et al. 2007).

Heavy or toxic metals are trace metals with a density at least five times that of water. As such, they are stable elements (meaning they cannot be metabolized by the body) and bio-accumulative (passed up the food chain to the humans). These heavy metals include: mercury, nickel, lead, arsenic, cadmium, aluminum, platinum, and copper (the metallic form versus the ionic form

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required by the body). Heavy metals have no function in the body and can be highly toxic (Mohammad et al. 2010).

The rate of urbanization and industrialization has been in the increase for the last two decades in Saudi Arabia. Besides many problems associated with such social changes, the pollution is considered to be a major concern for the health of the nation. Among the numerous types of environmental pollutions that constitute as a danger to humanity, the contamination of food chain appears to be a growing threat that requires immediate attention and action. (Khan et al. 1996; Bachman et al. 2002; Anne et al. 2007; Mohammad et al. 2010).

Most heavy metals tend to be associated with sulphar in protein (Rossi and Santaroni 1976). The heavy metals content of streams, lakes and rivers did not normally exceed 0.1 ppm although some water sources located near different heavy metals deposits might contain mixed amounts up to 80 ppm (Wershow 1970; Bachman et al. 2002). Limited data is available for the concentration of heavy metals in rain -water and snow. Manahan (1989) reported that most notorious mercury compounds (for example) in the environment are mono-methyl mercury salts and diethyl mercury salts which are water soluble.

The contaminated water with metals is the route of health hazards in the mankind human and animals. Among various pollutants in the environment, heavy metals are directly related to diseases in humans. Although it is difficult to classify the trace metal into essential and toxic groups, yet it is a well known fact that an essential metal becomes toxic at sufficiently high intakes (Khurshid and Qureshi 1984; Harter et al. 2002; Anne et al. 2007; Akbar Jan et al. 2010).

Lead may enter the environment during the mining, smelting, refining, and manufacturing processes and by the use of lead containing products. Lead intake occurs from the consumption of whisky, fruit juices, food stored in lead lined containers, cosmetics, cigarettes and motor vehicle exhaust etc (Harter et al. 2002; Aradhi et al. 2009). Excess lead can cause serious damage to the brain, kidneys, nervous system and red blood cells. Young children, infants and fetuses are particularly vulnerable to lead poisoning than the adult. According to the US Environment Protection Agency (EPA) says that lead may be implicated in causing leukemia (Anonymous 2002).

Zinc is essential for the normal functioning of the cells; including protein synthesis, carbohydrate metabolism, cell growth and cell division (Saeed 1998). However, if Zn concentration in the air is over 15 mg/m3, "metal fume fever" may result, which causes fever, depression, malaise, cough, vomiting, salivation and headache. cadmium replaces Zn, in many enzymes. Therefore, a higher amount of Zn is required to overcome the toxic effects of cadmium (Khan et al. 1990).

High level of tissue concentration of iron causes increased risk of myocardial infarction (Harvey and Champe 1994) and high or low level of magnesium causes kidney failure and heart problems. High level of calcium is responsible for thirst, increased volume of urine, muscle fatigue, poor mental concentration and formation of kidney stones (Saeed 1998).

Water pollution with mercury is one of the major problems confronting the health officials everywhere. Mercury is a widespread environmental and industrial pollutant, which induces severe alterations in the tissues (Timbrell 1982; Manahan 1989; Lund et al. 1993; Mahboob et al. 2001; Sener et al. 2007), causes numerous neurological abnormalities (Kingman et al. 2005; Auger et al. 2005) and produces peripheral neuropathy (Boyd et al. 2000; Chuu et al. 2007) in the experimental animals and human beings.

Mercury poisoning can result from the inhalation, ingestion, or absorption through the skin and may be highly toxic and corrosive once absorbed into blood stream. Furthermore, it combines with proteins in the plasma or enters the red blood cells but does not readily pass into the brain or fetus and instead, may enter other body organs (El-Shenawy and Hassan 2008). The liver is a major site of metabolism for the mercury and it can accumulate in the liver resulting in severe hepatic damages. Previous studies have revealed that $HgCl_2$ caused histopathological and ultrastructural lesions in the liver evidenced by periportal fatty degeneration and cell necrosis. Schurz et al. (2000) reported that DNA was a vital molecule in the cell activities and was the main target for $HgCl_2$-induced cell injuries.

This study is a surveyed the heavy metals pathway from the environment through ground water to chicken in some particular areas in the province of Mecca Almokaramah where poultry farms were established. This study endeavored to measure these metals as environmental pollutants in the ground water, and investigated the

bioaccumulation of mercury residues (as one of the major concentrated heavy metals) in chicken tissues of major poultry farms. To date no such studies have been conducted in this area where the chicken consumers have been on the increase. The outcome of this study might help in taking precautionary steps in monitoring the metal contamination in the ground water and to advice the authorities of the health impact that might have in Jeddah population as consumers.

MATERIALS AND METHODS

This study was carried out on four farms of poultry production located at four sites in the province of Mecca Almokaramah, Saudi Arabia. The first farm (A) was located in Om Al-Jood area, 150 Km east of Jeddah governorate. The second farm (B) was located in Hada El-Sham area, 80 km east north Jeddah; the third one (C) in Al-Wazeeria region, 60 km east south Jeddah and the fourth one was Al-Fakeih poultry farm (D) in Jeddah governorate, which acted as a control group. The first three farms, A, B and C mainly depended on the ground water and regularly used the water from separate wells I, II and III respectively, while the fourth farm (D) was regularly used the healthy pure drinking water source (IV). The management of the four farms was identical, except the water source. The water samples were collected in clean glass bottles for chemical analysis according to APHA (1995).

The qualitative determination of cadmium (Cd), zinc (Zn), chromium (Cr), magnesium (Mn), copper (Cu), mercury (Hg), lead (Pb) and nickel (Ni) were carried out using Flameless Atomic Absorption Spectrophotometer (Perkin Elmer 2380, U.S.A.). The actual representative concentration (ppm) of the most abundant heavy metal was determined per source. Precautions were taken to avoid the contamination during the sample collection.

The domestic white farm chickens *(Gallus domesticus)* were used in this study. Ten chickens, six weeks old, were chosen from each farm and slaughtered. Liver, kidneys, muscles and blood were taken to determine the mercury residues. Mercury was also determined in the tissue samples by flameless atomic absorption spectrophotometer equipped with a deuterium arc background corrector.

Serum biochemical assay and estimation of MDA, SOD, CAT in liver and kidney tissues:

Serum enzymes aspartate aminotransferase (AST) and serum glutamate pyruvate transaminase (ALT) were determined according to Reitman and Frankel (1957). Liver and kidney samples were dissected and washed immediately with ice cold saline to remove as much blood as possible. Each tested tissue homogenates (5% w/v) were prepared in cold 50mM potassium phosphate buffer (pH 7.4) using glass homogenizer in ice. The cell debris was removed by centrifugation at 5000 rpm for 15 at 40°C. The supernatant was used for the estimation of malondialdehyde (MDA) (Yagi and Rastogi 1979), superoxide dismutase (SOD) (Kakkar et al. 1972) and catalase (CAT) (Smna, 1972) levels.

Histopathological studies
The target organs (liver and kidney) tissues were dissected and fixed in 10% formalin, dehydrated in gradual ethanol (50-99%), cleared in xylene, and embedded in paraffin. Sections were prepared and then stained with hematoxylin and eosin dye for the microscopic investigation.

Statistical analysis
Statistical analysis was performed on a PC using SPSS, V.13, (special package for social sciences). Data are presented as the arithmetic mean ± S.D., The difference among the means has been analyzed by one way ANOVA followed by student t test. A value of $P < 0.05$ was considered as statistically significant.

RESULTS

Table 1 shows the mean concentration (ppm) of cadmium, zinc, chromium, magnesium, copper, mercury, lead and nickel in (A-C). There were significant variations between sites $(P < 0.05)$. The mean concentrations (ppm) of heavy metals ranged from 0.001 to 0.003 for Cd, 0.09 to 0.23 for Zn, 0.03 to 0.15 for Cr, 0.12 to 0.16 for Mn, 0.11 to 0.27 for Cu, 0.38 to 7.34 for Hg, 0.06 to 0.10 for Pb, and 0.04 to 0.06 for Ni. (Table 1). Mercury was highest at farm A and B, which received water from the wells I and II, the levels which exceeds the permissible metal limits of drinking water (6.78 to 7.34 ppm with a mean of 7.06 ppm; Table, 2). The samples from C and D showed Hg concentration lying in the permissible metal limits

of drinking water (WHO, 1980, 1984). For this reason the residual quantities of mercury, which were the most abundant heavy metals in the analyzed wells water samples were estimated in different chicken tissues to find the bioaccumulation of this toxic metal in the tissues and blood of the chicken in poultry farms supplied with this water.

The highest Hg concentrations were detected in the kidney, which is considered the main target organ followed by the liver. The residual quantities of mercury in the liver tissues ranged from 110 to 179 µg/100g with a mean of 143.4 µg/100g in 8 cases from farm A, while the rest revealed undetectable amount of mercury; 123 to 183 µg/100g with a mean of 137.4 µg/100g in 9 cases from farm B, a mean of 132 µg/100g in 4 cases from farm C; and a mean of 117.2 µg/100g in 4 cases from farm D. The rest of the samples revealed undetectable mercury levels (Table 2).

Table 1 - Heavy metal concentrations (ppm) in water sources supplying the chicken farms in different the study sites.

Farm	Level	Concentrations of heavy metals in farm water (ppm)							
		Cd	Zn	Cr	Mn	Cu	Hg	Pb	Ni
A	Mean	0.002	0.09	0.03	0.12	0.11	7.34	0.10	0.05
	Median	0.001	0.07	0.02	0.09	0.05	4.22	0.06	0.03
	Range	0.00–0.002	0.00-0.12	0.00-0.12	0.00–0.18	0.00–0.84	3.09–9.01	0.00-0.37	0.00-0.09
B	Mean	0.003	0.17	0.09	0.14	0.15	6.78	0.06	0.04
	Median	0.001	0.09	0.06	0.08	0.03	3.74	0.04	0.02
	Range	ND–0.004	0.02-0.25	0.00-0.10	0.00–0.28	0.00–0.44	2.99–8.22	0.00-0.39	0.00-0.07
C	Mean	0.001	0.23	0.15	0.15	0.15	1.36	0.08	0.06
	Median	0.002	0.15	0.13	0.10	0.11	1.21	0.05	0.04
	Range	ND–0.003	0.03-0.37	0.00-0.27	0.00–0.43	0.00–0.53	1.00–1.48	0.00–0.25	0.00-0.09
D	Mean	0.002	0.20	0.06	0.16	0.27	0.38	0.07	0.05
	Median	0.001	0.17	0.05	0.12	0.18	0.18	0.04	0.03
	Range	ND–0.004	0.02-0.27	0.00-0.18	0.00–0.33	0.00–0.51	0.00–0.41	0.00–0.09	0.00-0.6
Permissible limits of heavy metals (ppm) in water (WHO, 1984)		0.00–0.005	1.02-2.99	0.00-1.20	0.00–1.05	0.00–1.00	1.00–2.68	0.00–0.50	0.00-1.12

Table 2 - Relationship between the concentrations of mercury in various tissues of chickens (6 weeks old) and in the ground water in four different poultry farms in Mecca Al-Mokaramah Province.

Well No.	Mean Conc. of Mercury in well water (ppm)	Farm	Organ	Concentration of mercury in the collected samples(µg/100 g) wet weight									
				1	2	3	4	5	6	7	8	9	10
I and II	7.06±1.4	A	Liver	110±3.37	155±4.02	126±3.89	152±3.66	ND	165±3.86	ND	179±3.42	143±2.75	129±2.13
			Kidney	734±9.55	508±5.72	351±5.09	646±7.41	491±5.33	410±4.22	_ _	516±4.76	485±3.71	561±4.76
			Muscle		_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _
			Blood	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _
		B	Liver	183±3.76	129±3.40	136±3.09	134±3.33	160±2.15	ND	131±2.11	123±3.06	135±2.13	132±2.66
			Kidney	627±7.58	484±4.11	457±3.66	645±6.03	532±4.50	616±7.11	406±4.29	505±3.35	ND	645±6.55
			Muscle	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _
			Blood	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _
III and IV	Not detectable	C	Liver	154±3.75	130±2.66	ND	ND	141±2.34	_ _	_ _	103±2.05	_ _	_ _
			Kidney	487±4.01	542±4.64	463±3.99	504±5.22	_ _	_ _	_ _	_ _	_ _	_ _
			Muscle	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _
			Blood	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _
		D	Liver	100±2.12	_ _	_ _	124±3.55	ND	127±2.15	_ _	118±2.11	_ _	_ _
			Kidney	_ _	_ _	_ _	457±4.41	498±5.77	_ _	_ _	437±4.44	_ _	_ _
			Muscle	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _
			Blood	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _	_ _

Data are expressed as µg/100 g wet tissue as mean ± S.E. of five samples from each individual, ND: Not detectable.

The residual quantities of mercury, estimated in kidney tissues ranged from 351 to 734 µg/100g with a mean of 542 µg/100g in nine cases from farm A, 406 to 645 µg/100g with a mean of 548.3 µg/100g in nine cases from farm B; a mean of 499 µg/100g in four detectable cases from farm C; a mean of 464 µg/100g in three detectable cases from farm D. The rest of the samples revealed undetectable (Table 2). All the examined blood and muscle samples showed concentrations below the detectable level.

Mercury contents in the liver and kidney were significantly elevated in all the samples collected from farms A and B than from farms C and D (P <0.001). The well water analysis in the four tested farms also clearly showed higher concentration of mercury in farms A and B than those from farms C and D. This was accompanied by a significant increase in the concentration of mercury level in the kidney as well as in liver (P <0.01). The accumulation of mercury in kidney of all tested samples was highly significant (P<0.001) than those in the liver (Table 3).

Enzyme activities of liver and kidney of chickensof the three polluted well farms (A - C) and the control farm (D) are illustrated in Table 4. Serum AST and ALT were significantly increased in the three contaminated farms as compared to the control group (P<0.001). The elevated activities of serum AST and ALT were significantly reduced in

the animal groups supplied with pure water (control). Supplying with well water I and II in farm A and B respectively showed significantly more enzyme activity (*P* < 0.001) than those supplied with well water III (Farm C). Results obtained also revealed an increase in the level of liver and kidney MDA in polluted water farm chickens groups compared to the control group. The activities of SOD and CAT were significantly reduced in the first two contaminated farms (farm A and B), while they were significantly elevated near the normal values in the third group (farm C) or control group (farm D) of non-detected mercury levels.

The chicken liver of the farms A and B which were regularly supplied the water from the polluted wells I and II respectively showed massive fatty changes, necrosis, and broad infiltration of the lymphocytes (Figs. 1A, 1B). The histological architecture of the liver sections of the chickens supplied with undetectable mercury water well III (Fig. 1C) showed more or less normal patterns, with a mild degree of necrosis and slightly lymphocyte infiltration, almost comparable to those of the control group. The histological examination of liver sections of control animals (Fig. 1D) showed normal hepatic cells with well preserved cytoplasm prominent nucleus.

Table 3 - Means of mercury concentration in farm water (ppm) and in organs (µg/100g wet weight).

Mean con. Of mercury in farm water	7.06±1.40			No detectable		
Farm	A	B	A+B	C	D	C+D
Mean of all liver samples	107.5±3.22	126.0±3.08	116.7±3.11	044.0±1.42	048.0±2.05	046.0±1.66
Mean of all detectable samples	143.4±2.45	137.4±2.18	140.1±3.07	132.0±2.33	117.2±2.75	123.7±2.82
Mean of all kidney samples	406.5±4.14	456.9±3.67	431.7±3.08	166.3±2.41	116.0±1.55	141.0±3.72
Mean of all detectable samples	542.0±3.56	548.3±5.11	545.3±4.26	499.0±3.87	464.0±4.27	484.0±4.23

T – Test data				
Farm	Organ	Mean ± S.E.	Value of t	P
(A+B)	Liver	140.1±3.07	194.392	< 0.001
	Kidney	545.3±4.26		
(C+D)	Liver	123.7±2.82	111.013	< 0.001
	Kidney	484.0±4.27		
(A+B) + (B+C)	Liver	131.9±3.74	006.047	> 0.05
(A+B) + (B+C)	Kidney	514.6±5.12	022.371	< 0.01

Insignificant difference (P> 0.05); significant difference (P< 0.01) and highly significant difference (P< 0.001)

Tabela 4 - Enzyme activities of liver and kidney of chickens of the control and three well polluted farms; superoxide dismutase (SOD), catalase (CAT), lipid peroxide product or Malendialdlyde (MDA) and serum aminotransferase enzymes (ALT and AST) of all studied groups (Mean±SD).

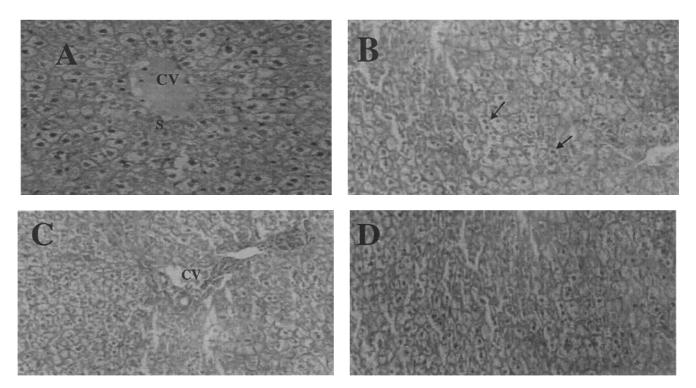| Chicken group parameter	Control (Farm D)		Farm A		Farm B		Farm C	
	Liver	Kidney	Liver	Kidney	Liver	Kidney	Liver	Kidney
Serum ALT (IU/ml)	36.9 ±4.49	37.1±4.44	57.8±7.91	56.2 ±8.88	45.8±6.99	46.0±7.09	39.9±4.99	40.2±4.67
P_1 Value	-	-	P ≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001
P_2 Value	-	-	-	-	P≤ 0.001	P≤ 0.001	P ≤ 0.05	P≤ 0.05
P_3 Value	-	-	-	-	-	-	P≤ 0.05	P≤ 0.05
Serum AST(IU/ml)	40.8±4.55	41.1±4.49	56.7±8.01	55.2±7.99	48.8±6.69	49.1±7.007	44.8±4.72	43.9±4.32
P_1 Value	-	-	P ≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001
P_2 Value	-	-	-	-	P≤ 0.001	P≤ 0.001	P ≤ 0.05	P≤ 0.05
P_3 Value	-	-	-	-	-	-	P≤ 0.05	P≤ 0.05
MDA (m mol/mg protein)	2.33±0.16	2.35±0.18	3.21±0.55	3.28±0.49	2.98±0.23	3.01±0.31	2.56±0.32	2.61±0.47
P_1 Value	-	-	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001
P_2 Value	-	-	-	-	P≤ 0.001	P≤ 0.001	P≤ 0.01	P≤ 0.01
P_3 Value	-	-	-	-	-	-	P≤ 0.05	P≤ 0.05
SOD (MU/mg protein)	221.3 ±14.1	224.1±13.8	106.9±21.7	110.2±2704	203.3±23.8	207.1±27.7	187.7±23.3	188.9±24
P_1 Value	-	-	P≤ 0.001	P≤ 0.001	N.S.	N.S.	P≤ 0.001	P≤ 0.001
P_2 Value	-	-	-	-	P≤ 0.001	P≤ 0.001	P≤ 0.01	P≤ 0.01
P_3 Value	-	-	-	-	-	-	P≤ 0.01	P≤ 0.01
CAT(n mol/min/mg protein)	9757.1± 122.1	9747.9± 142.1	2174.8± 139.1	2300.4± 144.8	8300.4± 144.7	8382.7± 137.7	3989.1± 177.6	4010.7± 146.4
P_1 Value	-	-	P≤ 0.001	P≤ 0.001	N.S.	N.S.	P≤ 0.001	P≤ 0.001
P_2 Value	-	-	-	-	P≤ 0.001	P≤ 0.001	P≤ 0.01	P≤ 0.01
P_3 Value	-	-	-	-	-	-	P≤ 0.05	P≤ 0.05

Figure 1 - (A) Hepatic tissues showing hepatic strands with necrosis around the central vein (CV) leaving blood sinusoids (S) X 400, (B) hepatic tissues of farm 'B' group showing highly cellular necrosis (arrows) around the central veins X 250, (C) hepatic tissues of farm "C" showing degenerating central vein (CV) and (D) hepatic tissues of farm 'D' showing clear regular hepatic strands X 250 (H&E stains).

The chicken kidneys of farms A and B which were regularly supplied the water from the polluted wells I and II respectively showed tissues with tubular epithelial damage, capillary proliferation certain degenerated uriniferous tubules and dilatation of Bowman's capsule (Figs. 2A and 2B).

The above pathological changes were disappeared in chickens supplied with undetectable mercury water well III (Fig. 2C). The histological examination of liver sections of the control animals (Fig. 2D) also showing normal renal tissues and normal uriniferous tubules and glomeruli.
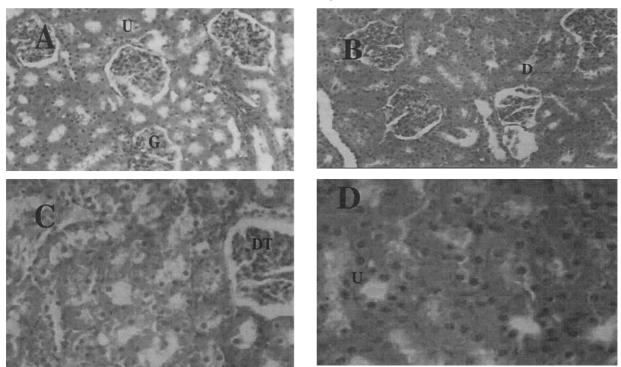

Figure 2 – (A)Renal tissues showing convoluted urineferous tubules (u) and glomeruli (G) X440, (B) renal tissues of farm "B" group showing certain degenerated urineferous tubules (D) X 440, (C) renal tissues of farm "C" showing dilatation of Bowman's capsule (DT) X 440, and (D) renal tissues of farm "D" showing normal renal structure with regulated nuclear arrangement of urineferous tubules (u) X 500 (H&E stains).

DISCUSSION

Table 1 presented the mean concentration (ppm) of cadmium, zinc, chromium, magnesium, copper, mercury, lead and nickel in polluted well supplied farms (A-C) and healthy water supplied farm (D). Although the concentrations of these metals in ground water and fresh tap water lie within the permissible limits recommended by WHO (1984), yet the concentrations of mercury in polluted well water was considered highly significant increased than those of control healthy water and highly elevated than the recommended values by WHO (1984). Nearly similar findings were obtained by Dall Aglio (1968), Warshaw (1970), Zaki et al. (1994) and Abd El-Nasser et al. (1996), and by Youssef and Haleem (1999).

Limited data were available for concentrations of mercury in wells water. Dall Aglio (1968) measured 300 samples from natural water for mercury in Italy and found values in the range of 10-15 ppm. The present study showed undetectable mercury levels in 2 wells and 7.06 ppm in the other two wells. Similar results were reported by Wershaw (1970), who revealed that the mercury content of streams, lakes and rivers does not exceed 0.1 ppm but some water sources located near mercury deposits may contain mercury up to 8.0 ppm. It is obvious that the examined water samples from wells I and II exceeded the permissible limits indicative of water pollution (1.00 – 2.68 ppm) and water quality standard (WHO, 1984).

The trace amounts of mercury detected in the liver, kidneys, muscle and blood tissues of chickens showed the current status and the background Hg concentrations in water supply the poultry farms. The detected Hg levels in chicken organs are in accordance with those reported by other authors (Pribilincov et al. 1997; Marettova et al. 2003; Cabanero et al. 2005).

Most mercury compounds of the contaminant water are inorganic form. Absorption of inorganic mercury compounds may be 15% or less (WHO, 1980), whereas methyl mercury is almost completely absorbed. Inorganic mercury compounds are rapidly accumulated in the kidney which is the main target organ for these compounds. Animal data indicate that the kidney accumulate the highest tissue concentration no matter what form of mercury is administrated (WHO, 1976). This opinion gets along with the present results which showed that the kidney followed by liver are the organs with the highest bioaccumulation of mercury in all farm samples. Similar results were previously reported by Manahan (1989), Lund et al. (1993), Mahboob et al. (2001), Sener et al. (2007).

Although the kidney tissue showed the highest concentration of mercury, its residue could not be detected in all samples which may be explained by uncertain and indirect nature of relationships calculated between the intake of mercury through water and levels of mercury in the indicator organs (WHO 1972; Manahan 1989; Pribilincov et al. 1997; El-Shenawy and Hassan 2008). Considerable individual variation around the average values of mercury residual have been noted, which must be taken into account in the estimation of risk in exposed populations.

Regarding to the presence to the presence of mercury in the kidney and/ or liver in chicken supplied by undetectable mercury level well-water, WHO (1972) reported that the rate of mercury accumulation is independent of the intake level and that of toxic level would be reached eventually, even at a very low intake level. Such conclusion is in agreement with that of Cabanero et al. (2005). It is evident therefore, that even very small concentration of mercury in the environment may constitute eventual toxicological hazard.

In the present study, alteration in the normal levels of various serum biochemical parameters accompanied by the histopathological necrosis and degenerative changes in the liver and kidney tissue were the main toxic effects observed in the chickens drinking polluted water. In fact, some physiological changes has been accompanies with mercury toxicities. Such decrease in body weight gain of chicken due to mercury exposure has been reported earlier also (Marettova et al. 2003). Furthermore, it has also been reported that mercury exposure increased the activities of AST and qualitative vascular degenerative of kidney tissues and necrotic changes were also observed in the liver of chickens (Zraly et al. 2008). These observations are clearly in agreement with the present findings.

Serum AST and ALT activities were used as a marker of tissue damage. Mercury toxicity's produces tissue damage due to its toxic metabolites (Sharma et al. 2002). The toxic metabolite free radical is produced by cytochrome p450 which further reacts with oxygen to produce trichloromethyl peroxy radicals (Borg et al. 2003). These radicals bind covalently with the macromolecule and cause peroxidative degradation of lipid membranes of the liver and kidney. Increased lipid peroxidation under pollution conditions can be due to increased oxidative stress in the cell as a result of depletion of antioxidant scavenger systems. Associated with the changes in lipid peroxidation the affected tissues showed decreased activities of key antioxidants SOD and CAT and increase MDA which play an important role in scavenging the toxic intermediate of incomplete oxidation. SOD and CAT are the two major scavenging enzymes that remove toxic free radicals in vivo. Previous studies have reported that the activity of SOD is low after mercury toxicities (Sobutskii et al. 2007) who measured biochemical indexes of blood after low doses of mercury exposures which come in agreement with our results.

Elevation in the activity of serum AST and ALT, the cytoplasmic enzymes, indicates for necrotic lesions in the liver and tissue degeneration of the kidney, while a decrease in serum SOD and CAT levels indicates for no congestion or cholestasis (Lysenko 2000; Borg et al. 2003; Cabanero et al. 2005). These researchers reported that chickens treated with $HgCl_2$ showed significant elevations in serum glutamate oxaloacetate transaminase (SGOT) and serum glutamate pyruvate transaminase (SGPT) activities, whereas a significant decline in the SOD and CAT activities and also come in agreement and confirm our results.

The present study also shows that chickens supplied with mercury polluted water have elevated levels of serum AST and ALT, whereas a reduced level of SOD and CAT as compared to the control group, indicating clearly that our results are in agreement with other studies on chickens (Borg et al. 2003; Cabanero et al. 2005; Zraly et al. 2008).

On the other hand, in non-chicken model also (teleost fish), Sastry and Sharma (1980) reported that SOD activity decreased in acute exposure to $HgCl_2$ and increased in chronic exposure to $HgCl_2$, however, there was elevation of both AST and ALT either in acute or chronic exposure to $HgCl_2$. Thus, ALT activity in serum, could serve as a marker enzyme to evaluate functional status of liver as suggested by Sobutskii et al. (2007). Furthermore, Jagadeesan and Pillai (2007) also reported significant increase in the level of serum AST and ALT in rats due to $HgCl_2$ treatment for longer time period (30 days). In another study, a significant rise in the serum ALT and AST also been reported in mercury exposed rats (Singh et al. 2007).

Altogether, the present results in the light of the above cited literature clearly indicate that increase in serum ALT and AST and decrease in serum SOD and CAT, can be used as potential enzyme biomarkers for mercury-induced hepatotoxicosis and nephrotoxicois which ultimately affects the general health by altering the functional and structural integrity of liver and kidney and serve as possible bio-indicators for mercury poisoning. However, in order to establish these serums enzyme levels as biomarkers for mercury poisoning, further detailed studies are required at experimental as well as clinical levels.

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