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PROTECTIVE EFFECTS OF SOME LIPOTROPES AGAINST AFLATOXICOSIS IN BROILER CHICKENS.

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ABSTRACT

An experiment was conducted using 120- 7 days old Cobb chicks to study the possible protective effects of some lipotropes (choline and/or methionine against aflatoxicosis. The used concentration of aflatoxin (Afl) was 200 ug/kg diet to achieve a chronic form of aflatoxicosis. The chicks were fed on a basal diet (BD) and at the age of 7 days old, they were divided into 8 equal groups and fed on eight diets as follows:
BD, BD–Afl, BD-choline, BD-Afl-choline, BD-methionine, BD-Afl-methionine, BD-choline-methionine and BD-Afl-methionine. The experiment was extended for 6 weeks. The obtained results indicated that the addition of choline and/or methionine to Afl-contaminated diets singnificantly improved the suppression in body weight caused by Afl contaminated diet at the last week of the the study. A decreased overall feed intake and an improved feed conversion were noticed in the lipotropes treated groups compared to groups fed on Afl–contaminated diet. The dressing % as well as the relative weights of edible organs were not affected by any of the dietary treatments. However, the liver lipid content showed a highly significant increase in the Afl group while the addition of choline or choline– methionine to Afl contaminated diet caused a significant decrease of liver lipid content. A numerical decrease in the liver lipid content was only observed in the Afl–methionine group. The results also showed that the serum triglycerides level decreased in the group fed on Afl–contaminated diet and increased by choline and / or methionine supplementation to Afl–contaminated diet. The serum alkaline phosphatase level was increased in Afl group and decreased numerically by the addition of methionine and sigificantly by the addition of choline as well as choline and metionine to

the Afl contaminted diet. The liver tissue showed some histopathological changes in the Afl group. No residue traces of Afl was detected in the liver tissue with exception of B_2 and G_2 in the Afl diet. It could be concluded that the addition of lipotropes above the requirements could minimize the adverse effects caused by aflatoxicosis.

Keywords: Broiler, aflatoxins, lipotropes, choline, methionine, liver fat, residue, GOT, Alkaline phosphatase, histopatholgical changes.

INTRODUCTION

Aflatoxicosis is a serious toxicity in poultry resulting from the ingestion of feeds contaminated with aflatoxin; the primary toxic metabolite of the fungus *Aspergillus flavus* as well as *Aspergillus parasiticus*. This condition results in large economic losses to poultry industry. The acute toxic effects include hemorrhaging and death (Muirhead, 1989). While the subacute or chronic forms can adversely affect growth, feed utilization efficiency and the general well-being of broilers (Hamilton, 1984). Aflatoxicosis results in increased bruising in broilers (Tung et al, 1971) from coagulopathy and capillary fragility (Bababunmi and Bassir, 1982) hypoproteinemia (Brown and Abrams 1965) and enlarged hemorrhagic livers with increased lipid content (Fukal et al, 1988). Tung et al (1972) reported that the impaired lipid transport could be attributed to inhibited RNA synthesis which ultimately results in a marked increase in liver lipids but decrease in plasma lipids.

Since contamination by aflatoxins can not be completely avoided, a variety of physical, chemical and biological approaches to remove inactivate or destroy the contaminating toxins have been reported (Newberne et al 1968, Conway et al, 1978, Marth and Doyle 1979). They also reported that aflatoxins content in feeds were reduced after heat treatment or roasting of the contaminated feeds. Detoxfication of aflatoxins by chemicals such as ammonia, sodium bicarbonate, formaldehyde and sodium bisulfite were also reported by many research workers (Mashaly et al, 1983, Elnabarawey, 1986 and Hegazy 1988).

A more economical and practical solution would achived be through the development of dietary additives or modification that would make the brid more resistant to aflatoxicosis. *In-vitro* studies in which methionine was utilized to augment fungal growth had indicated that this amino acid failed to support growth and toxin production of *Aspergillus flavus* (Payne and Hagler, 1983 and Buchanan et al, 1986). There are several reports from *in-vivo* studies indicate that tolerance of aflatoxins can be stablished by considering contents of lipotropic factors in the diets. Follis (1957) reported that animals fed diets low or deficient in lipotropic factors had less hepatic reserve of these lipotropes and this may result in decreased ability to metabolize or detoxify noxious agents. Rogers and Newberne (1967) stated that aflatoxins exert a more pronounced carcinogenic effect on animals fed on low lipotropic diet. Newberne et al, (1968) found that a diet low in lipotropes made the livers of rats more vulnerable to aflatoxin. Sondergard et al (1985) repored that choline deficient enhanced the liver carcinogensis induced by aflatoxin B_1in rats while Hamilton and Garlish (1972) found that a vitamin mixture containing choline chloride, inositol, vitamin E and B_{12} had no effect on the development of the toxic effect of aflatoxin. On the other hand , methionine prevented significantly the depression of weight gains caused by aflatoxin contaminated diet in broiler chickens (Kryukov et al, 1984 and wyatt, 1985) and weaned pigs (Coffey et al, 1989).

The objective of the present experiment was to study the effects of excess dietary lipotropic factors (choline–methionine) as dietary modification to minimize the adverse effects of aflatoxicosis in broiler chickens.

MATERIAL AND METHODS

A total of 120, 7 days old apparently healthy commercial broiler chicks (Cobb) obtained from El Nile Co. were weighed and assigned to eight groups (15 chicks per group). The chicks were reared in electrically heated separated floor pens. A constant lighting program was employed during the whole experimental period. All the recommended vaccinations were adopted.

EXPERIMENTAL DIETS

Two types of broilers diets were formulated, starter and finisher (Table 1) to serve as a basal diet to which contaminated yellow corn, choline

(4000 ppm, and/or methionine (5000 ppm.) were added. Yellow corn was contaminated by an identified strain of *Aspergillus flavus* (2999). The contaminated yellow corn was prepared as described by El–Banna (1987). The experimental diets were prepared to be isonitrogenous and the calorie/protein ratio was kept as constant as possible. The diets with the added toxin were prepared every week to ensure the accurate toxin concentration (200 ug/kg diet). The experimental design is shown in Table (2).

The experimental diets were given *ad-libitum* for the different groups during the 6 weeks of the study period.

MEASUREMENTS

Food consumption and body weight development were recorded on weekly basis and the feed conversion was calcuated. Mortality rates were recorded during the experimental period. Biweekly blood samples were individually collected from birds of each group, separated and sera were analyzed for sGOT using the method described by Reitman and Frankel, (1957), alkaline phosphatase according to Belfield and Goldberg (1971), and plasma triglycerides according to Wieland (1963).

At the end of the experimental period five birds from each group were slaughtered and dressing % abdominal fat, the weight of the liver, heart, gizzard were recorded and calculated in relation to body weight.

The lipid content of the liver was obtained using the equation reported by Mendonca and Jenson. (1983).

$$Y = 102.5 - 1.33 x$$

Where Y represent the lipid content

X is the moisture content of the liver.

Three birds from each group were also taken to determine the aflatoxins residue in the liver according to AOAC (1984). The histopathological examination was done by taking specimens from the liver of three birds from each group and fixed in 10% formalin soluion, dehydrated, cleared and embedded in paraffine wax blocks then sectioned at 5 microns, stained by haematoxlin-eosin stain (Carlton et al, 1967).

Table (1) The composition of the diets used in the study.

Ingredient %	Starter Diet	Finisher Diet
Yellow corn	63.3	74.5
Soybean oil meal (44 % protein)	31	19.8
Meat and bone meal (60 % protein)	3	3
Bone meal	1.6	1.6
Ca Co_3	0.38	0.38
Nacl	0.35	0.35
Vitamin & mineral mixture	0.3	0.3
D-L Methionine	0.07	0.07
ME Kcal / Kg	2910	2994
Cp %	21	17.1
Calorie / protein ratio	138	175

Nutrients supplied per kilogram: Vit. A 7700 IU; Vit. D_3 1650 ICU; Vit. E 5 mg; Vit K_3 30 mg ; Vit. B_2 4.5 mg; Niacin 28 mg ; Pantothenate 6.6 mg; Vit. B_1 15 mg; Folic acid . 44 mg; Biotin 750 mg;choline 60 mg; Vit. B12- 9µg; zinc 60 mg; selenium 1 mg; manganese 75 mg; copper 4 mg; iron 40 mg; menadione sodium bisulfite 2 .2 mg; ethoxyquin 62 mg and iodine 1 mg.

Table (2) Experimental Design of the study

Group	Basal Diet (BD)	Aflatoxin (Afl) 200 ug /kg	Choline 4000 ppm	Methionine 5000 ppm
1-BD	+	-	-	-
2-BD+AFl	+	+	-	-
3-BD+C	+	-	+	-
4-BD+Afl+C	+	+	+	-
5-BD+M	+	-	-	+
6-BD+Afl+M	+	+	-	+
7-BD+C+M	+	-	+	+
8BD+Afl+C+M	+	+	+	+

Table (3) : Effect of chline and/or methionine on body weight development in chickens fed aflatoxin contaminated diets

	BD	BD+ AFl	C	l+C	M	fl+M	+M	+C+M
Lnitial WT First W	139 +8.0	142 ±2.8	141 ±2.3	137 ±5.7	153 ±2.3	138 ±9.2	138 ±8.6	137 ±3.4
Second W	358 ±13.7	307 ±16.5	357 ±9.4	343 ±11.1	371 +9.2	354 ±13.5	346 ±10.1	323 ±12.8
Third W	667 ±28.7	603 ±20.6	676 ±12.8	635 ±14.1	683 +13.9	630 ±11.1	682 ±14.3	596 ±20.2
Fourth W	947 ±22.7	884 ±31.5	918 ±19.8	909 ±19.3	950 +24.9	862 ±35.5	1000 ±26.2	903 ±21.2
Fifth W	1293 ±32.6	1793[a] ±30.7	1305 ±27.3	1304[b] ±24.9	1358 +29.7	1270 ±26.1	1369 ±31.7	1279[c] ±21.9
Sixth W	1635[a] ±50.3	[abc] [d]±34.7	1631 ±47.8	1549[b] ±50.2	1679 ±44.1	1553 ±37.5	1605 ±37.3	1561[d] ±30.4

Values are means + standard error

Values in the same row with the same superscript differ significantly at ($p<0.05$).

Statistical Analysis

The obtained results were statistically analyzed using Student "t" test and one way analysis of variance was performed using minitab computer software, 1986.

RESULTS AND DISUSSION

The experimental design was planned to study the protective effects of liptropes supplementation (choline and / or methionine) against affatoxin induced toxicosis.

1- Body weight development (BW)

Choline and /or methionine supplementation did not signifocantly affect Bw as compared to the basal diet (Table 3) Differences in Bw due to aflatoxin were significant $p<0.05$ at the last two weeks of the experiment. Similarly, Hamilton (1984) found that the chronic form of aflatoxin can affect

growth.

Choline supplementation to aflatoxin contaminated diet (Afl – choline) resulted in a numerical increase in Bw as compared to Afl contaminated diet. The increase in body weight became significant ($p<0.05$) at the last two weeks of the study, as in case of Afl- choline / methionine group. However, Afl- methionine diet resulted in a significant ($p< 0.05$) increase in Bw at the last week of the experiment. Our findings come in agreement with Kryukov et al, (1984) and Wyatt (1985) who stated that increasing methionine level in broiler diet significantly prevented the depression of gain caused by aflatoxicosis.

2- Feed intake and conversion:

Table (4) shows a lower feed intake, a higher gain and feed conversion in the Afl – choline, Afl –methionine and Afl- choline / methionine groups compared to Afl group. The result related to the gain is supported by wyatt (1985). However, kryukov et al, (1984) reported that methionine improved the appetite of birds fed Afl-contaminated diet.

3- Dressing percentages and edible organs weights:

The dressing % and relative weights of edible organs were not affected by any of the dietary treatments (Table 5).

4- Liver fat content:

Table (6) showes that Afl contaminated diet resulted in a highly significant ($p<0.05$) increase in liver lipid contents compared to Afl free diets. Similar results were reported by Tung et al, (1972) and Fukal et al,(1988). Afl-methionine group showed a numerical decrease in liver lipid content, while, Afl-choline and Afl-choline/methionine groups showed a significant ($p<0.05$) decrease in liver lipids. These results are in

Agreements with Newberne and Young, (1966) who observed that rats fed a diet containing lipotropes (choline-methionine), their livers did not accumulate excessive amount of fat.

Table (4) : Effect of choline and/or methionine on overall food consumption, weight gain and feed conversion efficiency in chickens fed on aflatoxin contaminate diets.

	BD	BD +Afl	BD +C	BD+ C	BD+ M	BD+Af l+M	BD+ C+M	BD+Af l+C+M
Food Consumption (g.)	3591	3609	3650	3529	3785	3457	3374	3560
Gain (g.)	1496	1336	1489	1411	1526	1366	1467	1424
Conversion	2.4	2.7	2.45	2.5	2.48	2.53	2.3	2.5

Table (5): Effect of choline and/or methionine on dressing % and elative weight of edible organs in chickens fed aflatoxin contaminated diets

	BD	BD+ AFl	BD+ C	BD+A Fl+C	BD+ M	BD+A Fl+M	BD+ C+M	BD+Af l+C+M
Fat%of the abdomen	. 91 ±.23	.9 ±.38	.79 ±.4	.89 ±.28	1.17 ±28	.84 ±37	.62 ±18	1 ±.44
Relative heart weight	.45. ±.1	.57 ±07	.46 ±.07	.41 ±.03	.40 ±.06	.40 ±.04	44. ±07	39 ±.O5
Relative gizzard weight	2.85 ±.38	3.03 ±.53	3.4 ±.49	3.18 ±.58	2.91 ±.36	3.03 ±.45	3.28 ±47	3.05 ±.38
Ralative liver weight	2.26 ±.2	2.31 ±.28	2.64 ±.28	2.46 ±.23	2.54 ± 17	2.38 ±.39	2.70 ±.39	2.42 ±.41
Dressing %	69.5 ±2.5	70.3 ±3.7	68.2 ±1.9	70.9 ±1.6	72.5 ± 4.7	69.9 ±6	66.6 ±2.5	70.0 ±.9

Values are means + standard error

3- Blood Constituents:

Data of serum triglycerides, sGOT and alkaline phosphatase are presented in table (6). The birds fed Afl contaminated diet showed a sharp decrease in the

Table (6) : Effect of choline and/or methionine on liver fat, serum riglycerides,alkaline phosphtase and SGOT .in chickens fed aflatoxin contaminated diets.

	BD	BD+AFl	BD+C	BD+C	BD+M	BD+AF1+M	BD+C+M	BD+Afl+C+M
Fat%of the liver	9.2 ± 1.7	19.9^{ab} ±3.6	7.83 ±0.8	6.56^{aj} ±0.92	8.43 ±10.	11.2 ± 1.2	8.1 ±.1	10.2^{a} ±1.2
Alkaline ase u/l	445 ±13	498^{ab} ±16	475 ±15	441 ±16	435 ±14	440^{a} ±10	457 ± 17	435^{b} ±1
Triglycerides mg/dl	152 ±20	107^{ab} ±2.5	189^{ab} ± 33	123 ± 7.5	144 ± 6.4	156 ± 6.6	156 ± 21	134^{b} ± 4.1
SGOT u/l	180 ± 13	180 ± 12	195 ± 21	185 ± 19	186 ± 21	185 ± 18	175 ± 15	187 ± 18

Values are means ± stsndard error Values in the same row with the same superscript differ significanrly at ($p < 0.05$).

serum triglycerides. The previous resuilts obtained from liver lipid content and serum triglycerides are closely in agreement with Tung et al,(1972) as well as Hamilton (1977) who reported that the impaired lipid transport due to Afl , caused a marked increase in liver lipids and a decrease in plasma triglyserides.

The Afl-choline group showed a marked increase in serum triglycerides. A significant increase ($p < 0.05$) in serum triglycerides was observed in the groups fed on Afl–methionine and Afl- choline / methionine. This result could be attributed to the used lipotropes which could counteract the impaired lipid transport by preventing accumulation of excess lipids in liver tissue (Newberne and Young,1966). On the other side, Coffey et al, (1989) reported that methionine did not affect serum triglycerides of pigs fed Afl.

The sGOT levels did not affected by any of the treatments. While alkaline phosphatase level incresed in the group fed Afl contaminated diet compared to the other groups. Meanwhile, a significant decrease in alkaline

phosphatase levels in the Afl- methionine and Afl-choline /methionine groups and a slight decrease in its level in Afl-choline group was observed.

These results agree with those previously reported by Lind et al (1967) as well as Varley et al, (1980) who stated that the increase in alkaline phosphatase is an indication of active hepatic cell damage. This finding suggests that the addition of choline and/or methionine to Afl contamdiet can decrease the severity of the toxin on hepatic cells (Rogers and Newberne, 1967 and Sondergard et al, (1985).

4- Aflatoxins Residue in the liver tissue

Since the liver is the principle organ of aflatoxin metabolism (Hsieh, 1981), the liver tissue was used to detect the aflatoxin residue and to study the histopathological changes.

No residues were detected in all the experimental groups with exception of traces of B_2 and G_2 in the liver tissue of the Afl group which received 200 ug/kg diet aflatoxin. This result could be attributed to the low level of the toxin used in this study. This result was supported by those obtained by Teleb and Fakhry (1988).

5- Histopathological Examination

The liver of birds fed Afl diet only showed an obvious increase in the size of some hepatocytes and their nuclei. (megalocytosis) together with diffuse necrobiotic and fatty changes (Fig 1 & 2). These observations were specific to the adverse effect of Afl on the hepatic cells (Newberne et al, 1968 and Sondergard et. al, 1985 as well as El-Banna, 1987).

Liver tissue of the other Afl groups showed only slight vacuolation of hepatocytes and focal aggregation of inflammatory cells, moderate megalocytosis which were non specific to aflatoxicosis (Fig.1&2). These results indicated that increasing the lipotropes in broiler diets can reduce the hepatic cell damage and protect against the adverse effects of aflatoxin on hepatic tissues. (Newberne et al, 1968).

The method by which dietary lipotropes function protect against

Figure (1): Liver showing megalocytosis, vacuolation of hepatocytes and focal areas of necrosis in chicks fed aflatoxin contaminated feed without additives. (H & E stain)

Figure (2): Liver showing a slight vacuolation, megalocytosis of some hepatocytes, no necrobiotic changes in chicks fed aflatoxin contaminated diet and supplemented with choline. (H & E stain)

aflatoxicosis is not completely uncerstood, but their effect can be attributed to the fact that afl is metabolized in the liver by mixed function of oxidase enzyme. Perhaps, increasing the dietary methionine (the most limiting amino acid for poultry) enhances this enzyme leading to an increase in the conversion of Afl into a less toxic metabolite (Smith, 1980). In other words, excess supplementation of dietary methionine may function to protect against the detrimental effect of aflatoxin via glutathione (Mgbodile et al, 1980).Increasing the dietary methionine led to increasing in liver glutathione (Seligson and Rotruck, 1983) which has been demonstrated to suppress the effects of Afl (Mgbodile et al, 1980 and Novi, 1981).

The present study indicated that addition of lipotropes above the normal requirements for optimal performance could minimize the adverse effect caused by aflatoxicosis.

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التأثيرات الواقية لبعض المواد المضادة للتسمم فى تقليل سمية الأفلاتوكسينات فى أفراخ الدجاج

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أجريت التجربة باستخدام عدد ١٢٠ من أفراخ (سلالة الكوب) عمر سبعة أيام لدراسة إمكانية استخدام بعض العوامل المحبة للشحم (الكولين والمثيونين) لحمايتها من آثار التسمم بالأفلاتوكسين باستخدام جرعة قدرها ٢٠٠ جزء فى البليون وذلك لإحداث تسمم مزمن.

غذيت الأفراخ على العليقة الضابطة حتى عمر ٧أيام ثم قسمت إلى ثمانى مجموعات متساوية (١٥ لكل مجموعة) وغذيت على العليقه الضابطة + الأفلاتوكيسينات، المجموعة الثالثة غذيت على العليقة الضابطة + ٤٠٠٠ جزء فى المليون كولين + الأفلاتوكسين، المجموعة الخامسة غذيت على العليقة الضابطة + ٥٠٠٠ جزء فى المليون مثيونين، المجموعة الثامنة غذيت على العليقة الضابطة + ٤٠٠٠ جزء فى المليون كولين + ٥٠٠٠ جزء فى المليون مثيونين + الأفلاتوكسين.

هذا وقد استمرت التجربة لمدة ٦ أسابيع. وقد بينت النتائج أن إضافة الكولين و/ أو المثيونين إلى العلائق الملوثة بالأفلاتوكسين قد حسن معنويا الانخفاض الحادث فى الوزن بسبب تسمم بالأفلاتوكسين فى نهاية التجربة وعند هذه التجربة. وقد لوحظ انخفاض فى استهلاك العلف وتحسن معامل التحويل الغذائى فى المجموعات التى غذيت على علائق مضافاً إليها الكولين والمثيونين إذا ما قورنت بالمجموعة التى غذيت على العلف الملوث بالأفلاتوكسين كما لوحظ عدم تأثر نسبة التصافى وكذلك الوزن النسبى لبعض الأعضاء الداخلية بالمعالجات المختلفة بينما كان هناك زيادة معنوية فى محتوى الكبد من الدهون فى المجموعة التى غذيت على العليقة الملوثة بالأفلاتوكسين فقط بينما أدى إضافة الكولين و / أو المثيونين إلى انخفاض معنوى فى مستوى الدهن بالكبد. كما أوضحت النتائج أيضاً اختلاف محتوى ثلاثى الجلسريد فى أمصال الطيور التى غذيت على العليقة الملوثة بالأفالتوكسين وقد زاد مستواها بإضافة الكولين و / أو المثيونين إلى العلائق. أما إنزيم الفوسفاتيز القلوى فقد زاد فى المجموعة التى غذيت على العليقة الملوثة بالأفلاتوكسين وقد نقص رقمياً بإضافة المثيونين ومعنيا بإضافة الكولين أو المثيونين مع الكولين. هذا وقد لوحظ بعض التغيرات الباثولوجية فى خلايا كبد الطيور فى المجموعة التى غذيت على العليقة الملوثة بالأفلاتوكسين كما لم يستدل على أى بقايا للأفلاتوكسين فى خلايا الكبد باستثناء وجود كمية ضئيلة من السم فى أكباد المجموعة التى غذيت على العليقة الملوثة بالأفلاتوكسين. وقد خلصت النتائج إلى أن إضافة الكولين و/ أو المثيونين بجرعات أعلى من الاحتذياجات الغذائية لعلائق الطيور من الممكن أن يقلل من التأثيرات السلبية نتيجة للتسمم الفطرى بالأفلاتوكسين.