Egypt. J. Rad. Sci. Applic. **Vol. 31,** No.1, pp. 63 - 75 (2018)

Wholesomeness and Safety of Meat of Laying Hens Fed Irradiated Aflatoxin B_1-contaminated Diet

M. Diaa El-Din H. Farag[1], E.A. Abdallah[2], A.M. Abdul Azeem[1#] and Nashwa A.H.Ahmed[3]

[1]Food Irradiation Research Department, National Center for Radiation Research & Technology, Atomic Energy Authority, Cairo; [2]Animal Production Research Institute, Giza, Cairo and [3]Hormones Unit, Chemistry Department, Animal Health Research Institute, Giza, Cairo, Egypt.

THE AIM of this study is to evaluate the effect of gamma (γ) radiations on the safety of processed diets to eliminate the negative effects of aflatoxin-B_1 (AFB_1) on the health of consumers. One hundred fifty adult female Westar rats were used. The rats were divided into 5 experimental groups (G). G_1; rats served as a control which were fed a meal of flesh (breast and thigh muscles) and organs (liver, kidney, spleen and heart) of laying hens that fed before slaughtering diet non-contaminated with AFB_1. G_2; the rats were fed flesh of laying hens fed before slaughtering contaminated diet with 0.2mg $AFB_1 kg^{-1}$, G_3, G_4 and G_5 rats were fed flesh and organs of slaughtered laying hens fed before being slaughtered contaminated diet with 0.2mg AFB_1 and treated with γ-irradiation at 10, 20 and 30kGy, respectively. The feeding continued for 3 weeks (experimental duration), then followed by another 3 weeks period on commercial non-contaminated ration (recovery duration). The obtained results showed that the feed intake, live body weight, blood total protein, albumin, and globulin were significantly decreased ($P \leq 0.05$) in rats of G_2 during the experimental duration compared to G_1. However, an improvement was observed in all parameters for rats of G_3, G_4 and G_5. This improvement was parallel with increasing the dose levels of γ-ray (at 10, 20 and 30kGy), respectively. On the other hand, G_2 showed an increase in the relative organ weights and AFB_1 residues in breast tissues and organs including (liver, kidneys, spleen and heart), creatinine, liver function including aspertate aminotransferase (AST), alanine aminotransferase (ALT) and alkaline phosphatase (ALP). The experimental regimes for rats of G_3, G_4 and G_5 were effective at reducing the deleterious effect of AFB_1 on residues in breast tissues and organs. They normalized the relative organ weights as a function of radiation doses. Moreover, an improvement in serum liver enzymes, thyroid hormones and sexual hormones occurred, which reflects their effects on metabolism and reproductive efficiency of rats and this improvement was proportional with increasing the radiation doses up to 30kGy. It could be concluded that the radiation processing (at the applied radiation doses) of contaminated diet with AFB_1 has significantly improved all parameters ($P<0.05$) which were negatively affected by AFs. Also, improvements were noticed in G_3 and G_4. A greater amelioration was obtained for group G_5 followed a recovery phase.

Keywords: Aflatoxins-B_1, Rat, Fed, Contaminated diet, Safety, Radiation processing.

Introduction

Food and feed safety are fundamental requirements to guarantee human health and there is a worldwide solicitude about the dangers of food poisoning (Chibanga et al., 2014). Food and feed are susceptible to be contaminated with mold species belonging to *Aspergillus* genus, mainly *Aspergillus flavus* and *Aspergillus parasiticus*. These moulds are capable of producing toxic secondary metabolites called aflatoxins (B_1, B_2, G_1 and G_2). Among these aflatoxins, aflatoxins B_1 (AFB_1) is the most important member of mycotoxins showing hepatoxic, terategenic, immunosuppressive and potential carcinogenic that pose severe hazards to animal and human

[#]Corresponding author email: alyncrrt@yahoo.com
DOI: 10.21608/ejrsa.2018.2307.1036

health. It is classified by the International Agency for Research on Cancer (IARC) as group 1 carcinogen (IARC, 2002).

Acute or chronic aflatoxicosis in poultry results in diminished egg and meat production, liver necrosis and hyperplasia of the bile duct leading to reduced proteins, fatty acids digestibility (Hussein & Brasel, 2001), and immunosuppressive (Bailey et al., 2006 and Shi et al., 2006). The biological mycotoxins toxicity depends on the type, dose and duration of mycotoxins intake, species, gender, genetic and animal age, general health, immune and nutrition, as well as certain environmental factors (Bryden, 2007). However, when focusing on how AFs play a role in food safety, solicitude should be limited to AFs that are known to be exposed directly by consuming the contaminated commodities or transferred from feed to food of animal origin, as this food represents a significant route of exposure for humans (Bhat et al., 2010).

The permissible level of aflatoxins adopted in most of the world countries is 5ppb as level of human contamination risk (Yosef et al., 2013). AFs residues in food are a major harmful to public health and could potentially create health problems as they suppress the immune response, hepatotoxic and nephrotoxic effects. The problem with AFs does not end with production losses as many of them are readily transferred to the poultry production (egg/meat) (Denli et al., 2009). AF is metabolized in the body into active metabolites and accumulates in egg, muscle tissues, organs of liver, kidney, breast, legs, and gizzard and blood. AFB_1 transfers from feed to food and metabolized into milk as AFM_1 and B_2 in the liver, AFB_1 to aflatoxin B_1 to eggs as aflatoxicol. AFs residues may also appear in body tissues and samples collected from the poultry feed AFs contaminated rations showed detectable levels of AFs in liver and their edible tissues like liver and muscle (Bintvihok et al., 2002).

Since the contamination of food and feed with mycotoxins in particular with AFB_1, is unavoidable, it is important to seek for possible way to prevent it, through the inhibition of aflatoxgenic moulds growth, or by reduction of the presence AFB_1 in food and feed (Kabak et al., 2006).

Food/feed safety is one of the main challenges for technology. Food/ feed irradiation is one among many of available technologies that has been recognized as a reliable and safe method for the preservation of food and feed that contributes to improving the hygienic quality and nutritional value of them (EPA, 2013). Food irradiation is a processing technology that aims at the improvement of food safety and it is a physical method of food processing that involves exposing prepackaged or in bulk foodstuffs to ionizing energy to eliminate the microbial load or to control the presence of fungi and mycotoxins production in food and in feed (Farkas, 1989; Waje et al., 2009 and Markov et al., 2015). The objective of this study is to evaluate and explore the adverse effects of feeding rats on flesh and organs of laying hens fed before slaughtering AFB_1- contaminated diet and irradiated AFB_1-contaminated diet as a sensitive animal model to ensure the safety of processed materials through radiation treatments for consumption. In addition, the study aims to investigate the effect of AFB_1 on the studied indices through AFs free duration (recovery duration).

Materials and Methods

The experimental work of the present study was carried out at the Food Irradiation Department, National Center for Radiation Research and Technology, Atomic Energy Authority, Cairo, Egypt, and the Animal Production Research Institute, Ministry of Agriculture, Dokki, Giza and Animal Health Research Institute, Ministry of Agriculture, Giza, Egypt.

Experimental design

Five groups, of 60 laying hens each ,were contributed in this study. The first group was fed daily on non-contaminated AFB_1 ration and served as a control. The second group was fed contaminated AFB_1 ration (0.2mg AFB_1 kg^{-1}) according to Farag et al. (2017). The third to fifth groups were fed γ-irradiated contaminated ration (0.2mg AFB_1 kg^{-1} irradiated with 10, 20 and 30kGy, respectively). After 3 weeks of feeding, hens in each group were slaughtered and flesh (breast and thigh muscles) and organs (liver, kidney, spleen and heart) were dissected out and dried, grinded and stored frozen as prepared meals (M_1, M_2, M_3, M_4 and M_5, respectively) for the experimental rats. A total number of 150 adult female Westar rats, aged 90 days, weighing around 120±5gm, were divided into five groups of 30 rats each. All groups were adapted for one week under normal ration before starting the

experiment and then fasted for 24 h before feeding with the prepared meals. The first group (G_1) was fed daily on the prepared meal (M_1) of the first control group of hens (non-contaminated ration). The second group of rats (G_2) was fed on M_2 meal of the second group of hens (AFB_1 contaminated ration). The third to fifth groups (G_3, G_4 and G_5) were fed on their analog prepared hens meals (M_3, M_4 and M_5) (AFB_1 contaminated irradiated ration with 10, 20 and 30kGy, respectively). The feeding continued for 3 weeks (Experimental duration) and then followed by another 3 weeks period on commercial non-contaminated ration (Recovery duration) to explore the possibility of reducing and a polished the residue of AFB_1 that may be present in the experimental rats. Indices as feed intake and live body weight of all rats were recorded weekly during the experimental and recovery periods. Three rats in each group were killed (slaughtered) as representative samples and their internal organs (liver, kidney, spleen and heart) were immediately dissected out and weighed. The AFB_1 residues in these samples were measured by HPLC according AOAC (1995).

Blood sampling and hormones assay

Blood samples were collected weekly, 5 rats were chosen randomly in each group. Serum total protein, albumin, globulin, liver enzymes (AST, ALT & ALP) and creatinine were measured by spectrophotometer, using available commercial kits produced by Biodiagnostic, Egypt. Blood serum samples of rats at the end of experimental and recovery duration (the 3rd and the 6th weeks) were tested for hormones determination. All tested hormones were performed by enzyme-linked immunosorbent assay (ELISA) according to manufacturer instructions. Thyroid hormones; triiodothyronine (T_3), thyroxine (T_4) and thyroid stimulating hormone (TSH) and progesterone were determined using kits purchased from Immunospec Corporation. Testosterone and estradiol were determined using kits purchased from Diagnostic Biochem CandaInc .

Statistical analysis

Data were analyzed for all variables using the General Linear Models procedure to test the differences between means using SAS software version 9.1 (SAS Institute, 2004). Means showing significant differences were compared using Duncan's Multiple Range procedure (Duncan, 1955). All statements of statistical significance were based on probability $P< 0.05$.

Results and Discussion

Feed intake and live body weight

The weekly feed intake and the live body weight of rats during the experimental and recovery durations are presented in Table 1. These indices were severely depressed in rats in G_2 during the experimental duration (1st – 3rd week) compared to those in G_3 , G_4 and G_5. On the other hand, these values were increased gradually by the rate of irradiation to reach the same values of the control group (G_1).

These results are in agreement with El-Shewy & Ebrahem (2004), who reported a significant decrease in body weight and growth rate of rats dosed AFB_1 (7.5ug/200g body weight orally) daily for 3 successive weeks (26.84%) than the control group (53.55%). The loss of body weight may be due to improper assimilation or metabolism of feed produced from the hepatotoxic effects of AFB_1 (Ibeh & Saxena, 1998). In addition, lower feed intake and growth rate might be due to the decreased activity of important enzymes in the carbohydrates digestion, proteins, lipids, and nucleic acids and impaired and defects in some of the nutrient. The reduction in protein synthesis was affected by AFs may due to disruption of transcription mRNA and transport amino acid, thus protein synthesis and DNA were prevented (Thaxton et al., 1974). The improvements in feed intake and body weight gain by irradiation treatments of AFB_1- contaminated diets may due to residual degradation of AFB_1 levels in diets which was reflected in laying hens' tissues and organs and the degradation level increased with increasing the level of γ-radiation. In the recovery phase (4th, 5th and 6th weeks), a slight improvement of these indices was recorded in G_2, with a significant increase ($P < 0.05$) in G_3, G_4 and G_5 (Table 1).

Relative organ weights

The relative weights of some organs including liver, kidneys, spleen and heart to live weights of rats in different groups are presented in Table 2. The relative weight of these organs in G_2 were significantly increased ($P <0.05$) compared to those of the control (G_1). The effect was directly proportion with the duration. Treatment of the laying hens' diet with γ-radiation at different doses up to 30kGy reduced the toxic effects of AFB_1 on the relative weights of rats' organs. It is concluded that γ- irradiation at dose level of 30kGy normalized the relative weight of

organs in corresponding to the control group. Increased liver weight was probably due to the fat deposition consequently to impair fat metabolism. Liver is considered the target organ for AFB_1 because it is the organ where most aflatoxins are bioactivated to the reactive 8, 9-epoxide form, which is known to bind DNA and proteins, damaging the liver structures and increasing liver weight (Pasha et al., 2007). Another explanation for increased weight of liver, fatty liver is mainly mediated by inhibiting synthesis of cholesterol and phospholipids, hence the fat transported through hepatic tissue (Manegar et al., 2010). In addition to the irritation of gastrointestinal mucosa by aflatoxins, provoking inflammation and thickening of the wall (El-Ghany et al., 2013). Increased relative weight of kidneys was probably due to lipaemia (increased fat deposition) (Sharghi & Manafi, 2011). Lower relative weights of the liver, spleen and kidneys in rats challenged with only AFs compared to untreated control rats could be due to necrosis and reduced density of lymphoid cells (Perozo & Rivera, 2003). During the recovery period (4^{th}–6^{th} week), the relative weight of the rat organs in G_5 reached the nearest normal weights of the control group (G_1).

TABLE 1. Feed intake and live body weight of the experimental rats in different treated groups (T) and during experimental duration (D) and recovery duration (R).

Duration (week)		Experimental treated groups (T)	Feed intake (g)	Live body weight (g)
Experimental duration (D)	1^{st}	G_1	222.30^{a} ±1.28	134.00^{a} ±1.15
		G_2	117.63^{e} ±3.1	116.33^{d} ±1.45
		G_3	123.86^{d} ±1.29	121.66^{c} ±0.88
		G_4	143.30^{c} ±0.75	126.66^{b} ±0.33
		G_5	162.40^{b} ±0.43	131.66^{a} ±0.88
	3^{rd}	G_1	246.00^{a} ±0.96	144.33^{a} ±1.76
		G_2	117.70^{d} ±1.48	114.66^{d} ±0.81
		G_3	117.40^{d} ±2.75	118.66^{c} ±0.85
		G_4	129.03^{c} ±0.33	121.33^{c} ±0.66
		G_5	135.53^{b} ±0.57	127.66^{b} ±0.66
Recovery duration (R)	4^{th}	Commercial non-contaminated diet with AFB_1	263.56^{a} ±1.18	151.00^{a} ±0.57
			118.40^{d} ±0.64	114.00^{d} ±1.00
			119.26^{d} ±1.26	118.33^{c} ±0.33
			224.80^{c} ±2.59	120.66^{b} ±0.33
			230.93^{b} ±0.27	122.33^{b} ±0.88
			274.43^{a} ±0.91	159.33^{a} ±0.88
	5^{th}		123.60^{e} ±2.23	119.00^{e} ±0.57
			133.20^{d} ± 2.85	125.33^{d} ±0.88
			144.90^{c} ±1.85	129.33^{c} ±0.66
			156.36^{b} ±2.60	134.00^{b} ±1.52
			291.33^{a} ±1.99	165.66^{a} ±1.20
	6^{th}		139.43^{e} ±1.75	126.33^{e} ±1.20
			159.03^{d} ±4.27	132.33^{d} ±1.45
			173.03^{c} ±1.03	137.66^{c} ±1.33
			187.36^{b} ±0.95	148.33^{b} ±0.66
			289.43^{a} ±4.67	176.00^{a} ±1.15
			157.63^{e} ±1.54	137.00^{e} ±0.57
			177.20^{d} ±1.27	145.33^{d} ±2.33
			189.73^{c} ± 1.56	157.00^{c} ±1.52
			227.13^{b} ±0.23	162.00^{b} ±1.52

Means of each value in the same column within each duration with the same letter are not significantly different ($P < 0.05$). Each value represents mean±standard error. The P value of Duration (D), Treatment (T), D*T, Recovery (R), Treatment (T), R*T was 0.0001.

TABLE 2. Relative organ weights of the experimental rats in different treated groups (T) and during experimental duration (D) and recovery duration (R).

Duration (week)		Experimental treated groups (T)	Live body weight (g)	Liver (g/100g live body weight)	Kidney (g/100g live body weight)	Spleen (g/100g live body weight)	Heart (g/100g of live body weight)
Experimental duration (D)	1st	G_1	146.33^{a}±2.18	3.26^{e}±0.02	1.49^{d}±0.04	0.68^{d}±0.01	0.75^{d}±0.02
		G_2	122.33^{d}±0.88	4.27^{a}±0.01	1.90^{a}±0.01	1.19^{a}±0.01	1.19^{a}±0.02
		G_3	127.00^{cd}±1.52	4.06^{b}±0.02	1.80^{b}±0.04	1.12^{b}±0.01	1.10^{b}±0.01
		G_4	131.33^{c}±2.60	3.82^{c}±0.03	1.76^{b}±0.01	1.0^{b}±0.02	1.05^{b}±0.01
		G_5	137.00^{b}±1.15	3.54^{d}±0.02	1.62^{c}±0.01	0.98^{c}±0.03	0.86^{c}±0.02
	2nd	G_1	149.66^{a}±2.40	3.30^{e}±0.02	1.47^{e}±0.02	0.66^{c}±0.02	0.68^{e}±0.01
		G_2	114.66^{d}±0.88	4.32^{a} ±0.02	1.95^{a}±0.02	1.28^{a}±0.01	1.27^{a}±0.01
		G_3	117.66^{cd}±0.66	4.16^{b}±0.01	1.82^{b}±0.01	1.21^{ab}±0.06	1.19^{b}±0.01
		G_4	121.33^{c}±0.66	3.86^{c}±0.06	1.74^{c}±0.02	1.17^{ab}±0.01	1.11^{c}±0.01
		G_5	127.00^{b}±1.15	3.67^{d}±0.02	1.66^{d}±0.02	1.11^{b}±0.01	0.88^{d}±0.02
	3rd	G_1	153.66^{a}±2.84	3.31^{e}±0.02	1.40^{d}±0.05	0.67^{c}±0.04	0.69^{e}±0.04
		G_2	114.00^{c}±1.00	4.40^{a}±0.02	1.93^{a}±0.03	1.33^{a}±0.03	1.31^{a}±0.01
		G_3	118.33^{bc}±0.33	4.24^{b}±0.01	1.84^{ab}±0.01	1.28^{ab}±0.04	1.24^{a}±0.01
		G_4	120.33^{b}±0.33	3.88^{c}±0.04	1.78^{b}±0.01	1.21^{b}±0.01	1.16^{b}±0.01
		G_5	122.33^{b}±0.88	3.64^{d}±0.02	1.69^{c}±0.01	0.67^{c}±0.01	0.93^{c}±0.02
Recovery duration (R)	4th	Commercial non-contaminated diet with AFB1	157.00^{a}±3.05	3.35^{e}±0.02	1.43^{e}±0.03	0.69^{c}±0.01	0.71^{e}±0.01
			119.00^{d}±0.57	4.30^{a}±0.01	1.85^{a}±0.01	1.23^{a}±0.04	1.21^{a}±0.01
			125.33^{c}±0.88	4.13^{b}±0.01	1.74^{b}±0.01	1.22^{a}±0.03	1.15^{b}± 0.01
			129.33^{bc}±0.66	3.67^{c}±0.02	1.67^{c}± 0.02	1.13^{a}±0.02	1.02^{c}± 0.01
			134.00^{b}±1.52	3.55^{d}±0.01	1.59^{d}±0.01	0.97^{b}±0.03	0.80^{d}± 0.01
	5th		160.33^{a}±1.76	3.33^{e}±0.03	1.39^{e} ±0.01	0.65^{e}±0.02	0.68^{d}±0.01
			126.33^{e}±1.20	4.20^{a}±0.01	1.76^{a}±0.01	1.09^{a}±0.02	1.12^{a}±0.03
			132.33^{d}±1.45	3.93^{b}±0.02	1.66^{b}±0.01	0.97^{b}±0.02	1.03^{b}±0.01
			137.66^{c}±1.33	3.61^{c}±0.02	1.56^{c}±0.01	0.83^{c}±0.01	0.93^{c}±0.01
			148.33^{b}±0.66	3.44^{d}±0.01	1.48^{d}±0.02	0.73^{d}±0.01	0.73^{d}±0.01
	6th		173.00^{a}±2.08	3.31^{d}±0.02	1.39^{d}±0.01	0.65^{d}±0.01	0.65^{d}±0.01
			133.33^{d}±0.88	3.89^{a}±0.03	1.66^{a}±0.01	0.91^{a}±0.01	0.95^{a}±0.01
			148.00^{c}±2.30	3.77^{b}±0.02	1.57^{b}±0.01	0.82^{b}±0.01	0.88^{b}±0.01
			157.00^{b}±1.52	3.54^{c}±0.01	1.48^{c}±0.03	0.74^{c}±0.02	0.76^{c}±0.01
			168.66^{a}±1.85	3.34^{d}±0.01	1.39^{d}±0.01	0.67^{d}±0.02	0.67^{d}±0.01

Means of value sample in the same column within each duration with the same letter are not significantly different (P <0.05). Each value represents mean±standard error. The P value of Duration (D), Treatment (T), D*T, Recovery (R), Treatment (T), R*T was 0.0001.

AFB_1 residues in organs

The detectable amount of AFB_1 residues in the rats breast tissues and organs of G_2 were significantly higher ($P < 0.05$) than those in G_1, G_3, G_4 and G_5 (Table 3). In general, AFB_1 residues were decreased dramatically in these organs in parallel with elevation of the radiation dose up to 30kGy. It is concluded that the residual levels of AFB_1 were diminished by γ-radiation processing of contaminated diets, as a physical detoxifying agent. These results are agreed with Zaghini et al. (2005) who reported that AFB_1 residue was found in the livers of laying hens fed 2.5mg/kg AFB_1 diet for four weeks. Residue levels may be different owing to the type of animal and diet, however, the concentrations of AFB_1 and the length of exposure, and enhanced tolerance to AFs (Bennett & Klich, 2003). During the recovery phase, AFB_1 residues in different organs were decreased with increasing the duration especially for those rats in groups (G_3, G_4 and G_5) AFB_1 residual in breast tissues and organs until there were no detectable residues could be seen.

TABLE 3. AFB_1 residues content in breast tissues and organs of the experimental rats in different treated groups (T) and during experimental duration (D) and recovery duration (R).

Duration (week)		Experimental treated groups (T)	Breast tissues ($ng.g^{-1}$)	Liver ($ng.g^{-1}$)	Kidney ($ng.g^{-1}$)	Spleen ($ng.g^{-1}$)	Heart ($ng.g^{-1}$)
		G1	**ND**	**ND**	**ND**	**ND**	**ND**
		G_2	$0.80^{a}\pm0.04$	$0.91^{a}\pm0.01$	$0.76^{a}\pm0.02$	$0.75^{a}\pm0.01$	$0.85^{a}\pm0.01$
	1st	G_3	$0.66^{b}\pm0.02$	$0.80^{b}\pm0.03$	$0.65^{b}\pm0.02$	$0.68^{a}\pm0.03$	$0.73^{b}\pm0.01$
		G_4	$0.55^{c}\pm0.02$	$0.72^{c}\pm0.01$	$0.45^{c}\pm0.02$	$0.52^{b}\pm0.03$	$0.60^{c}\pm0.01$
		G_5	$0.48^{c}\pm0.02$	$0.65^{c}\pm0.02$	$0.32^{d}\pm0.02$	$0.31^{c}\pm0.02$	$0.31^{d}\pm0.01$
		G_1	ND	ND	ND	ND	ND
Experimental duration (D)		G_2	$1.04^{a}\pm0.07$	$1.26^{a}\pm0.01$	$0.96^{a}\pm0.01$	$0.86^{a}\pm0.03$	$1.15^{a}\pm0.01$
	2nd	G_3	$0.88^{b}\pm0.02$	$1.11^{b}\pm0.01$	$0.84^{b}\pm0.03$	$0.75^{b}\pm0.02$	$0.89^{b}\pm0.02$
		G_4	$0.75^{bc}\pm0.02$	$0.87^{c}\pm0.01$	$0.68^{c}\pm0.02$	$0.65^{c}\pm0.02$	$0.69^{c}\pm0.03$
		G_5	$0.66^{c}\pm0.01$	$0.75^{d}\pm0.02$	$0.44^{d}\pm0.03$	$0.46^{d}\pm0.02$	$0.45^{d}\pm0.03$
		G_1	ND	ND	ND	ND	ND
		G_2	$1.47^{a}\pm0.03$	$4.40^{a}\pm0.72$	$1.28^{a}\pm0.05$	$1.05^{a}\pm0.01$	$1.27^{a}\pm0.04$
	3rd	G_3	$1.28^{b}\pm0.03$	$2.61^{b}\pm0.23$	$1.05^{b}\pm0.02$	$0.89^{b}\pm0.01$	$1.10^{b}\pm0.03$
		G_4	$1.13^{c}\pm0.03$	$1.35^{c}\pm0.06$	$0.80^{c}\pm0.01$	$0.76^{c}\pm0.02$	$0.83^{c}\pm0.03$
		G_5	$0.89^{d}\pm0.04$	$1.21^{c}\pm0.02$	$0.57^{d}\pm0.02$	$0.65^{d}\pm0.02$	$0.62^{d}\pm0.02$
			ND	ND	ND	ND	ND
			$1.26^{a}\pm0.02$	$2.52^{a}\pm0.05$	$0.61^{a}\pm0.03$	$0.88^{a}\pm0.01$	$0.98^{a}\pm0.07$
	4th		$1.15^{b}\pm0.03$	$2.23^{b}\pm0.01$	$0.38^{b}\pm0.04$	$0.79^{b}\pm0.01$	$0.80^{b}\pm0.03$
			$0.92^{c}\pm0.02$	$1.20^{c}\pm0.01$	$0.24^{c}\pm0.02$	$0.57^{c}\pm0.02$	$0.61^{c}\pm0.03$
			$0.70^{d}\pm0.02$	$0.94^{d}\pm0.03$	$0.15^{d}\pm0.02$	$0.38^{d}\pm0.02$	$0.36^{d}\pm0.02$
			ND	ND	ND	ND	ND
Recovery duration (R)		Commercial non-contaminated diet with AFB1	$1.07^{a}\pm0.02$	$1.64^{a}\pm0.05$	$0.44^{a}\pm0.01$	$0.74^{a}\pm0.02$	$1.10^{a}\pm0.05$
	5th		$0.78^{b}\pm0.05$	$1.37^{b}\pm0.02$	$0.33^{b}\pm0.03$	$0.63^{b}\pm0.02$	$0.90^{b}\pm0.02$
			$0.58^{c}\pm0.02$	$0.89^{c}\pm0.01$	$0.16^{c}\pm0.01$	$0.43^{c}\pm0.01$	$0.72^{c}\pm0.02$
			ND	$0.27^{d}\pm0.02$	ND	ND	$0.17^{d}\pm0.03$
			ND	ND	ND	ND	ND
			$0.50^{a}\pm0.01$	$0.72^{a}\pm0.04$	$0.32^{a}\pm0.01$	$0.46^{a}\pm0.02$	$0.68^{a}\pm0.05$
	6th		$0.42^{b}\pm0.03$	$0.63^{b}\pm0.03$	$0.20^{b}\pm0.01$	$0.31^{b}\pm0.05$	$0.51^{b}\pm0.04$
			$0.20^{c}\pm0.02$	$0.46^{c}\pm0.02$	ND	$0.16^{c}\pm0.01$	$0.20^{c}\pm0.04$
			ND	ND	ND	ND	ND

-Means of value sample in the same column within each duration with the same letter are not significantly different ($P < 0.05$). Each value represents mean±standard error. The P value of Duration (D), Treatment (T), D*T, Recovery(R), Treatment (T), R*T was 0.0001.
-ND: no detectable.

Serum biochemical attributes

It is well known that, AFB_1 residue in hen's meals has a harmful and stressful effect on liver tissue. AST, ALT and ALP enzymes are famous biomarkers of liver damage. The results of the current study revealed that exposure to AFB_1 residues resulted in a significant increase in AST, ALT and ALP (Table 4).

TABLE 4. Liver enzymes of the experimental rats in different treated groups (T) and during experimental duration (D) and recovery duration (R).

Duration (week)		Experimental treated groups (T)	AST (UL)	ALT (UL)	ALP (UL)
Experimental duration (D)	1st	G_1	37.73^{e}±1.26	56.48^{e}±1.96	27.86^{e}±1.28
		G_2	81.34^{a}±0.68	93.62^{a}±2.52	56.95^{a}±1.17
		G_3	73.34^{b}±2.52	86.12^{b}±1.13	49.64^{b}±0.60
		G_4	60.38^{c}±1.65	80.23^{c}±0.88	42.79^{c}±0.96
		G_5	45.38^{d}±2.24	64.45d±2.20	38.88^{d}±0.50
	2nd	G_1	38.37^{d}±0.60	53.98^{d}±0.33	26.97^{e}±1.19
		G_2	84.83^{a}±1.21	98.43^{a}±1.02	61.75^{a}±0.67
		G_3	68.88^{b}±1.56	79.57^{b}±2.28	44.40^{b}±1.19
		G_4	53.45^{c}±2.48	71.43^{c}±0.68	39.75^{c}±0.52
		G_5	41.75^{d}±0.89	57.21^{d}±3.18	31.62^{d}±0.85
	3rd	G_1	36.50^{d}±0.51	50.37^{d}±1.98	25.49^{d}±1.14
		G_2	77.18^{a}±1.11	90.68^{a}±2.37	73.60^{a}±3.52
		G_3	62.61^{b}±1.60	75.00^{b}±2.35	43.35^{b}±1.21
		G4	47.15^{c}±0.68	65.71^{c}±2.12	34.59^{c}±1.65
		G_5	38.47^{d}±0.68	50.14^{d}±2.12	26.80^{d}±1.65
Recovery duration (R)	1st	Commercial non-contaminated diet with AFB1	35.11^{d}±1.17	45.42^{d}±0.60	25.24^{d}±0.34
			67.85^{a}±1.22	85.78^{a}±2.07	65.91^{a}±0.88
			56.64^{b}±0.87	69.48^{b}±0.89	41.88^{b}±0.90
			47.90^{c}±0.40	59.49^{c}±1.85	33.34^{c}±0.47
			36.73^{d}±0.32	47.35^{d}±1.31	25.34^{d}±0.49
	2nd		35.15^{d}±0.69	44.16^{d}±0.84	22.75^{d}±0.38
			62.84^{a}±0.79	76.53^{a}±1.18	58.52^{a}±1.22
			53.07^{b}±1.15	64.71^{b}±0.54	38.59^{b}±1.57
			41.49^{c}±0.71	54.98^{c}±1.45	30.76^{c}±0.81
			35.70^{d}±0.26	44.08^{d}±1.36	23.77^{d}±1.05
	3rd		34.28^{d}±0.70	42.42^{d}±0.69	23.21^{d}±0.30
			58.44^{a}±1.16	70.93^{a}±0.71	53.01^{a}±1.39
			48.47^{b}±0.86	59.32^{b}±0.75	36.64^{b}±1.54
			40.31^{c}±0.58	51.67^{c}±0.58	29.89^{c}±0.63
			34.64^{d}±0.33	42.56^{d}±1.06	23.18^{d}±1.23

Means of each value in the same column within each duration with the same letter are not significantly different ($P < 0.05$). Each value represents mean ± standard error. The P value of Duration (D), Treatment (T), D*T, Recovery(R), Treatment (T), R*T was 0.0001.

The highest level of liver enzymes observed in G_2 which were reduced with increasing levels of γ-irradiation for those rats of G_3, G_4 and G_5 during the experimental duration, especially at 30kGy (G_5). AST and ALT are cytoplasmic in location and the increased serum levels may be due to leakage of these enzymes into blood stream as a result of autolytic breakdown or cellular damage (Gaskill et al., 2005). Elevated levels of ALT and ALP are suggestive of liver or bile duct disease, while ALT is a prime marker of bile duct epithelial proliferation that is typical of aflatoxicosis (Kramer, 1989). During the recovery duration, an improvement in liver enzymes has been observed in G_3, G_4 and G_5, especially of those in group 5. Blood serum creatinine, protein, albumin and globulin levels in different groups and during the experimental and recovery duration were recorded in Table 5. Serum creatinine value was significantly increased ($P < 0.05$), while serum protein, albumin and globulin levels were significantly decreased ($P < 0.05$) in rats of G_2 fed on hen aflatoxicated meat.

The adverse effects were ameliorated by feeding the rats γ-irradiated hens' meal, particularly in group 5. The increase in serum creatinine level to nephrotoxic action causes renal impairment by destruction of epithelial cells of proximal and distal convoluted tubules and alteration in tubular function (Abu-Aita et al., 2008). While, the decrease in albumin and globulin values, which are commonly in association with the chronic disease (Coles, 1986) may be due to the effect of AFs on protein synthesis and RNA production (Agag, 2004). It was noticed that at the end of the recovery duration all blood parameters especially for those in G_5 were improved.

Blood Serum Hormone Levels

Serum thyroid and sexual hormone values in different groups of rats at the end of the experimental and recovery duration were presented in Table 6. Rats in G_2 which consumed prepared hen's meal of AFB_1 contaminated diet showed a significant increase in TSH and a significant decrease in T_3 & T_4 compared to control group. The significant decrease in T_3 and T_4 has been shown by Salem & Selim (1994), Eraslan et al. (2005) and Hassan et al. (2010) which was in agreement with these results. The levels of blood T_3 and T_4 in all treated groups (G_2 - G_5) were decreased compared to (G_1). The mechanism concerning the decrease in serum T_3 and T_4 levels may be associated with the decrease in blood iodine level, which is essentially significant in the synthesis of these hormones (Markou et al., 2001). It is obvious that AFs residues cause damage of the epithelium cells of the digestive tract (Johri et al., 1990). The T_3 & T_4 in the groups (G_3, G_4 & G_5) received the prepared hen's meal of aflatoxicated and γ-irradiated ration, were tended to reach the values as in the control group. As for the sexual hormones (estradiol, testosterone and progesterone) a significant decrease is clearly shown in group of non-irradiated AFs diet (G_2). These results are in concomitant with those reported by EL-Shewy & Ebrahem (2004), Hasanzadeh et al. (2011) and Adedara et al. (2014). These authors also reported that Aflatoxins are very potent toxins affecting the growth of all animals, delay in genital system growth (Hafez et al., 1982), high disturbances in estrous cycle, reduced pregnancy rate and number of live new born, failure indication and intrauterine death of the foetus (Shapour & Saeedeh, 2013). In this study, the results were confirmed by Kourousekos et al. (2008) who also proposed that AFB_1 has a direct effect either on ovarian secreting cells or on the hypothalamus–hypophysis–ovarian axis. Most authors' hypotheses focus on the fact that AFs may affect the reproductive system by its toxic effect on the liver, where the cellular hepatic damage could inhibit enzyme synthesis and/or enzyme activity, inhibition of lipid metabolism or fatty acid synthesis, which is essential for synthesis of precursor molecules for AFs may influence the reproductive system by its toxic effect on the liver, where the cellular hepatic damage could inhibit enzyme synthesis and/or enzyme activity, inhibition of lipid metabolism or fatty acid synthesis, causing decreased synthesis of precursor molecules of gonadal as well as gonadotropic hormones (Handan & Guleray, 2005).

The improvement in thyroid and sexual hormones of G_3, G_4 and G_5 may be due to effectiveness of γ-radiation in degradation of AFB_1 for those hens fed irradiated contaminated diets that leads to decrease of aflatoxin residue in prepared hens' meals. An improvement was noticed also in a recovery period through keeping the same trend as experimental duration.

The amelioration in most tested parameters in both experimental and recovery periods are radiation dose dependant. The 30kGy dose is safe and effective for modulation of AFB_1 deleterious effects on poultry production and could protect consumers health.

TABLE 5. Creatinine, serum proteins of the experimental rats in different treated groups (T) and during experimental duration (D) and recovery duration (R).

Duration (week)		Experimental treated groups (T)	Creatinine (mg.dl^{-1})	Total protein (g.dl^{-1})	Albumin (g.dl^{-1})	Globulin (g.dl^{-1})
Experimental duration (D)	1st	G_1	1.37^{d}±0.01	7.73^{a}±0.06	4.30^{a}±0.12	3.43^{a}±0.09
		G_2	1.71^{a}±0.01	4.70^{e}±0.09	2.47^{e}±0.05	2.22^{c}±0.06
		G_3	1.62^{b}±0.02	5.44^{d}±0.03	2.96^{d}±0.02	2.48^{b}±0.01
		G_4	1.58^{b}±0.01	5.94^{c}±0.04	3.27^{c}±0.09	2.67^{b}±0.07
		G_5	1.49^{c}±0.01	6.60^{b}±0.08	3.91^{b}±0.05	2.69^{b}±0.10
	2nd	G_1	1.35^{e}±0.01	7.86^{a}±0.06	4.34^{a}± 0.11	3.52^{a}±0.15
		G_2	1.78^{a}±0.02	4.51^{e}±0.06	2.31^{e}±0.02	2.80^{b}±0.05
		G_3	1.57^{b}±0.02	5.30^{d}±0.02	2.85^{d}±0.01	2.63b^{c}±0.04
		G_4	1.50^{c}±0.02	5.77^{c}±0.02	3.14^{c}±0.02	2.45^{cd}±0.01
		G_5	1.41^{d}±0.01	6.40^{b}±0.09	3.59^{b}±0.04	2.20^{d}±0.08
	3rd	G_1	1.36^{d}±0.02	7.74^{a}±0.04	4.31^{a}±0.10	3.43^{a}±0.07
		G_2	1.87^{a}±0.02	4.40^{e}±0.07	2.25^{e}±0.02	2.14^{d}±0.05
		G_3	1.52^{b}±0.01	5.0^{d}±0.07	2.68^{b}±0.01	2.39^{c}±0.06
		G_4	1.43^{c}±0.01	5.54^{c}±0.03	2.94^{c}±0.02	2.60^{b}±0.05
		G_5	1.37^{cd}±0.01	6.00^{b}±0.10	3.29^{b}±0.04	2.70^{b}±0.05
Recovery duration (R)	4th	Commercial non-contaminated diet with AFB1	1.33^{d}±0.01	7.64^{a}±0.18	4.39^{a}±0.09	3.25^{a}±0.28
			1.81^{a}±0.03	4.91^{d}±0.08	2.47^{e}±0.04	2.44^{b}±0.06
			1.50^{b}±0.01	5.44^{c}±0.06	3.01^{d}±0.02	2.42^{b}±0.04
			1.40^{c}±0.01	6.03^{b}±0.08	3.32^{c}±0.02	2.71^{b}±0.08
			1.36^{cd}±0.01	6.34^{b}±0.04	3.65^{b}±0.07	2.68^{b}±0.08
	5th		1.34^{c}±0.01	7.73^{a}±0.04	4.36^{a}±0.05	3.37^{a}±0.09
			1.75^{a}±0.02	4.95^{e}±0.08	2.62^{e}±0.05	2.33^{c}±0.11
			1.46^{b}±0.01	5.56^{d}±0.02	3.21^{d}±0.03	2.35^{c}±0.02
			1.38^{c}±0.01	6.12^{c}±0.02	3.48^{c}±0.02	2.63^{b}±0.03
			1.33^{c}±0.01	6.43^{b}±0.05	3.71^{b}±0.04	2.71^{b}±0.08
	6th		1.32^{c}±0.01	7.80^{a}±0.09	4.42^{a}±0.05	3.38^{a}±0.14
			1.69^{a}±0.01	5.23^{e}±0.07	2.81^{e}±0.05	2.42^{c}±0.08
			1.42^{b}±0.01	5.78^{d}±0.01	3.36^{d}±0.02	2.41^{c}±0.02
			1.35^{c}±0.01	6.38^{c}±0.02	3.69^{c}±0.02	2.68^{b}±0.03
			1.32^{c}±0.01	6.88^{b}±0.04	4.01^{b}±0.06	2.87^{b}±0.06

Means of each value in the same column within each duration with the same letter are not significantly different ($P < 0.05$). Each value represents mean±standard error. The P value of Duration (D), Treatment (T), D*T, Recovery (R), Treatment (T), R*T was 0.0001.

TABLE 6. Serum blood of TSH, T_3, T_4, estradiol , testosterone and progesterone of the experimental rats in different treated groups (T) and during experimental duration (D) and recovery duration (R).

	Duration (week)	Experimental treated groups (T)	TSH (µIU/ml)	T3 (ng/ml)	T4 (µg/dl)	Estradiol (pg/ml)	Testosterone (ng/ml)	Progesterone (ng/ml)
Experimental duration (D)	3rd	G_1	0.17^{e}±0.01	3.49^{a}±0.11	21.54^{a}±0.45	478.00^{a}±2.88	2.40^{a}±0.11	1.33^{a}±0.02
		G_2	0.92^{a}±0.03	1.73^{e}±0.11	13.53^{e}±0.46	239.66^{e}±4.37	0.41^{e}±0.02	0.57^{e}±0.03
		G_3	0.55^{b}±0.01	2.28^{d}±0.02	15.82^{d}±0.34	290.66^{d}±3.75	0.92^{d}±0.02	0.96^{d}±0.01
		G_4	0.41^{c}±0.02	2.54^{c}±0.05	17.46^{c}±0.16	343.66^{c}±6.88	1.30^{c}±0.05	1.07^{c}±0.03
		G_5	0.30^{d}±0.02	3.11^{b}±0.01	19.23^{b}±0.05	429.33^{b}±3.17	1.86^{b}±0.03	1.22^{b}±0.02
Recovery duration (R)	6th	Commercial non contaminated diet with AFB1	0.33^{d}±0.02	3.68^{a}±0.04	24.01^{a}±0.62	480.00^{a}±0.57	2.47^{a}±0.07	1.34^{a}±0.01
			0.95^{a}±0.01	2.33^{e}±0.03	17.01^{d}±0.41	281.67^{d}±2.33	0.93^{d}±0.03	0.72^{e}±0.02
			0.90^{a}±0.02	2.82^{d}±0.03	19.50^{c}±0.27	334.00^{c}±6.80	1.27^{c}±0.02	1.0^{d}±0.01
			0.68^{b}±0.01	3.21^{c}±0.02	21.50^{b}±0.07	391.33^{b}±1.45	1.76^{b}±0.03	1.19^{c}±0.01
			0.53^{c}±0.02	3.57^{b}±0.02	23.53^{a}±0.39	470.67^{a}±3.52	2.44^{a}±0.02	1.30^{b}±0.01

Means of each value in the same column within each duration with the same letter are not significantly different ($P < 0.05$). Each value represents mean± standard error. The P value of Duration (D), Treatment (T), D*T, Recovery(R), Treatment (T), R*T was 0.0001

Conclusion

AFB_1 is a major toxic contaminant of foods and feeds. The results of the present study suggest that treatment of feed with γ-irradiation at up 30kGy is safe and helpful to reduce the toxicity of AFB_1 as well as improving the feed intake, live body weight, internal organ weights and blood biochemical attributes in the rats without any negative changes. These findings suggest that γ-irradiation treatment seemed to be successfully used safely to prevent any adverse effects of AFB_1 at concentration level 0.2mg/kg in contaminated feed of lying and subsequently, produced safe animal products for consumers.

References

Adedara, I.A., Nanjappa, M.K., Farombi, E.O. and Akingbemi, B.T. (2014) Aflatoxin B_1 disrupts the androgen biosynthetic pathway in rat Leydig cells. *Food Chem Toxicol*. **65**, 252-259.

Abu-Aita, Nashwa A., Mansour, Mogda K., Moneir, Samer M. and Nada A.A. (2008) Influence of the probiotic pediococcus acidilactici on chromosomal aberrations, clininicopathological alterations and immunological changes in aflatoxicated rabbits. *Vet. Med. J. Giza,* **56.**

Agag, M. (2004) Mycotoxins in foods and feeds 1-aflatoxins. *Ass. Univ. Bull. Environ. Res.* **7**(1).

AOAC (1995) Aflatoxins in corn and peanut butter. In: "*Official Methods of Analysis of AOAC International*", Cunniff, P (Ed.), Vol. 2., AOAC International Gaithersburg, MD (Method 990, 33), 18.

Bhat, R., Ravishankar V.R. and Karim, A.A. (2010) Mycotoxins in food and feed: Present status and future concerns. Comprehensive Reviews in *Food Science and Food Safety*, **(9)**, 57-81.

Bailey, C.A., Latimer, G.W., Barr, A.C., Wigle, W.L., Haq, A.U., Balthrop, J.E. and Kubena, L.F. (2006) Efficacy of montmorillonite clay (NovaSil PLUS) for protecting full-term broilers from aflatoxicosis. *J. Appl. Poult. Res*. **15**, 198-206.

Bennett, J.W. and Klich, M. (2003) Mycotoxins. *Clinical Microbiological Review,* **16**(3), 497-516.

Bintvihok, A., Thiengnin, S., Doi, L. and Kamagai, S. (2002) Residues of aflatoxin in liver, muscles and eggs of domestic Fowls. *J. Vet. Sci*. **6**, 1037-1039.

Bryden, W.L. (2007) Mycotoxins in the food chain: Human health implications. *Asia. Pac. J. Clin Nutr*. **16**(1), 95-101.

Chibanga, J.F., Nyirenda, B.D. and Simbaya, J. (2014) Effect of supplementing different levels of vitamin A to aflatoxin B_1 contaminated diets on the performance of broiler chickens. *Asian J. Natur. Appl. Sci*. **3**(4), 35-45.

Coles, E.H. (1986) "*Veterinary Clinical Pathology*" 4th ed., pp.10-79. W.B. Saunders Co., Philadelphia.

Denli, M., Blandon, J.C., Guynot, M.E., Salado, S. and Perez, J.F. (2009) Effects of dietary AflaDetox on performance, serum biochemistry, histopathological changes, and aflatoxin residues in broilers exposed to aflatoxin B_1. *Poult.Sci.* **88,** 1444-1451.

Duncan, D.B. (1955) Multiple range and multiple F tests. *Biomet.* **11**, 1.

El-Ghany, W.A.A., Hatem, M.E. and Ismail, M. (2013) Evaluation of the efficacy of feed additives to counteract the toxic effects of aflatoxicosis in broiler chickens. *Inter. J. Anim. Vet. Advan*. **5**, 171-182.

El-Shewy, E. and Ebrahem, M.F. (2004) Ameliorative effect of vitamin E against the toxicity of aflatoxin B_1 on rats with special reference to its effect on male fertility, *1rst Ann. Confr.* , FVM., Moshtohor, Sept, pp. 189-213.

EPA (Environmental Protection Agency) 402-F-12-002. (2013) EPA's Radiation Protection Standards.

Eraslan, G., Essiz, D., Akdogan, M., Sahindokuyucu, F., Altintas, L. and Hismiogullari, S.E. (2005) Effects of dietary aflatoxin and sodium bentonite on some hormones in broiler chickens, *Bull. Vet. Inst. Pulawy,* **49**, 93-96.

Farag, M., Diaa El-Din, H., Abdalla, E.A., Abdul Azeem, A.M and Ahmed, Nashwa A.H. (2017) The Use of radiation to suppress the impact of aflatoxin B_1 contaminated diets on the productive performance and immunological response of laying hens. *Arab J. Nucle. Sci. and Appli*. **50**(2), 162-180.

Farkas, J. (1989) Microbiological safety of irradiated foods - Review. *Intl J. Food Microbiol*. **9**, 1-15.

Gaskill, C.L., Miller, L.M., Mattoon, J.S., Hoffmann, W.E., Burton, S.A., Gelens, H.C.J., Ihle, S.L., Miller, J.B., Shaw, D.H. and Cribb, A.E. (2005) Liver histopathology and liver serum alanine aminotransferase and akaline phosphatase activities in epileptic dogs receiving phenobarbital. *Vet. Pathol*. **42**, 147-160.

Hafez, A.H., Megalla, S.E., Abdel-Fattah, H.M. and Kamel, Y.Y. (1982) Aflatoxin and aflatoxicosis. II. Effects of aflatoxin on ovaries and testicles in mature domestic fowl. *Mycopathlogica,* **77**(3),137-139, 9.

Handan, U. and Guleray, A. (2005) Selenium protective activity against aflatoxin B_1 adverse effects on *Drosophila melanogaster*. *Braz. Arch. Biol. Technol*. **48**, 2.

Hasanzadeh, S., Hosseini, E. and Rezazadeh, L. (2011) Effects of aflatoxin B_1 on profiles of gonadotropic (FSH and LH), steroid (testosterone and 17β-estradiol) and prolactin hormones in adult male rat Iran. *J. Vet. Res., Shiraz University,* **12**(4), 332-336.

Hassan, A.A., Rashid, M.A. and Koratum, K.M. (2010) Effect of aflatoxin B_1, zearalenone and ochratoxin A on some hormones related to fertility in male rats. *Life Sci. J*. **7**(3), 64-72.

Hussein, H.S. and Brasel, J.M. (2001) Toxicity, metabolism, and impact of mycotoxins on humans and animals. *Toxicology,* **167**(2), 101-134.

IARC (2002) Some traditional herbal medicines, some mycotoxins, naphthalene, and styrene, *IARC Monographs,* **82**, Lyon, IARC.

Ibeh, I.N. and Saxena, D.K. (1998) Effect of alpha-tocopherol supplementation on the impact of aflatoxin B_1 on the testes of rats. *Exp.Toxic.Pathol.* **50**, 221-224.

Johri, T.S., Sadagopan, V.R., Shrivastava, M.A. and Jumdar, S. (1990) Effect of dietary aflatoxin on the performance of purebred broiler chicks. *Indian J. Anim. Sci*. **60**, 1246-1248.

Kabak, B., Dobson, A.D.W. and Var, I. (2006) Strategies to prevent mycotoxin contamination of food and animal feed: A review. *Crit. Rev. Food Sci. Nutr*. **46**, 593-619.

Kourousekos, G.D., Theodosiadou, E., Ververidis, H., Belibasaki, S., Boscos, C., Saratsis, P. and Lymberopoulos, A.G. (2008) Effect of aflatoxin B_1 administration on serum steroid hormones

concentrations of goats. *Reproduction in Domestic Animals*, **43**, 102.

Kramer, J.W. (1989) Clinical enzmology. In: "*Clinical Biochemistry of Domestic Animals*", (4th ed.), Kaneko, J.J. (Ed.), *Academic Press, San Diego*. 338.

Manegar, G. A., Shambulingappa, B. E. and Ananda,K. J. (2010) Studies on tolerance limitof aflatoxin in commercial broilers. *Libyan Agri. Res. Cent. J. Intern*. **1**, 177-181.

Markou, K., Georgopoulos, N., Kyriazopoulou, V. and Vagenakis, A.G. (2001) Iodine-induced hypothyroidism. *Thyroid*, **11**, 501-510.

Markov, K., Mihaljevic, B., Domijan, A.M., Pleadin, J., Delas, F. and Frece, J. (2015) Inactivation of aflatoxigenic fungi and the reduction of aflatoxin B_1 in vitro and in situ using gamma radiation. *Food Control*, **54**, 79-85.

Pasha, T.N., Farooq, M.U., Khattak, F.M., Jabbar, M.A. and Khan, A.D. (2007) Effectiveness of sodium bentonite and two commercial products as aflatoxin absorbents in diets for broiler chickens. *Anim. Feed. Sci. Technol*. **132**, 103-110.

Perozo, F. and Rivera, S. (2003) Effect of aflatoxin B_1 exposure and selenium supplementation on immune response in broilers. *Indian Vet. J*. **80**, 1218-1221.

Salem, M.I. and Selim, M.(1994) Effect of aflatoxin B_1 on thyroid hormones metabolism in young male albino rats. *J. of Environmental Sciences, Mansoura Uinv.* **7**, 141-158.

SAS Institute Inc. (2004) SAS/ STAT 9.1 User's Guide. SAS Institute Inc., Cary, NC, USA.

Sharghi, Y. and Manafi, M. (2011) The additional effects of aflatoxin and T-2 toxin combination on commercial broilers: I: Effects on performance parameters and internal organs. *Research J. Fisher. and Hydrobiol*. **6**, 549-553.

Shapour, H. and Saeedeh, A.(2013) Aflatoxin B_1 effects on ovarian follicular growth and atresia in the rat. *Comp.Clin. Pathol*. **22**, 563-572.

Shi, Y.H., Xu, Z.R., Feng, J.L. and Wang, C.Z. (2006) Efficacy of modified montmorillonite nanocomposite to reduce the toxicity of aflatoxin in broiler chicks. *Anim. Feed Sci. Technol*. **129**, 138-148.

Thaxton, J.P., Tung, H.T. and Hamilton, P.B. (1974) Immunosuppression In thrombocytes during af1atoxicosis. *Poult. Sci*. **58**, 562-566.

Waje, C.K., Jun, S.Y., Lee, Y.K., Kim, B.N., Han, D.H., Jo, C. and Kwon, J.H. (2009) Microbial quality assessment and pathogen inactivation by electron beam and gamma irradiation of commercial seed sprouts. *Food Contr*. **20**, 200-204.

Yosef, T.A., Al-Julaifi, M.Z., Salah-El-Dein, W.M. and AL-Rizqi, A.M. (2013) Assessment of aflatoxin M1 residues in raw cow milk at Al-Riyadh area with reference to some detoxification applications. *Life Sci. J*. **10**, 3365-3369.

Zaghini, A., Martelli, G. and Roncada, P. (2005) Mannanoligosaccharides and Aflatoxin B_1 in Feed for Laying Hens: Effects on Egg Quality, Aflatoxins B_1 and M_1 Residues in Eggs and Aflatoxin B_1 Levels in Liver. *Poult. Sci*. **84**, 825-832.

(Received 23/12/2017;
accepted 7 / 2 /2018)

السلامه الصحية و الأمان السمي للحوم الدجاج البياض المغذي على علائق ملوثه بالافلاتوكسين ب$_1$ و مشععة

محمد ضياء الدين حامد فرج، ايهاب أحمد عبدالله*، علي محمد عبد العظيم و نشوي احمد حسن احمد**
قسم بحوث تشعيع الأغذيه - المركز القومي لبحوث وتكنولوجيا الإشعاع- هيئة الطاقة الذرية، *معهد بحوث الإنتاج الحيوانى - الجيزة - القاهره و**وحده الهرمونات - قسم الكيمياء- معهد بحوث صحة الحيوان - الجيزه - القاهره - مصر.

تهدف هذه الدراسة إلى تقييم اثر تناول المستهلك للطيور التي تم تغذيتها على علائق ملوثة بالافلاتوكسين - ب$_1$ (بتركيز 0.2 ملجم/كجم علف) ومقارنتها بالعلائق التي تحتوي علي نفس التركيز من الافلاتوكسين - ب$_1$ ولكن بعد معالجتها باشعة جاما عند جرعات 10 و 20 و 30 كيلوجراى. تم اجراء التجربه علي مرحلتين ثلاثة أسابيع للمعاملة و تليها ثلاثة أسابيع للاستشفاء. استخدم عدد 150 من إناث الفئران تم تقسيمها الى خمس مجموعات كل مجموعة تحتوى على عدد 30 فآر كالتالي:- المجموعة الأولى: تغذت على لحوم الدواجن (لحم الصدر ولحم الفخذ) وكذلك الأعضاء (الكبد - الكليه - الطحال - القلب) للدجاج البياض الذي تغذي قبل ذبحه على عليقه غير ملوثة ب الأفلاتوكسين ب-$_1$ المجموعة الثانية: تغذت على لحوم الدواجن و بعض الأعضاء الداخلية للدجاج البياض الذي تغذى قبل ذبحه على عليقه ملوثة بافلاتوكسين ب$_1$ بتركيز 0.2 ملجم/كجم علف بدون معالجه بالإشعاع. المجموعة الثالثه و المجموعه الرابعه و المجموعه الخامسه تغذت على لحوم الدواجن و بعض الأعضاء الداخلية للدجاج البياض الذي تغذى قبل ذبحه على عليقة ملوثة بالأفلاتوكسين ب$_1$ ومعرضة لاشعة جاما (10 و20 و 30 كيلوجراى على التوالي). واظهرت النتائج بعد عمليه ذبح الفئران أن مجاميع الفئران التي تغذت على لحم الدجاج وعلى اعضاء الدجاج البياض الذي تغذي قبل الدبح على علائق ملوثة بالافلاتوكسين ب$_1$ ومشععه عند 30 كيلو جراي حدث لها تحسن في كمية الغذاء المستهلك وبالتالي تحسن في الوزن وعدم تغير في وزن الأعضاء (الكبد - الكليه - الطحال - القلب) وحدوث ايضا انخفاض في نسبه المتبقيات من الافلاتوكسين ب$_1$ في الاعضاء المختلفه (الكبد- الكليتين -الطحال و القلب) للفئران مقارنه بالمجموعة الضابطة و قد لوحظ أنه في فتره الإستشفاء حيث تغذت جميع المجاميع على عليقة غير ملوثة حدث تحسن ملحوظا في وظائف الكبد و الكلى و مستويات هرمونات الغدة الدرقية و الهرمونات الجنسية مما يعكس التحسن في التمثيل الغذائي و الكفاءة التناسلية جراء استخدام تقنية التشعيع للحد من مخاطر تلوث الأعلاف بالأفلاتوكسين. نستنتج من هذه الدراسة أن استخدام المعالجة الإشعاعية حتى 30 كيلو جراي قد قلل من التأثير الضار للأفلاتوكسين ب$_1$ ولم يظهر له تأثيرات سلبيه على الحيوان وبالتالي على المستهلك.