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Effect of Turmeric (*Curcumin longa*) Powder on Performance, Oxidative Stress State and Some of Blood Parameters in Broiler Fed on Diets Containing Aflatoxin B1

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Abstract: This research was carried out to evaluate the efficacy of Turmeric powder (*Curcumin longa*) on performance traits, oxidative stress state and some of blood parameters in broiler fed diets containing different level of aflatoxin B1. In this study, CM was used as an antioxidant, to ameliorate the negative effects of aflatoxin B1 (AFB1) in broiler chicks. A total of 240 one-d-old male broiler was allocated in a completely randomized design (CRD) with four replicate. The chickens were divided into 6 groups, including one control and 5 experimental. The control group was fed commercial broiler feed free from aflatoxin (AFB1) and *curcumin longa*, while another the experimental groups, namely, groups 2, 3, 4, 5 and 6 were containing, respectively, T2) control+0.3 g CM; T3) control +1 ppm AFB1; T4) control + 1AF + 0.3 g CM; T5) BD +1AF+0.6 g CM; T6) control +1AF 0.9 g CM during study (1-42days). Performance parameters as weekly recorded and at the 42 days calculated. Results showed that, the addition of 0.3 and 0.6 g/kg CM ameliorated adverse effects of AFB1 diet and improved growth performance. Liver weight had Increased in birds fed on AFB1 ($P<0.05$) and was significantly reduced with the addition of 0.3, 0.6 and 0.9 g/kg CM to the diet contains AFB1 ($P<0.05$). The inclusion of 0.6 and 0.9 g/kg CM Continuous Improvement the adverse affects of AFB1 on serum metabolites such as, total protein, albumin, globulin, uric acid and blood glucose of broiler. Also, the decreased antioxidant defense due to AFB1 was also alleviated by supplemented diet with 0.6 and 0.9 g/kg CM. so, on the basis of this results we can concluded that the addition of 0.6 or 0.9 g/kg CM to the 1.0 mg/kg AF diet demonstrated maximum antioxidant activity against AFB1.

Abreviation: CM: *Curcumin longa* · AFB1: Aflatoxin B1 · BD: Basal diet
Key words: Aflatoxin B1 · Performance · Oxidative Stress enzymes · *Curcumin longa*

INTRODUCTION

Aflatoxins (AFs) are synthesized by certain fungi, namely the *Aspergillus flavus* and *A. parasiticus* species, which are included in the disfurocumarocyclopentanon series and cause intoxication even if taken at low doses. Although there are numerous AF types, AF B1, B2, G1, G2 are the most commonly known and studied ones. Poultry are included among the most susceptible animal species to AFs [1, 2]. *Aspergillus flavus*, are major contaminants of common feed ingredients used in poultry rations [3]. Poor on-farm storage of feeds is a primary reason for aflatoxicosis in farm animals [4]. Exposure occurs predominantly by the ingestion of contaminated feed, when contaminated cereals such as corn, wheat, peanuts and sorghum, as well as other raw materials, are used in the preparation of animal feed [2]. Aflatoxin B1 (AFB1) is the most biologically active form of AF and causes poor performance, liver lesions and immunosuppressant in poultry [5, 6]. Negative effects of AFB1 include cell damage, release of free radicals and lipid per oxidation [7]. Since lipid per oxidation plays a major role in the toxicity of AF, a protective effect of antioxidants is possible [8, 9]. *Curcuma longa* is a medicinal plant native to the Asian subcontinent and cultivated in the tropical regions, is known to possess antimicrobial and antioxidant properties. The powder of dried roots and rhizomes of turmeric are used as one of the spices in Indian curries and other cuisine. Traditionally, it has been used to treat various diseases/disorders e.g. liver obstruction, jaundice,

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ulcers, inflammation, dysentery, diabetes, stomach disorders, fresh wounds, insect stings and viral infections including chickenpox and smallpox [10]. Plant extracts have been found to have antioxidative [11], antimutgenic [12] and imunomodulatory [13]. A number of pharmacological activities of *Curcuma longa* have been reported which include nematocidal [14], anti-inflamatory [15], internal and external injuries [16] and food additives on aflatoxininduced mutagenecity and hepatocarcinogenecity [12]. Yello wish pigments present in turmeric powder, have shown antioxidant/protective effects against AFB1 [12, 17]. Most antioxidants have a dose-dependent efficacy against free radicals and hence need to be evaluated accordingly [18]. The objectives of this present was:

- To introduce graded levels of *curcumin longa* and using it as an ameliorating aflatoxicosis in poultry nutrition.
- The results of this research may be determine dose of AF that in broiler chickens was caused oxidative stress and applied it in nutrition management.

MATERNALS AND METHODS

Experimental Design and Birds: A total of 240 1-day-old (Ross 308) male broilers was purchased from a commercial hatchery (Kurdistan poultry farm), weighed, wing banded and assigned to experimental pen with 1.5 ×1×1 m dimensions. The chicks were maintained on a 24 h (23L=1D) continuous light regime and allowed *ad libitum* access to feed and water from 22 to 42 days. The temperature of the shed ranged optimum towards the end the experiment. A completely randomized design was used with six treatment and 3 replicates of 15 chicks assigned to each of six dietary treatments.

Experimental Diets: A maize-soybean meal-based basal diet (mash and crumble form) was formulated to meet the nutritional requirements of broiler grower (22-42d) as recommended by the National Research Council (NRC1994 [19]) (Table 1). Dietary treatments evaluated included:

T1 (control with out any additive)
T2 (BD+0.6g/kg CM)
T3 (BD+1.0 ppm AFB1)
T4 (BD+1.0 AFB1+0.3g/kg CM)
T5 (BD+1.0 AFB1+0.6 g/kg CM)
T6 (BD+1.0 AFB1+0.9g/kg CM

Table 1: Ingredient composition and calculated analysis of the basal diet

Ingredient	Composition (%)
Maize	59·38
Soyabean meal	34·61
Maize oil	5·89
Dicalcium phosphate	1·03
Limestone	0·75
Salt	0·20
DL-Methionine	0·19
premixture1	0.5
Total	100
Nutrient composition	
Crude protein (%)	22·32
Metabolisable energy (kcal/kg)	3050
Lys (%)	1·25
Met (%)	0·54
Met + Cys (%)	0·84
Ca (%)	1·00
Available P (%)	0·47

[1]Supplied the following per kilogram of diet: vitamin A, 25,000 IU; vitamin D, 5,000 IU; vitamin E, 12.5 IU; vitamin K, 2.5 IU; vitamin B1, 1.0 mg; vitamin B2, 8.0 mg; vitamin B6, 3.0 mg; vitamin B12, 15 µg; folic acid, 250 µg; nicotinic acid, 17.5 mg; calcium pantothenate, 12.5 mg; Fe, 80 mg; Cu, 10 mg; Mn, 80 mg; Se, 0.15 mg; I, 0.35 mg.

Commercially available food grade turmeric powder (*Curcuma longa*) containing an analysed CM content approximately, 81.30% AFB1, 10.40% AFB2, 5.75% AFG1 and 2.55% AFG2 [17]. Dietary AFB1 concentrations were confirmed by analysis [20]. All diets were screened for the presence of citrinin, T-2 toxin, vomitoxin, zearalenone, fumonisins and ochratoxin A [21, 22] before the start of the experiment and were found to be negative and side effect that caused error in current research.

Sample Collection: On day 42, all birds were weighed by pen and total feed intake recorded for each it's. Average feed intake was corrected for mortality when calculating feed conversion for each cage by considering the total bird days. Four birds from each treatment were selected as randomly, weighed and growth performance determined.

Biochemical Analysis: Before slaughtering bird samples, about 5 ml blood sample take from vein wing. Blood was centrifuged at 3000 g for 150 min and serum was separated and preserved at -20°C until submitted for biochemical analysis. Serum samples were analyzed for glucose, total protein, albumin, globulin, aspartate aminotransferase (AST) (EC 2.6.1.1), alaninaminotrasferase (ALT), MDA and uric acid and using an auto analyser (Kodak Ektachem Analyser, Eastman Kodak Co.

Rochester, NY, USA). The parameters measured included total antioxidant concentration, superoxide dismutase (SOD) (EC 1.15.1.1), catalase (CAT) (EC 1.11.1.1.6). Erythrocytes were washed three times with both 140 nM NaCl and phosphate buffer (7. 4 pH) [23]. The erythrocyte suspension was haemolysed with mercaptoethanol and used to measure haemolysate MDA and haemoglobin levels and SOD, GSH-Px, CAT. Drapper and HadleyÕs method was used to measure the MDA level of erythrocytes [24], whereas Woolliams *et al.* method was used to detect SOD activity [25], AebiÕs method to detect CAT activity [26], Paglia and ValentineÕs method to detect GSH-Px activity [27].

Statistical Analysis: Data were analyzed by one-way ANOVA using the general linear model procedures of Statistical Analysis System (SAS Institute, Cary, NC, USA) [19]. Cages were used as the experimental unit for all parameters. The means for treatments showing significant differences in the ANOVA were compared using Fisher's protected least significant difference procedure at a significance based on the 0.05 level of probability.

RESULTS

Performance of Broiler Chicks: Performance and liver weights of birds fed dietary treatments are showed in table 2. Feeding a diet with 0.3 g/kg CM alone had no effect on growth with birds performing as well as the controls. Similarly, 0.6 g/kg CM did not affect relative liver weights in comparison to control birds. Bird's fed on 1 ppm AFB1 /kg had significantly lower feed intake and weight gain, Compared with controls ($p<0.05$). Addition of CM (0.3 and 0.6 g/kg) to the AF diet had no effect on feed intake, but significantly increased weight gain and improved feed conversion when compared with birds fed AF alone. Relative liver weight was increased significantly in birds fed AFB1 ($p<0.05$) Compared with control. The addition of all levels of CM significantly ameliorated the increase in relative weight of liver observed in birds fed AF alone ($p<0.05$), but relative liver weights were still heavier than those of control birds.

Serum Parameters: Feeding diets containing 1.0 ppm AFB1/kg to broilers significantly reduced total protein, albumen and globulin ($p<0.05$). But, increased the activities of aspartate aminotransferase (AST) and uric acid content in the serum (Table 3and 4) ($p<0.05$). Supplementation of 0.6 g/kg CM to the AFB1 diet significantly improved the serum values of total protein, albumen, globulin and aspartate aminotransferase compared with chickens fed AFB1 alone or those fed AFB1 plus 0.3 or 0.9g/kg CM ($p<0.05$). Birds fed diets containing CM with or without AFB1 had significantly lower serum glucose concentrations in comparison to control ($p<0.05$).

Table 2: Performance of broilers fed diets containing total curcumin longa (CM) and aflatoxin (21-42 d)*

treatment	Feed intake(g/bird)	Bodyweight (g)	Feed conversion ratio (FCR)	liver weight (%)
T1 (Control)	2367a	1926a	1.22	2.21c
T2 (BD+0.3g/kg CM)	2250b	1932a	1.16	2.61b
T3 (BD+1 ppm AFB1)	2142ab	1761c	1.28	3.45a
T4 (BD+1 AFB1+0.3g/kg CM)	2189ab	1832b	1.19	3.13a
T5 (BD+1 AFB1+0.6 g/kg CM)	2178ab	1945a	1.11	2.75b
T6 (BD+1 AFB1+0.9g/kg CM	2054c	1823bc	1.12	2.61b
SEM	1.71	47.21	0.36	0.27

*Mean values within a column with unlike superscript letters were significantly different (P<0·05).

BD: Basal diet; ppm, parts per million; AFB1, Aflatoxin B1.

Table 3: Effect of curcuminoid (CM) and aflatoxin on blood parameters of broilers*

Blood metabolites1	T1	T2	T3	T4	T5	T6	SEM
Glucose (g/l)	228.09	202.12	223.06	192.98	192.34	209.3	9.46
Total protein (g/l)	26.12	25.01	17.21	21.23	22.09	16.43	1.1
Albumin (g/l)	9.6	8.2	5.61	7.1	7.83	7.1	0.57
Globulin (g/l)	14.5	13.33	10.46	11.33	11.09	11.87	0.68
Uric acid (mg/l)	55.43	42.1	59.88	52.32	49.76	53.47	5.39

*T1. BD (Control): T2. BD + 0.3g CM; T3. BD + 1.0 ppm AFB1; T4. BD + 1·0 AFB1+0.6g CM; T5. BD + 1.0 ppm AFB1 + 0.6g CM ; and T6. BD + 1·0 AFB1 + 0.9 CM

[1]Mean values within a row with unlike superscript letters were significantly different (P < 0·05).

Table 4: Effect of diets with total curcuminoid (CM) and aflatoxin on oxidative enzymes in liver of broilers*

Blood parameters1	T1	T2	T3	T4	T5	T6	SEM
AST (U/L)	221b	229b	312	263	251	310	25.19
ALT (U/L)	418.71c	442.32c	601.9a	547.56ab	498.23ab	529.27ab	243.06
Catalase(U/mgHb)	20·6ab	21·7a	18·4c	19·6bc	20·4ab	20·7ab	1·01
SOD (U/gHb)	2254.66d	2369.33cd	2410.00bc	2538.50abc	2732.13a	2631.09ab	723.54
MDA (nmol/gHb)	1619.27c	1528.39c	1795.66ab	1821.03ab	1945.33a	1888.51ab	691.09

*T1. BD (Control): T2. BD + 0.3g CM; T3. BD + 1.0 ppm AFB1; T4. BD + 1·0 AFB1+0.6g CM; T5. BD + 1.0 ppm AFB1 + 0.6g CM ; and T6. BD + 1·0 AFB1 + 0.9 CM

[1]Mean values within a row with unlike superscript letters were significantly different (P< 0.05).

DISCUSSION

Performance of Broiler Chickens: Performance of birds fed AFB1 alone is in agreement with earlier reports of performance-depressing effects of AFB1 [4, 11]. Supplementation of CM at 0.3 and 0.6 mg/kg to the AFB1 diet partially improved the performance of chickens in the present study. Curcumin, the major pigment in CM of turmeric, is known to protect the liver against AFB1 [20] by inhibiting the biotransformation of AFB1 to aflatoxicol in liver [9]. The other beneficial compounds of turmeric are tetrahydro curcumin, niacin, turmer one, curlone and cinnamic acid [21], but are present in very low concentrations and contribute very little to the overall antioxidant activity. Previously, partial protection with 0.3 mg/kg CM against AFB1 has been reported [11]. However, increasing the supplemental levels of CM to 0.6 and 0.6 mg/kg in the present study did not completely ameliorate the toxic effects of aflatoxin. The failure of increased levels of CM to further ameliorate the toxic effects of AFB1 is not unexpected since oxidative damage is not the only mode of action of AFB1. For example, AFB1 has also been shown to decrease the expression of hepatic genes involved in energy production and fatty acid metabolism, detoxification, coagulation and immune protection of broiler chickens [22]. The poor performance of chickens fed the diet containing 0.9 mg/kg CM with AFB1 could be attributed to the pro-oxidant action of curcuminoids at higher concentrations. Some polyphenolic compounds have been reported to exhibit both antioxidant and pro-oxidant functions due to metabolic transformations in the presence of transition metals like Cu and Fe [12, 23]. However, the absence of performance depression in birds fed the diet containing 0.9 mg/kg CM alone suggests an interaction between the metabolites of curcuminoid pigments and AFB1, resulting in much poorer performance in chicks fed the combination of CM (0.9 mg/kg) and AFB1 when compared with lower levels 0.3 and 0.6 mg/kg) of CM supplementation. The beneficial effects observed in the present study are attributed to the CM content of turmeric powder. It should be, however, noted that different turmeric species are known to have different levels of curcuminoids (2–7%) [24] and hence requires analysis before using the commercial turmeric powder as a supplement. Also curcumin is highly sensitive to light, heat and alkaline pH and hence care need to be exercised while using curcumin in feed pellets prepared at 75–80°C (http://www.fao.org/inpho/content/compend/text/ch29/ch29.htm).

Biochemical Blood Parameters: The reduced levels of total protein, albumin, globulin, Ca and increased level of g-glutamyl transferase, aspartate aminotransferase (AST) and uric acid are indicative of the toxic effects of AFB1 on hepatic and renal tissue and the findings are in agreement with previous reports of aflatoxicosis [25, 26]. The positive effect of CM on serum values demonstrated its ameliorating effect against AFB1, as the curcuminoid pigments of turmeric powder possess antioxidant activity against oxidative stress caused by free radicals [27]. Similarly, plant extracts of cumin (*Nigella sativa*), *clove* (*Syzygium aromaticum*) and African nutmeg (*Monodora myristica*) have also been shown to have protective properties against AFB1 in both rats and chickens [28, 29]. The reduction in serum glucose levels in birds supplemented with CM demonstrated a hypoglycaemic effect and this finding is in agreement with an earlier report on the hypoglycaemic effect of curcumin pigment in diabetic rats [30].

Liver Antioxidant Status: The antioxidant status in liver homogenates suggests that supplementation with CM stimulated and imprved the antioxidant system situation in the liver of birds to work against the oxidative damage caused by AFB1. Aflatoxin B1 is a strong carcinogen that forms bring towards the body's central axis with DNA induces cellular oxidative damage [31] and causes lipid per oxidation in liver [32]. Supplementation of root extracts of *Picrorhiza kurroa* and seeds of *Silybum marianum*

enhanced the activity of antioxidant enzymes and reduced peroxide levels in liver of rats fed AFB1 [33]. The carbonyl functional group of curcuminoids from turmeric was shown to be responsible for its antimutagenic and anticarcinogenic action [34]. Moreover, curcumin has been shown to powerfully inhibit superoxide anion generation [35] and biotransformation of AF to aflatoxicol in liver [9]. These findings support the hypothesized action of curcuminoids as antioxidants and the results of the present study suggest that 0.6 g/kg CM provided maximum protection against AFB1. Free radicals, in addition to initiating lipid per oxidation, also release cytoplasmic Ca^{2+} that plays a crucial role in subsequent propagation of tissue injury and hence protection against oxidative stress cannot be completely achieved by the action of radical scavenging antioxidants alone[36]. Supplementation of CM at 0.9 g/kg to the AFB1 diet, although it significantly increased total antioxidant concentration in the liver, did not increase the activity of superoxide dismutase or catalase and hence did not reduce the peroxide levels. The performance of these chickens was similar to those fed AFB1 alone. This finding is supported by the fact that certain naturally occurring polyphenolics like *catechin, galangin* and quercetin have been shown to inhibit lipid oxidation, but also showed a pro-oxidant action during the lag phase of the oxidation due to transition metal (Cu/Fe)-induced generation of free radicals [23,37]. Other phenolics like eugenol (2-allyl-4-methoxyphenol) are modulated as a pro-oxidant or antioxidant under certain circumstances [12]. From the present study, it is concluded that dietary supplementation of 0.6 g/kg CM to a diet containing 1ppmAB1/kg provided the greatest amelioration and demonstrated highest antioxidant activity.

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REFERENCES

1. Smith. J.E., G. Solomon and C. Lewis, 1995. Role of mycotoxins in human and animal nutrition and health. Nat Toxins, 3: 187-192.
2. Smith R.B. Jr, J.M. Griffin and P.B. Hamilton, 1976 Survey of aflatoxicosis in farm animals. Appl. Environ. Microbial., 31: 385-388.
3. Kubena, L.F., R.B. Harvey and T.D. Phillips, 1993. Effect of hydrated calcium aluminosilicates on aflatoxicosis in broiler chicks. Poult Sci., 72: 651-657.
4. Ledoux, D.R., G.E. Rottinghaus and A.J. Bermudez, 1999. Efficacy of hydrated sodium calcium aluminosilicate to ameliorate the toxic effects of aflatoxin in broiler chicks. Poult Sci., 78: 204-210.
5. Surai, P.F., 2002. Natural antioxidants and mycotoxins. In Natural Antioxidants in Avian Nutrition and Reproduction, 1st ed, pp: 455-509.
6. Galvano, F., A. Piva and A. Ritieni, 2001. Dietary strategies to counteract the effects of mycotoxins: a review. J. Food. Protect, 64: 120-131.
7. Aherne, S.A., J.P. Kerry. and N.M. O'Brien, 2007. Effects of plant extracts on antioxidant status and antioxidant induced stress in CaCo-2 cells. Br. J. Nutr., 97: 321-328.
8. Coulombe, R.A., J.A. Guarisco and P.J. Klein, 2005. Chemoprevention of aflatoxicosis in poultry by dietary butylated hydroxytoluene. Anim. Feed. Sci. Technol., 121: 217-225.
9. Lee, S.E., BC. Campbell and J. Russell, 2001. Inhibitory effects of naturally occurring compounds on aflatoxin B1 biotransformation. J. Agric. Food. Chem., 49: 5171-5177.
10. Soni, K.B., M. Lahiriand P. Chackradeo, 1997. Protective effect of food additives on aflatoxin-induced mutagenicity and hepatocarcinogenicity. Cancer Lett., 115: 129-133.
11. Gowda, N.K.S., DR. Ledoux and G.E. Rottinghaus, 2008. Efficacy of turmeric (*Curcuma longa*), containing a known level of curcumin and a hydrated sodium calcium aluminosilicate to ameliorate the adverse effects of aflatoxin in broiler chicks. Poult. Sci., 87: 1125-1130.
12. Fujisawa, S., T. Atsumi and Y. Kadoma, 2002. Antioxidant and prooxidant action of eugenol-related compounds and their cytotoxicity. Toxicol., 177: 39-54.
13. Antony, S., R. Kuttan and G. Kuttan, 1999. Immuno modulatory activity of Curcumin. Immunol. Invest, 28: 291-303.
14. Shotwell, O.L., CW. Hesseltine and RO. Stubblefield, 1966. Production of aflatoxin on rice. Appl. Microbiol., 14: 425-428.
15. Reif, K. and W. Metzger, 1995. Determination of aflatoxins in medicinal herbs and plant extracts. J. Chromatogr. A., 692: 131-136.

16. Rottinghaus, G.E., B. Olsen and G.D. Osweiler, 1982. Rapid screening method for aflatoxin B1,zearalenone, ochratoxin A, T-2 toxin, diacetoxyscirpenol and vomitoxin. In Proceedings of 25th Annual American Association of Veterinary Laboratory Diagnosticians, Nashville, TN, pp: 477-484. Davis, CA: American Association of Veterinary Laboratory Diagnosticians.
17. Rottinghaus, G.E., C.E. Coatney and H.C. Minor, 1992. A rapid, sensitive thin layer chromatography procedure for the detection of fumonisin B1 and B2. J. Vet. Diagn. Invest, 4: 326-329.
18. Jayaprakasha, G.K., L.J.M. Rao and K.K. Sakariah, 2002. Improved HPLC method for the determination of curcumin, demehoxycurcumin and bisdemethoxy curcumin. J. Agric. Food. Chem., 50: 3668-3672.
19. SAS Institute, 1996. SAS User's Guide: Statistics. Cary, NC: SAS Institute.
20. Soni. K.B., A. Rajna and R. Kuttan, 1992. Reversal of aflatoxin induced liver damage by turmeric and curcumin. Cancer Lett., 66: 115-121.
21. Srimal, R.C., 1997. Turmeric. A brief review of medicinal properties. Fitoterapia, 6: 483-493.
22. Yarru, L.P., RS. Settivari and E. Antoniou, 2009. Toxicological and gene expression analysis of the impact of aflatoxin B1 on hepatic function of male broiler chicks. Poult. Sci., 88: 360-371.
23. Murakami, K., M. Haneda and S. Qiao, 2007. Prooxidant action of rosmarinic acid: transition metal-dependent generation of reactive oxygen species. Toxicol In Vitro, 21: 613-617.
24. Sasikumar, B., S. Syamkumar and R. Remya, 2005. PCR based detection of adulteration in the market samples of turmeric powder. Food Biotechnol., 18: 299-306.
25. Nath, R., P.C. Bisoi and M. Mohapatra,1996. Effect of livol powder on serum enzymes of birds affected by aflatoxin. Indian Vet. J., 73: 304-308.
26. Abdel-Wahhab, M.A. and S.E. Aly, 2003. Antioxidants and radical scavenging properties of garlic, cabbage and onion in rats fed aflatoxin contaminated diet. J. Agric. Food. Chem., 51: 2409-2414.
27. Paglia, P.E., 1967. Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase. J. Lab. Clin. Med., 70: 158-169.
28. Abdel-Wahhab, M.A .and S.E. Aly, 2005. Antioxidant property of *Nigella sativa* (black cumin) and Syzygium aromaticum (clove) in rats during aflatoxicosis. J. Appl. Toxicol., 25: 218-223.
29. Flora, O. and V.O. Taiwo, 2004. Reversal of toxigenic effects of aflatoxin B1 on cockerels by alcoholic extract of African nutmeg, Monodora myristica. J. Sci. Food Agric., 84: 333-340.
30. Sharma, S., S.K. Kulkarni and K. Chopra, 2006. Curcumin, the active principle of turmeric (Curcuma longa), ameliorates diabetic nephropathy in rats. Clin Exp Pharmacol Physiol., 33: 940-945.
31. Imlay, J.A. and S. Linn, 1988. DNA damage and oxygen radical toxicity. Sci., 240: 1302-1309.
32. Shen, H., C. Shi and H. Lee, 1994. Aflatoxin B1 induced lipid peroxidation in rat liver. Toxicol Appl Pharmacol., 127: 145-150.
33. Rastogi, R., A.K. Srivastava and A.K. Rastogi, 2001. Long term effect of aflatoxin B1 on lipid peroxidation in rat liver and kidney: effect of Picroliv and Silymarin. Phytother. Res., 15: 307-310.
34. Chun, K., Y. Sohn and H. Kim, 1999. Antitumor promoting potential of naturally occurring diarylheptanoids structurally related to curcumin. Mutation Res., 428: 49-57.
35. Iqbal, M., S.D. Sharma and Y. Okazaki, 2003. Dietary supplementation of curcumin enhances antioxidant and phase-I metabolizing enzymes in ddY male mice: possible role in protection against chemical carcinogenesis and toxicity. Pharmacol Toxicol., 92: 33-38.
36. Ichikawa, H. and T. Kanishi, 2002. In vitro antioxidant potential of traditional Chinese medicine, shengmaisan and their relation to in vivo protective effect on cerebral oxidative damage in rats. Biol. Pharm Bull., 25: 898-903.
37. Yoshino, M., R. Tsubouchi and K. Murakami, 2001. Biochemical effects of spice ingredients: structural specificity of antioxidant and prooxidant action. Urakami Found Mem, 9: 38-44.