https://doi.org/10.17221/86/2021-CJAS

Adverse effects of cadmium on poultry and role of selenium against it: An updated review

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Citation: Ketta M., Tůmová E., Stupka R., Čítek J., Chodová D. (2021): Adverse effects of cadmium on poultry and role of selenium against it: An updated review. Czech J. Anim. Sci., 66: 339–348.

Abstract: Cadmium (Cd) is a toxic heavy metal, discharged into environments naturally or anthropogenically, where industrial and agricultural revolutions have led to serious Cd pollution into the environment. Poultry feed may be contaminated by Cd due to its presence in concentrates and supplements, and also in the machinery and equipment used in the feed production and processing. After absorption in the digestive system, Cd largely accumulates in kidneys, liver, bones and other organs and causes irreversible damage to these organs. Higher doses of Cd in poultry diet reduced feed intake, body weight, bone mineral density, egg production and eggshell quality. Selenium (Se) is an essential micronutrient and important component of a variety of antioxidant enzymes, with the ability to antagonize metal toxicity including Cd. Hence, in the current review, we try to summarize recent information about the toxicological effects of Cd and provide updated information about the role of Se as an effective strategy in reducing Cd toxicity in poultry.

Keywords: Cd; toxicity; chickens; quails; Se

Heavy metal contamination accounts for 30.8% and 34.8% of the total pollution load of soil and groundwater contaminants in the European Union, respectively (Panagos et al. 2013). Along with many other heavy metals, cadmium (Cd) might be considered the most toxic heavy metal to poultry. This is due to the long biological half-life (10 to 30 years) in living organisms (Shi et al. 2017) and difficulty to discharge. Cadmium can accumulate in various vital organs, especially liver and kidney, causing damage to the animal's health (Tao et al. 2020). The bioaccumulation of Cd eventually leads to serious consequences for animal production, economic benefits, and even human health.

Different forms of exposure to Cd have recently been published, with Cd being present in the environment from natural or anthropogenic sources as a result of many human activities. When the contamination of the atmospheric environment with anthropogenic sources contributes three to 10 times more than the natural sources (Waisberg et al. 2003). Volcanic activity, the gradual process of erosion of rocks, and forest fires are among the main reasons for natural Cd occurrence in the living environment, where more than 90% of Cd in the surface environment is the result of industrial and agricultural processes (Pan et al. 2010). This includes copper and nickel smelting, fossil fuel combustion, mining waste especially from zinc and lead mines, phosphate fertilizers and use of sewage sludge in the soil (Waisberg et al. 2003; Thompson and Bannigan 2008). Moreover, Cd salts with chloride, oxide, and sulphide are produced from nickel-Cd batteries, mirrors, paints, vacuum tubes, plastic stabilizers, ceramics, and fluorescent screens (Rani et al. 2014). The sulphate, nitrate, chloride and acetate forms of Cd are highly soluble in water (NRC

https://doi.org/10.17221/86/2021-CJAS

2005) and transferred from soil to plant with relatively high bioavailability (Lu et al. 2017; Zhu et al. 2018). Therefore, from these natural and anthropogenic sources, once released into the environment, Cd can easily move to water and soil and accumulate in crops and enter the food chain, adversely affecting poultry health (Kar and Patra 2021).

Poultry feed may be contaminated by Cd due to its presence in concentrates and supplements, and also in the machinery and equipment used in the feed production and processing (Wang et al. 2013). In their survey, Wolf and Cappai (2021) processed 408 feed samples to estimate poultry feed contamination in Germany and indicated that Cd contents remained below the maximum levels permitted for feed materials of plant origin (1 mg/kg of feed). The authors also revealed that Cd content showed similar values both in cereal feeds and compound feeds, whereas its content in legume feeds turned out to be slightly higher (0.089 mg/kg of feed). Additionally, concentrations of Cd in poultry feed samples ranged from 0.10 mg/kg to 0.46 mg/kg of feed in commercial farms of England and Wales (Nicholson et al. 1999). However, in some reports Cd concentrations exceeded the higher limits in poultry feeds in Nigeria (3.19 mg/kg of feed) (Bukar and Sa'id 2014) and in India (5.61 mg/kg of feed) (Kar et al. 2018).

Selenium (Se) is an essential element and acts as a co-factor for antioxidant enzymes [glutathione peroxidase (GSH-Px), superoxide dismutase (SOD), and catalase (CAT)], (e.g., GPx, DOD and thioredoxin reductase) and iodothyronine deiodinase enzymes, which are responsible for the conversion of thyroxin (T_4) into active triiodothyronine (T_3) (El-Deep et al. 2016). Thus, Se is involved in several biological functions including antioxidative properties, basal metabolic rate, protein synthesis, and the metabolism of fat, carbohydrate, protein and vitamins (Ebeid 2012; Saleh and Ebeid 2019), which have the ability to antagonize metal toxicity including Cd. Therefore, the current review is an effort to throw the light on recent findings related to the toxicological effects of Cd and provides updated information about the role of Se as an effective strategy in reducing Cd toxicity in poultry.

Toxic levels of cadmium

Studies vary in an estimation of the toxic level of Cd in poultry. Lopez-Alonso (2012) assumed that in most of the domestic species Cd concentrations up to 5 mg/kg dry matter are most likely to start gross clinical symptoms. Olgun and Bahtiyarca (2015) reported 15 mg/kg dry matter as the toxic level of Cd for laying hens. More recent study (Wolf and Cappai 2021) permitted more than 1 mg of Cd/kg of feed of plant origin. On the other hand, Suttle (2010) reported that Cd levels in forages and cereals should be lower than 1 mg/kg. However, the National Research Council (NRC 2005) reported the toxic level of Cd for poultry as 10 mg/kg. Olgun (2015) revealed that 20 mg of Cd/kg of feed is a toxic level for laying quail while 40 mg of Cd/kg of feed for growing quail were indicated by Abou-Kassem et al. (2016). The maximum levels of Cd in feed materials permitted by the European Food Safety Authority (EFSA 2004) range between 1.14 mg/kg and 2.27 mg/kg of feed dry matter. However, the mean concentrations of Cd in feed and feed materials used for poultry are presented in Table 1. A recently published review by Olgun et al. (2020) indicated that 10–20 mg of Cd/kg of feed is sufficient to observe toxicity symptoms. These contrast reports estimating the toxic level of Cd might be reasoned by concentrations in feeds, different species of the birds, sex, age, systems of bird rearing and the duration of intake (Olgun et al. 2020; Kar and Patra 2021). Additionally, an interaction between Cd and some dietary elements like zinc and copper could be the main reason for a non-precise estimation of minimum and maximum levels of Cd for safe dietary concentrations. Even the cell type

Table 1. Total Cd concentration (mg/kg) in poultry feed of the European Union member states

	Mean	Min.	Max.	*n*
Compound feeds				
Poultry-layers	0.16	0.01	0.60	12
Poultry-broilers	0.19	0.1	0.50	8
Certain feed materials				
Fish meal	0.40	0.04	1.4	44
Rapeseed, extracted	0.15	0.02	0.50	20
Wheat and wheat by-products	0.19	0.05	0.75	27
Soybean meals	0.07	0.01	0.20	17
Maize grain and maize by-products	0.06	0.01	0.50	29
Minerals and pre-mixtures	0.58	0.01	2.34	–

Modified from EFSA (2004)

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might affect the absorption of Cd into the body. Nad et al. (2012) reported high Cd absorption affinity by somatic cells of kidney and liver of turkeys, while Li et al. (2018) obtained low Cd absorption affinity of the laying hen ovary.

Adverse effects of Cd on poultry performance

The toxic effect of Cd on hen performance has been of the outmost interest for researchers in the last decades. The impact of Cd toxicity in chickens and Japanese quails is presented in Table 2. Early studies by Leach et al. (1979), Nicholson et al. (1983) and Burger et al. (1992) illustrated that Cd toxicity reduced reproductive performance, egg production, eggshell thickness and increased stress and diseases. Nolan and Brown (2000) studied the effect of a lower concentration of Cd (0.32 mg/kg to 1.62 mg/kg of body weight) and reported non-significant effects on body weight and feed intake but reduced egg production. Erdogan et al. (2005) demonstrated a significant reduction in body weight after Cd supplementation (25 mg/l in drinking water) without affecting feed intake. Al-Waeli et al. (2013) reported that the addition of 100 mg of Cd/kg of feed caused significant negative effects on performance such as reduced body mass, decreased feed consumption and increased feed conversion ratio. While the addition of 10 mg of Cd/kg of broiler diets showed no negative effects on performance and mortality. Additionally, Olgun and Bahtiyarca (2015) studied the effect of dietary Cd at up to 15 mg/kg and 45 mg/kg of feed for 12 weeks and revealed negative effects of Cd on egg production and feed to egg ratio. It might be assumed that the negative effect of Cd on feed intake and feed conversion ratio could be related to the impaired nutrient digestion and absorption in the gut. This theory was supported by Teshfam et al. (2006), who indicated that administrations

Table 2. Adverse effects of Cd toxicity on chickens and Japanese quails

Species	Level of Cd	Symptoms	Reference
Chickens	10 mg/kg of feed	Non-significant effect on performance.	Al-Waeli et al. (2013)
	15–45 mg/kg of feed	Significant negative effects of Cd on egg production and feed to egg ratio.	Olgun and Bahtiyarca (2015)
	20 mg/kg of feed	Significant structural changes in the testis and in the qualitative properties of the semen.	Marettova et al. (2013)
	25 mg/l drinking water	Significant reduction in body weight.	Erdogan et al. (2005)
	30–60.67 mg/kg of feed	Eggshell quality parameters were significantly deteriorated, especially eggshell thickness and strength.	Leach et al. (1979); Olgun (2015); Zhu et al. (2020)
	50 mg/l drinking water	Induced pale, swollen, fragile, and focal necrotic spots in liver.	Singh et al. (2016)
	60 mg/kg of feed	Reduced production performance and elevated albumen and yolk residues. Severe hydropic degeneration in liver tissues.	Cinar et al. (2011); Tao et al. (2020)
	100 mg/kg of feed	Significant reduction in body mass, decreased feed consumption and increased feed conversion ratio.	Al-Waeli et al. (2013)
	150 mg/kg of feed	Interstitial oedema and partial loss of spermatogenic cells.	Li et al. (2010)
Quails	injection of 10 mg/kg of body weight	Significantly increased mortality (41.7%).	Rahman et al. (2007)
	20–80 mg/kg of feed	Significant reduction in feed intake, egg production and egg weight.	Olgun (2015)
	40–120 mg/kg	Significant decrease in livability rate, body weight and feed efficiency.	Abou-Kassem et al. (2016)
	100 mg/kg and 300 mg/kg of feed	Severe decrease of body weight and feed intake. Significant decreases in blood haemoglobin level. Pale and swollen kidneys with distended ureters. Vacuolar degeneration and necrosis.	Butt et al. (2018); Saleemi et al. (2019)

https://doi.org/10.17221/86/2021-CJAS

of Cd (5 mg/kg to 100 mg/kg of feed for 49 days) altered intestinal mucosal structures by decreased villus width and crypt depth. While the negative effects of Cd on egg production may be directly related to the damage in the digestive tract and ovaries. Inflammation in ovaries caused by Cd alters the production of progesterone and oestrogen from ovaries, which modify the functions and morphology of ovaries (Benoff et al. 2009). Yang et al. (2012) reported that dietary 140 mg and 240 mg of Cd/kg of feed increased Cd content in the ovary which caused oxidative damage of ovary tissues, decreased oestradiol and progesterone in serum, and disrupted the antioxidant defence mechanism. Butt et al. (2018) illustrated that oviducts were atrophied in female Japanese quails fed Cd (300 mg/kg of feed) for 60 days.

In quails, Rahman et al. (2007) reported that body weight and egg production were significantly decreased at contamination levels of 1 mg/kg and 3 mg/kg of body weight. Additionally, they indicated that the mortality after injection of 10 mg Cd/kg of body weight was 41.7%. Moreover, higher Cd administration (20–80 mg/kg of feed) in laying quails decreased egg production, egg weight and feed intake (Olgun 2015). Moreover, Abou-Kassem et al. (2016) obtained that livability rate, body weight and feed intake were negatively affected by increased dietary Cd levels (40 mg, 80 mg and 120 mg of Cd/kg of feed) in growing quails. Additionally, a severe decrease of body weight and feed intake at Cd levels of 100 mg or 300 mg of Cd/kg of feed was observed by Butt et al. (2018). On the other hand, Olgun and Yildiz (2014) found that the dietary Cd (20 mg/kg of feed) concentration had no effect on feed intake, feed conversion ratio and egg production in quails. Moreover, studies demonstrated that Cd negatively affects bone health by deteriorating the activity and metabolism of bone cells, reducing absorption of Ca from the intestines, increasing excretion of Ca from the kidneys, and preventing Ca incorporation and collagen production into bone cells (Olgun and Bahtiyarca 2015). Wang and Bhattacharrya (1993) demonstrated that Cd accumulation causes decalcification in bones. Olgun (2015) indicated that the addition of 5 mg to 80 mg of Cd/kg of feed to layer quail caused linear decreases in shear force and shear stress in the tibia.

Regarding eggshell quality, an early study by Leach et al. (1979) noted that eggshell thickness was significantly reduced by dietary supplementation (48 mg of Cd/kg of feed) during a long-term study (48 weeks), but the addition of Cd to the diet did not affect eggshell thickness in laying hens during the 12-week study. Recently, a linear relationship between cracked eggs and Cd concentration in diet was obtained (Olgun 2015). The author reported that the highest eggshell thickness value was obtained in quails receiving 5 mg of Cd/kg of feed, but with the addition of 40 mg of Cd/kg of feed, the eggshell quality parameters were deteriorated. Dietary supplementation of 30.55 mg and 60.67 mg of Cd/kg of feed had a negative significant effect on eggshell thickness, palisade layer thickness and eggshell strength while the eggshell outer surface became rougher (Zhu et al. 2020). However, Korenekova et al. (2007) and Skalicka et al. (2008) demonstrated that the eggshell thickness of quail fed Cd-contaminated diets did not change in comparison with the control. The reason for a negative impact of Cd on eggshell quality parameters is related to Cd functions as a potential endocrine disruptor that induces oxidative stress and inflammation in the eggshell glands of laying hens (Zhu et al. 2020). Moreover, Cd can interfere with the transport and metabolism of Ca^{+2} ions (Choong et al. 2014) where Ca^{+2} concentration is the main factor effecting the eggshell quality parameters, mainly thickness and strength (Ebeid et al. 2012; Tumova et al. 2016; Ketta et al. 2019).

Cadmium effect on internal organs, haematological and biochemical blood indicators in poultry

After oral intake, Cd is absorbed very slowly and only to a limited extent. In the animal body, it can be bound to metallothionein synthesized in the liver, to be slowly released in the bloodstream (Nordberg and Nordberg 1987). Afterwards Cd starts to affect the poultry internal organs regarding to concentration and period of exposure.

Liver is the first targeted organ by Cd after absorption from gastrointestinal tracts (DelRaso et al. 2003; Rani et al. 2014). Whereas the kidneys are the second most affected organs by Cd toxicity as they function for excretion of toxic compounds (Kar and Patra 2021). In broiler chickens, Cinar et al. (2011) reported severe hydropic degeneration in liver tissues with Cd administration (60 mg of Cd/kg of feed) for 42 days. Additionally, Singh et al. (2016) observed that the daily administration of Cd (50 mg/l in drinking water) for 45 days to chickens induced pale, swollen, fragile, and fo-

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cal necrotic spots in the liver. Moreover, hepatic injury was observed in laying hens that received 30 mg and 60 mg of Cd/kg of feed (Tao et al. 2020). The kidneys appear to be the most sensitive organs for Cd accumulation (Toman et al. 2005). Singh et al. (2016) reported that the kidneys were congested after administration of Cd (50 mg/l in drinking water) for 45 days. Histopathological changes of the kidneys with necrosis and degeneration in renal tubules were reported by Cinar et al. (2011) after Cd administration (60 mg of Cd/kg of feed) for 42 days. The toxic effect of Cd was found in the lungs of chickens by Himayun et al. (2018). The authors indicated that feeding a diet of 1.61 mg of Cd/kg of feed resulted in the congestion of blood vessels and air capillaries, haemorrhage in lung parenchyma, and infiltration of mononuclear cells. In the chicken brain, Cd caused marked pathological changes with loss of prominent axons and dendrites and cell necrosis in the cerebrum and cerebellum (Liu et al. 2014). Moreover, the distributions of several minerals in the brains of laying hens were changed due to feeding of Cd (150 mg of Cd/kg of feed) for 90 days (Zhang et al. 2016).

The reproduction system of cocks was also affected by Cd toxicity in the study of Li et al. (2010), who observed that a high dose of Cd (150 mg of Cd/kg of feed) affected the interstitial oedema and partial loss of spermatogenic cells and the number of apoptotic cells in the testes significantly increased. A more recent study by Marettova et al. (2013) indicated that a lower dose of Cd (20 mg of Cd/kg of feed) caused structural changes in the testis and in the qualitative properties of the semen.

Regarding Japanese quails, in the study investigating the impact of different levels of Cd (0.2 mg, 1 mg and 5 mg of Cd/kg of feed) for six months, Akter et al. (2019) reported degenerative changes of the liver depending on Cd concentration. The authors indicated necrosis of hepatocytes, pyknotic nuclei, and disarrangement of hepatic cord occurred in the liver of quails. Moreover, Karimi et al. (2015) increased the Cd concentration in feed (25 mg/kg of feed) for a shorter period (60 days) and reported vacuolar degeneration, single cell necrosis, and the increasing number of Kupffer cells in the liver. Furthermore, Saleemi et al. (2019) obtained vacuolar degeneration, necrosis, and inflammation along with pyknotic nuclei in hepatocytes throughout the parenchyma in immature Japanese quails fed Cd at 150 mg/kg and 300 mg/kg of feed for 60 days. Dietary 300 mg of Cd/kg of feed administered for 60 days resulted in different grades of tubular damage, pale and swollen kidneys with distended ureters (Butt et al. 2018). Moreover, Cd administration (25 mg of Cd/kg of feed) caused swelling of epithelial cells in renal tubules, hyperaemia, and necrosis of renal tubular epithelia (Karimi et al. 2016). Even a low dose of Cd (0.2 mg/kg, 1 mg/kg, and 5 mg/kg of feed) fed for a longer period (six months) resulted in necrosis, deformation of the normal structure of kidney tubules and congestion in the kidney (Akter et al. 2019). Akter et al. (2019) found that Cd (5 mg/kg of feed) in quail chick feed for six months caused haemorrhage in the lungs, but no such a type of changes was found after the administration of Cd at 0.2 mg/kg and 1 mg/kg of feed.

There are a few studies aimed to investigate the effect of Cd on haematological and biochemical blood indicators in poultry. Gutyj et al. (2019) reported that Cd administration (2 mg and 4 mg of Cd/kg of body weight) in 78-weeks-old Hisex White laying hens for 30 days resulted in significant decreases in the blood haemoglobin level but a significant increase in white blood cells in both concentrations. The authors also indicated changes in morphological parameters on the 21st day of the experiment in chickens that were given a Cd dose of 4 mg/kg of body weight. Moreover, Abdo and Abdulla (2013) reported a significant reduction in red blood cells, haemoglobin and packed cell volume after the administration of Cd (10 mg/kg body weight in drinking water) for 30 days in six-weeks-old chickens. Butt et al. (2018) reported significant decreases in the blood haemoglobin level of female Japanese quails fed either low or high dose of Cd (150 mg/kg and 300 mg/kg of feed). On the other hand, Haziri et al. (2012) illustrated that the red blood cells and blood haemoglobin levels were not changed in Isa Brown laying hens provided Cd in drinking water (2 mg, 4 mg and 6 mg of Cd/kg of body weight) for 19 days. Additionally, Salar et al. (2006) reported that the white blood cell count was not statistically significant in broiler chickens exposed to Cd (5 mg, 50 mg, and 100 mg of Cd/kg of feed) for seven weeks. In correspondence, Cinar et al. (2010) did not observe any changes in packed cell volume, haemoglobin levels, and red blood cells after Cd administration of 60 mg/kg of feed for 42 days compared with the control group. The conflicting results might be related to different conditions such as genotype, antioxidative status, immunity, and age (Antunovic et al. 2021).

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In their study, Khaled and Huda (2013) administered Cd at a dose of 10 mg/l in drinking water to six-weeks-old chickens and reported a significant increase in plasma glucose, alanine aminotransferase, aspartate aminotransferase and creatinine levels. In Japanese quail chicks, Cd administration of 40 mg/kg, 80 mg/kg, and 120 mg/kg of feed resulted in significant increases in total protein, albumin, creatinine and alkaline phosphatase levels (Abou-Kassem et al. 2016). In male Japanese quails, Tahir et al. (2017) used a high dose of Cd (150 mg/kg and 300 mg/kg of feed) and reported a significant increase in creatinine, alanine aminotransferase and aspartate aminotransferase, but significant decreases in total protein and albumin.

Role of selenium against cadmium toxicity

The development of safe and effective strategies against Cd toxicity and oxidative stress (free radical generation) is necessary; previous studies have shown that dietary supplements play an important role in reducing or preventing Cd toxicity and oxidative stress (Ebeid 2009; Al-Waeli et al. 2013; Zhang et al. 2017a, b; Xiong et al. 2020). Free radicals damage cell phospholipid membranes and attack the vital components within the cell such as DNA, mitochondria, lysosomes, etc., which is consequently related with disorders like apoptosis, several diseases and deteriorating muscle membrane integrity (Abdel-Moneim et al. 2021). Indeed, all antioxidants within the cell/body collaborate effectively together to preserve the optimum status of redox balance in the cell/body to provide protection against the damaging effects of free radicals (Eid et al. 2008; Ebeid et al. 2013). Thus, dietary supplementation of antioxidants is an effective tool to mitigate the adverse impacts of free radicals. Selenium (Se) is an essential micronutrient and important component of a variety of antioxidant enzymes, known to play a pivotal role in the antioxidant defence system, protecting the organism from oxidative stress (Ebeid 2012; Suchy et al. 2014; Saleh and Ebeid 2019) and positively impacts on the endocrine system (Beckett and Arthur 2005). Pappas et al. (2011) and Al-Waeli et al. (2013) demonstrated that the dietary addition of Se (0.3 mg/kg or 3.0 mg/kg of feed) may be helpful in preventing the toxic effects of Cd (10 mg/kg or 100 mg/kg of feed) in broilers. Zhang et al. (2017b) studied the pathological effect of Cd intoxication (150 mg/kg of feed for 90 days) on chicken liver. The authors reported that apoptotic cell numbers increased in response to Cd in the liver, which were attenuated by Se supplementation (2 mg/kg of feed). It was suggested that Se ameliorates Cd toxicity through binding Cd into biologically inert complexes helping its removal and also through the activities of Se-dependent antioxidant enzymes (Zwolak 2020). Moreover, Se (2 mg/kg of feed) lowered Cd accumulation in ovarian tissues, increased the oestradiol and progesterone concentrations, and decreased apoptosis via the endoplasmic reticulum stress pathway in chickens (Xiong et al. 2020). Semen quality (sperm count, motility, and volume) and antioxidative status in cockerels were improved by Se (Ebeid 2009), which were deteriorated by Cd exposure (Marettova et al. 2013). The selenium function to protect the renal and hepatic tissues against the toxicity of Cd is a result of lipid peroxidation reduction and increases the activities of antioxidant enzymes in these tissues (Newairy et al. 2007).

Selenium supplementation increased the essential minerals (Cr, Mn, Zn, and Se) and decreased toxic mineral (Cd, Li, and Pb) contents in kidney tissues of chickens, which were induced by Cd, suggesting that Se may decrease the negative effects of Cd and improve the trace mineral homeostasis in tissues of chickens (Zhang et al. 2017a; Bao et al. 2018). Dietary supplemental Se (2 mg/kg of feed) was effective in reducing the negative effects of Cd (150 mg/kg of feed for 90 days) on the keel bone whereby it reduced mineralisation in broilers and laying hens (Zhang et al. 2017a). Moreover, in their experiment with Hy-Line Brown laying chickens, Bao et al. (2018) demonstrated that Cd accumulation in pancreas decreased by 48.18% in the Se-treated group compared with the control group, and that of the Se/Cd-treated group decreased by 21.90% compared with the Cd-treated group. Additionally, accumulation in ovaries of Hy-Line Brown layers was decreased upon Se treatment by about 12.6% compared to the Cd-treated group (Li et al. 2018). Nad et al. (2007) found that Cd treatment (3 mg/kg Cd of feed) resulted in the damaged ovarian structure of Japanese quail with the number of atretic follicles significantly increased, and Se and Cd administration (0.4 mg of Se/kg of feed) conferred protective effects. Moreover, Liu et al. (2014) fed Isa Brown chickens basic diet supple-

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mented with Se (10 mg Na_2SeO_3/kg of feed) and Cd (150 mg $CdCl_2$/kg of feed) and reported that Se dietary supplementation reduced Cd accumulation in the brain and that Se supplementation markedly enhanced the antioxidant defence system, which prevented Cd-caused oxidative damage.

Conclusions

In conclusion, poultry exposure to Cd is a result extensive industrial, mining and agricultural practices in some regions around the world. The lower Cd concentration in poultry feed (less than 10 mg/kg) has a non-significant effect on poultry health and performance. However, the higher dietary concentration of Cd (more than 10 mg/kg of feed) in poultry caused a negative impact on performance parameters and eggshell quality resulting in significant economic losses. The adverse effects of Cd on performance and eggshell quality, and the Cd accumulation in the bone might be prevented by the addition of Se to the diets where the administration of Se (2 mg/kg of feed) might be useful to improve poultry performance and health status by reducing Cd absorption and metabolism.

Conflict of interest

The authors declare no conflict of interest.

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Received: May 19, 2021
Accepted: July 23, 2021
Published online: August 20, 2021