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Histopathologic Studies on Livers in Ducklings Administered Aflatoxin Produced by Korean Industrial Strain of *Aspergillus flavus*

Hwa Joong Yoon, D.V.M., M.S.

Department of Veterinary Medicine, College of Animal Husbandry
Kon Kuk University

Introduction

In 1960 the unexplained deaths of large numbers of turkey poults and ducklings in England were eventually traced to the feeding of a peanut meal diet which had become contaminated with strains of the common mold *Aspergillus flaves*[43]. These toxic groundnut meals also exert adverse effects on pigs and calves[27]. Further details of the investigations carried out on other toxic meals can be found in the papers of Allcroft and Carnaghan[1]. The investigations have revealed that the toxic factor was a metabolite of the fungus *Aspergillus flavus*[40].

The toxic metabolites of *A. flaves* have been called aflatoxin, various components of which have been described. The structure of these components, the blue fluorescent aflatoxion B and the green fluorescent aflatoxin G has been worked out by Asao et al[2]. An LD_{50} of aflatoxin B_1 to ducklings is approximately 18.2 μg and that of aflatoxin G_1 is about 39.2 μg[15].

Several works[4,6~12,16,30,32,33,36,45,46] have noticed pathologic lesions in the liver of laboratory animals, ducklings and chickens which were treated with aflatoxin. In Korea, biologits[17,23~26] have studied about *Aspergillus flavus* and aflatoxin, because Koreans like to consume fermented foodstuffs(fermented soybean mass and fermented soybean paste). Crane et al.[18] reported that many patients of stomach cancer had been taking fermented foodstuffs.

Especially in Korea, many varieties of Aspergillus spp. have grown in the feeds of livestock and in the barns during the rainy and higher humid seasons in spring and summer. The Korean poultry industry has suffered a great economic loss due to Aspergillus spp. during those seasons.

The purpose of this paper is to determine the toxicity of aflatoxin produced by Korean Industrial Strain of the *Aspergillus flavus* as the result of histopathologic studies on the liver of the ducklings which were administered the aflatoxin. In this experiment the growth ratio of the ducklings, the liver index, and various changes in the liver have been studied.

Materials and Methods

The animals used in this experiment were 37-53 grams Korean native breed ducklings. They were placed on the respective experimental regimens within 24 hours after hatching which time water and food were withheld. During the course of this investigation, the ducklings were housed in a standard chick battery brooder at temperatures of 80-95 F with sufficient food and water supplied ad libitum.

All the animals were weighed three times, on the initial day, 3rd day, and 7th day. The dead

ducklings during the experiment were also weighed when we found them. The survivors were killed by decapitation on day 3 and 7 of the experiment. The livers were removed, weighed, and the gross appearances were recorded, and then the livers were placed in 10 per cent neutral formalin solution to be processed for histopathologic study. Distal part of the left liver was cut and processed in a routine manner for paraffin sections of five to ten micromenters in thickness. The sections of the livers were stained with Mayer's hematoxylin and eosin, and frozen section were prepared stain Oil Red O. for the accumulation of large quantities of lipid. All sections were scored using an arbitrary system whereby the degrees of the lesions in the microscopic appearances were classified by the following five criteria,— within normal limits, ± minimal, + slight, ++ moderate, and +++ marked in degree.

Normal feeds used in the experiment were mixed in the department of feed science, college of animal husbandry, Kon Kuk university.

The *Aspergillus flavus*(Strain A-9) was isolated from the fermented soybean mass and then cultured. The aflatoxins that we used in this experiment were produced in the institute of applied microbiology, Kon Kuk university, according to Pons and Goldclatt method[38]. Diets were also prepared with contaminated rice substrates of aflatoxin that were autoclaved at 120 degrees C for 30 minutes. 70% ethanol was used as the diluent of the crystal aflatoxin, because any histopathological changes were not found in the liver of each duckling administered 0.01 ml of 70% ethanol by intubation in the preliminary test. In order to study the lesions by the dose of the toxin and the duration by a single oral intubation or a single feeding, we designed 4 experimental groups: 30 μg, 20 μg, 10 μg and 2μg of the aflatoxins. There were 5 ducklings in each group, and the survivors were decapitated on the 3rd and 7th day of the experiment.

Comparisons of the toxic effects of the aflatoxin B_1 produced by Korean Industrial Strain of the Aspergillus sp. with those of standard aflatoxin B_1 were studied in this experiment. This treatment was divided into 4 groups of 30 μg, 20 μg, 10 μg and 2μ, and there were 5 ducklings in each group. The formulas of growth ratio and liver index in this experiment are as follows: Increased Body Weight/Initial Body Weight, and Liver Weight× 100/Body Weight.

Results

Poisoning with Aflatoxin: The symptoms and gross lesions varied according to the toxicity of the sample which was realted to the survival time of the ducklings and the dose of aflatoxin. Ducklings given 2 μg and 10 μg aflatoxin lost body weight for about 2 days, although they appeared to eat and drink normally. Their condition was good, and there was no evidence of haemorrhages in the liver. By 3-7 days they had recovered their initial weight and continued to increase at a rate comparable to that of the control(Fig. 1).

At the early stages, up to 3 days, the livers

Table 1. Comparison of Growth Ratio after Administration of Three Aflatoxin Preparations

Dosage of Aflatoxin	After 3 Days			After 7 Days		
	Standard Aflatoxin	Korean Aflatoxin	Contaminated* Diet	Standard Aflatoxin	Korean Aflatoxin	Contaminated* Diet
2 μg	1.51	1.15	1.12	2.37	1.94	1.65
10 μg	1.45	1.07	1.02	1.55	1.61	1.39
20 μg	1.10	0.84	1.05	1.51	1.04	1.22
Control		1.71			3.07	

* Feed mixed with Korean aflatoxin

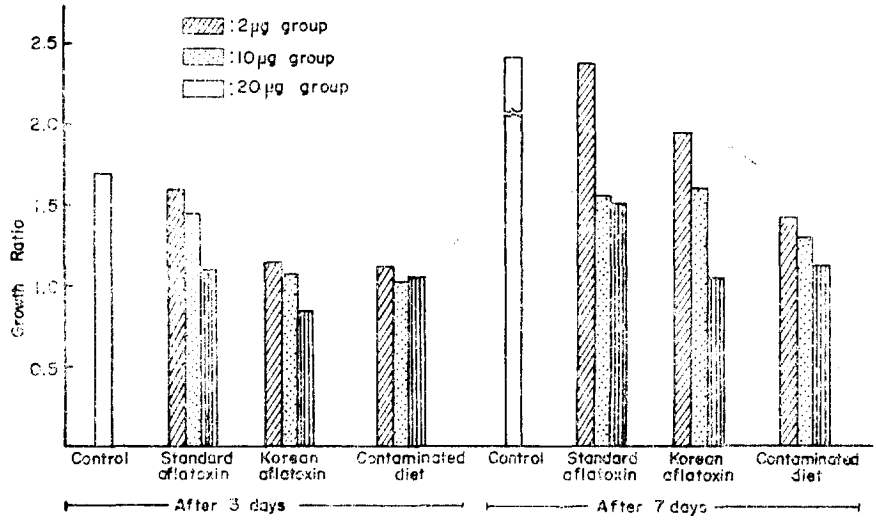


Fig. 1. Comparison of growth ratio after administration of three aflatoxin preparations.

Table 2. Comparison of Liver Index after Administration of Three Aflatoxin Preparations

Dosage of Aflatoxin	After 3 Days			After 7 Days		
	Standard Aflatoxin	Korean Aflatoxin	Contaminated* Diet	Standard Aflatoxin	Korean Aflatoxin	Contaminated* Diet
2 μg	4.4	7.9	6.2	4.3	6.2	5.6
10 μg	5.2	7.0	6.1	5.5	7.3	6.6
20 μg	4.9	7.5	7.3	5.8	7.6	7.8
30 μg	5.9	8.2	7.8	—	—	—
Control		5.0			5.3	

* Feed mixed with Korean aflatoxin

were pale yellow in contrast with the pink ones of the controls, otherwise they appeared to be normal: there were no gross haemorrhages. By 7 days the livers of the experimental animals were indistinguishable from those of the controls. Throughout the duration of the experiment little difference was noted between the body weight ratio of experimental and standard ducklings(Table 1 and Fig.. 1).

After higher doses(20 μg and 30 μg) all the animals lost body weight before death. Typical retardation of growth by aflatoxin B_1 is illustrated in Fig.. 1. Young ducklings developed ataxia followed by convulsions shortly before death and died in the form of opisthotonus. The liver was pale in

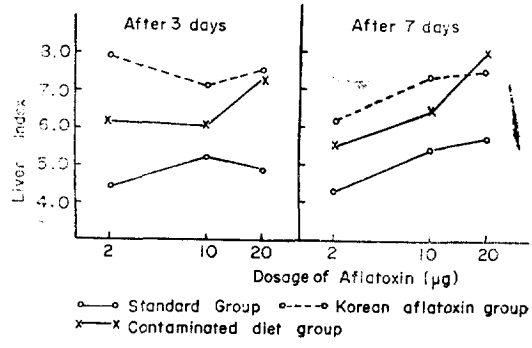


Fig. 2. Comparison of liver index after administration of three aflatoxin preparations.

color and reduced in weight, but there were dark red petechial hemorrhages on the liver, and the liver dead ducklings was slightly enlarged. The

Table 3. Histopathologic Findings of Livers That Were Adminisred Standard Aflatoxin

Tissue Changes \ Dosage of Aflatoxin / Duckling No.		30 μg					20 μg					10 μg					2 μg				
		1*	2*	3*	4*	5*	6*	7*	8	9	10	11	12	13	14	15	16	17	18	19	20
Hemorrhage	Central Area	##	+	+	+	+	+	+	—	—	—	—	—	—	—	—	—	—	—	—	—
	Periportal Area	###	###	###	##	##	##	##	—	—	—	—	—	—	—	—	—	—	—	—	—
Necrosis	Pyknosis	##	##	##	##	+	+	+	±	—	—	±	±	—	—	—	—	—	—	—	—
	Single-nucleoli	+	+	+	±	±	±	+	—	—	—	—	—	—	—	—	—	—	—	—	—
	Margination of Chromatin	+	+	+	±	±	±	+	±	—	—	—	—	—	—	—	—	—	—	—	—
Fat Change	Central Area	+	+	+	+	+	+	+	+	+	+	+	+	±	±	±	±	±	±	±	±
	Periportal Area	##	##	##	##	##	##	##	##	##	##	+	+	+	+	+	+	+	+	+	+
Bile Ductule Cell Proliferation	Proliferation of Bile Ductule Cells	+	+	+	+	+	+	+	##	##	##	+	+	+	+	+	±	±	±	±	±
	Formation of Bile Ductules	—	—	—	—	—	—	—	+	+	+	—	—	—	±	±	—	—	—	—	—
	Mitoses of Bile Duct Cells	—	—	—	±	±	±	±	+	+	+	—	—	—	+	±	—	—	—	—	—
Regeneration of Hepatic Cells	Mitoses of Hepatic Cells	—	—	—	—	—	—	—	±	±	±	—	—	±	+	+	—	—	—	—	—

Degree of histopathologic changes — Within normal limits ± Minimal in degree
+ Slight in degree ## Moderate in degree ### Marked in degree * Dead duckling

left lobe of the liver was usually more atrophic than the right lobe.

Histology: After exposure to 30 μg of aflatoxin, Korean aflatoxin kiled all of the animals by 36 hours while standard aflatoxin killed the animals by the 3rd day. The 2 ducklings treated with standard aflatoxin of 20 μg died within 24 hours. The 3 ducklings that were administered Korean aflatoxin died within 72 hours. The 3 ducklings that were administered the feed mixed with Korean aflatoxin also died within 72 hours. In all the dead ducklings, diffuse hemorrhage showed in the sections(Fig.. 3). Sometimes focal hemorrhages were observed in the liver of some of the birds; they varied from conspicuous(Fig.. 4) to small groups of extravasated erythrocytes. They were usually in the sinusoids (Fig.. 5), but they were in the periportal zone and were associated with early bile duct cell proliferation(Fig.. 6). Within 3 days after adminstration of aflatoxin the hepatic parenchymal cells were severely damaged; the most conspicuous sign was pyknosis of hepatic cells(Fig.. 7). There was a single nucleolus(Fig.. 9), margination of chromatin, and marked cytoplasmic vacuolization. Mitotic figures of bile ductule cells were frequently observed(Fig.. 10). Some of the bile ductule cell proliferation showed in the liver of the ducklings that were administered Korean aflatoxin(Table 2). Seʌere batty changes showed in the sections(Fig.. 13). In a few cases severer fatty changes showed in the periportal area than in the central lobes. Marked proliferation of bile ductule ells has progressed in this stage of 20μg dose(Fig. 12). The cells attempted to from duct-like structures were of ten seen in sections(Fig. 13). Occasionally, no mitoses were seen in the parenchymal cells of these experimental groups.

Marked hemorrhages, necroses and fatty changes were conspicuous in all the animals of the experiments. The prompt proliferation of oval cells were more prominent throughout the portal system in Korean aflatoxin than in aflatoxin mixed with feed and standard aflatoxin(Fig. 12). A few mitoses of bile duct cells showed in Korean aflatoxin. No regeneration of parenchymal cells was present.

By 3 days focal hemorrhages were present in the liver of the duckling, No. 13 in table 2. Small

Table 4. Histopathologic Findings of Livers That Were Administered Korean Aflatoxin

Tissue Changes		Dosage of Aflatoxin: 30 μg					20 μg					10 μg					2 μg				
	Duckling No.	1*	2*	3*	4*	5*	6*	7*	8*	9	10	11	12	13	14	15	16	17	18	19	20
Hemorrhage	Central Area	##	##	##	##	##	+	+	+	—	—	—	—	—	—	—	—	—	—	—	—
	Periportal Area	###	###	###	###	##	##	##	##	—	—	—	—	—	—	—	—	—	—	—	—
Necrosis	Pyknosis	###	###	###	##	##	+	+	+	—	—	±	±	—	—	—	—	—	—	—	—
	Single-nucleoli	##	##	##	+	+	+	+	+	—	—	—	+	—	—	—	±	±	—	—	—
	Margination of Chromatin	##	##	+	##	+	+	+	+	—	—	+	+	—	—	—	±	±	—	—	—
Fat Change	Central Area	##	##	+	##	+	+	+	+	+	+	+	+	+	+	+	±	±	±	±	±
	Periportal Area	###	###	##	##	##	##	##	##	##	##	##	##	+	+	+	+	+	+	+	+
Bile Ductule Cell Proliferation	Proliferation of Bile Ductule Cells	±	+	+	±	+	+	+	±	###	###	+	+	##	##	+	+	+	±	±	±
	Formation on Bile Ductules	—	—	—	—	—	—	—	—	##	##	—	—	+	+	±	—	—	—	—	—
	Mitoses of Bile Duct Cells	—	—	—	—	—	—	—	—	##	##	—	—	+	+	+	—	—	±	±	—
Regeneration of Hepatic Cells	Mitoses of Hepatic Cells	—	—	—	—	—	—	—	—	+	+	—	—	±	±	+	—	—	—	±	±

Degree of histopathologic changes — Within normal limits ± Minimal in degree
+ Slight in degree ## Moderate in degree ### Marked in degree * Dead duckling

Table 5. Histopathologic Findings of Livers That Were Administered Feed Mixed with Korean Aflatoxin

Tissue Changes		Dosage of Aflatoxin: 30 μg					20 μg					10 μg					2 μg				
	Duckling No.	1*	2*	3*	4*	5*	6*	7*	8*	9	10	11	12	13	14	15	16	17	18	19	20
Hemorrhage	Central Area	##	##	##	##	##	##	##	+	—	—	±	±	—	—	—	—	—	—	—	—
	Periportal Area	###	###	###	###	##	###	###	##	—	—	+	+	—	—	—	—	—	—	—	—
Necrosis	Pyknosis	###	###	###	###	##	##	##	+	—	—	+	+	—	—	—	—	—	—	—	—
	Single-nucleoli	##	##	##	##	##	+	+	+	—	—	±	±	—	—	—	—	—	—	—	—
	Margination of Chromatin	##	##	##	##	##	+	+	+	—	—	+	+	—	—	—	±	±	—	—	—
Fat Change	Central Area	##	##	##	##	##	##	##	##	+	+	+	+	+	+	+	±	±	±	±	±
	Periportal Area	###	###	###	###	###	###	##	##	##	##	##	##	+	+	+	+	+	+	+	+
Bile Ductule Cell Proliferation	Proliferation of Bile Ductule Cells	±	±	+	±	±	+	+	+	###	###	±	+	##	##	##	±	±	+	+	+
	Formation of Bile Ductules	—	—	—	—	—	—	—	—	##	##	—	—	##	##	##	—	—	—	—	—
	Mitoses of Bile Duct Cells	—	—	—	—	—	—	—	±	##	##	—	—	##	##	##	—	—	+	+	±
Regeneration of Hepatic Cells	Mitoses of Hepatic Cells	—	—	—	—	—	±	±	±	+	+	—	—	±	±	±	—	—	+	+	+

Degree of histopathologic changes — Within normal limits ± Minimal in degree
+ Slight in degree ## Moderate in degree ### Marked in degree * Dead duckling

groups of extravasated erythrocytes were observed in the sinusoids and periportal zone of the liver. Sometimes the erythrocytes were associated with oval cell proliferation. A few cells of pyknosis, margination of chromatin and single nucleolus were seen, but no frank necrosis in the groups of 10 μg aflatoxin. In the groups of 2 μg aflatoxin no necrotic and degenerative cells were observed. Marked changes in fat in the groups of 10 μg aflatoxin and moderate changes in fat in the groups of 2 μg aflatoxin showed the formation of cakes of fat in the parenchymal cells and the proliferating oval cells. Bile duct cell proliferation was present, and small ductules were made by proliferating oval cells. A few mitotic oval cells were present in the periportal system. These appearances showed more frequently in 10 μg aflatoxin than in 2 μg aflatoxin. Slight regeneration and mitoses of parenchymal cells were present around the periportal system in the groups of 10 μg Korean aflatoxin(Fig.. 14). But there were no regeneation and mitoses in the parenchymal cells in the groups of 10 μg standard aflatoxin and all the groups of 2 μg aflatoxin.

There were no observable hemorrhages within 7 days at this stage. There was moderate necrobiosis in the livers of ducklings in the 20 μg Korean aflatoxin(Table 2), and 20 μg aflatoxin contaminated feed, and slight necrobiosis in the livers of ducklings. The nuclei of the isolated residual hepatic cells were scattered among the proliferating ductules. The nuclei were enlarged, round, and contained a single chromatin clump in the center with margination at the nuclear membrane. Mitotic figures were frequent among the hepatic parenchymal cells as well as the bile ductular cells. Diffuse or periportal regeneration of the parenchymal cells waa present in the livers of the ducklings in 20μg, 10 μg, ang 2 μg aflatoxin groups respectively. Marked changes in fat were observed in most of the experimental ducklings. Proliferation of the ductule cells appeared to be explosive; they were present as clumps or arranged into poorly formed ductules with an irregular lumen. The nuclei of the ductule cells were round, oval, vesicular, and contained prominent chromatin clumps. These appearances were severer in the livers treated with Korean aflatoxins and feed mixed with Korean aflatoxin than in those treated with the standard aflatoxin.

Discussion

The various manifestations of toxicitz of the aflatoxins in this experiment were outlined in Table 1. Three toxin preparations resulted in histopathologic lesions were similar in nature but varying in intensity. In most cases, the microscopic changes were accompanied by a decrease in body weight and liver size except in dead ducklings. When the growth rate was compared with that of the controls, obviously the body weight of the dead ducklings was decreased, but the liver size was not decreased. The growth depression was also conspicuous in all of the cases of the sacrificed ducklings.

Newberne et al.[35] showed the growth depression was not a necessary concomitant of the toxic response in the liver of the duckling. Butler[7] has shown that ducklings given 15 μg aflatoxin lost weight for about 2 days, but by 3-4 days they had recovered their initial weight. By 7 days the livers of the experimental ducklings were indistinguishable from those of the controls. Asplin and Carnaghan[3] described that the first signs of disease were inappetence and poor growth rate, and diffuse degenerative changes in the paranchymal cells of the liver were the principal changes, Lancaster[22] showed the weights of rats fed the Brazilian groundnut meal containing aflatoxin were all lower than the controls. According to the report of Carlton et al.[12], the livers were obviously smaller than those of the control ducks, gray in color, and cut with increased resistance as compared with the livers from control birds. This atrophy is reflected in much reduced average liver weights. In this experiment, loss of body weight and liver weight is similar to other expriments, but the livers of dead ducklings within 3 days seems to be slightly increased because of congestion. The liver body weight ratio of the experimental animals was sim-

ilar to that of controls, because the decreasing of the liver weight.

Diffuse and petechial hemorrhages showed in the livers of dead ducklings, and in the sections diffuse andfocal hemorrhages were present. The death of the ducklings occurred after administration of higher doses(over 20 μg aflatoxin).

After higher doses the livers of the animals were putty-colored with a few small areas of hemorrhage, but were not enlarged, and the liver index was similar to that of the controls[8]. Recently hatched ducklings treated with toxic groundnut diedwithin 1 week of feeding. The livers were putty-colored and slightly enlarged from the feeding of the toxic groundnut[8]. At necropsy the livers of both male and female guinea-pigs were congested and very friable: a few macroscopic haemorrhages could be seen.[9]

Necroses of the liver may be divided into two main types. The first of these, called zonal necroses, are further subdivided on the basis of the portion of the lobule which is involved. The second main group is the massive necrosis of the liver in which all the liver cells in large areas throughout the liver substance become necrostic.

In zonal necroses, central necrosis of the lobules can follow exposure to such toxic substances as chloroform, carbon tetrachloride and some of the naphthalenes. The necrosis is often preceded by a fatty degeneration of the cells to be affected[43]. In peripheral necrosis, the peripheral zones of the lobules are regularly necrotic. This form is not common but results when strong toxic substances are carried to the lobule by the blood stream without any impairment in circulation of the blood and oxygenation of the cells. The peripheral cells receive the toxic blood first and suffer most from its effects[41].

In this experiment diffuse necrosis originated from periportal necrosis and was shown in the higher doses(over LD_{50}). The prominent microscopical appearances were pyknosis of the hepatic parenchymal cells. Karyorrhexis, karyolysis and margination of chromatin with a single nucleolus were also shown in the parenchymal cells. At a dose level of 10 μg and 2 μg aflatoxin, there were a few karyolysis and margination of chromatin with a single nucleoles by 3 days, but not by 7 days.

In guinea-pigs necrosis of aflatoxin showed more abundant in the central zone[10], but in ducklings it showed around the periportal zones with the formation of lakes of fat[7].

Fat changes have diffusely shown in the liver sections of the experimental poults. In an earlier stage of the administration, the vacuoles of the fat present were smaller than the later stage. They are in the cytoplasm of the hepatic parenchymal cells and in the proliferating bile duct cells.

Bacterial toxins are capable of producing fatty degeneration[39]. The parenchymal cells between the proliferating ducts show fatty degeneration with the formation of lakes of fat; some of the fat is in the biliary epithelium.[7] In paraffin preparations hepatic cells are severely vacuolated, as a result of the accumulation of large quantities of lipid as shown by staining frozen sections with Oil Red O., confirming the presence of lipids in these cells. The young duckling normally has a significant amount of lipid in its liver, but this is increased when the animal is exposed to aflatoxin.[36]

The duckling is a highly sensitive experimental animal to the acute effects of aflatoxin, and the liver show typical microscopic appearances to determine the infection of *Aspergillus flavus*. The typical microscopic appearance is the proliferation of bile duct cells. The biliary proliferation progressed with persistent parenchymal cell necrosis. By 7 days after adminstration of aflatoxin, mitotic figures were present in the parenchymal cells and the biliary proliferation was also present.[34,35] After administration of CCl_4 the appearance of the rat liver was similar to that of the liver fed aflatoxin. By 48 hours mitoses were found in the bile duct cells of the portal tracts, and after one week the bile duct mitotic rate was still higher than normal.[45] Also, bile duct proliferation was present, as well as, bile ductule cell hyperplasia

in dimethylnitrosamine poisoning of ducklings.[12] Asplin et al.[3] have shown in their aflatoxin experiment that rapid and extensive proliferation of bile duct epithelium takes place, being seen as cords of makedly basophilic cells forming tubules radiating from the portal tracts. This change is clearly visible within 6 days of feeding a ration containing 10 per cent Brazilian groundnut meal. As the condition progresses, bile duct proliferation proceeds rapidly until, after a period of about 3 weeks on the diet, only small islands of apparently normal parenchymal cells remain separated and surrrounded by dense masses of bile duct epithelial cells and fibrous tissue.

Throughout this experiment bile ductule cell proliferation and the mitoses were more prominent in the ducklings fed the feed mixed with Korean aflatoxin than the other groups, and prominent by 7 days more than by 3 days.

Regeneration and mitoses of hepatic parenhymal cells were present by 7 days in 20 μg, by 3 and 7 days in 10 μg and a few by 7 days in 2 μg aflatoxin.

By 48 hours mitoses were abundant amongst the parenchymal cells, but were also found in bile duct cells of the portal tracts at a single dose level of 0.05ml CCl_4 per 100 g. body weight of the rat.[45] By 24 hours after exposure to 15 μg aflatoxin the cords of biliary cells are even less distinct, and an active proliferation of parenchymal cells is going on at 14 days.[7] The first change that was seen in the parenchymal cells at about 5 weeks was that a few hepatic cells at the periphery of the lobules were larger than those of the remainder of the liver cords.[10]

The continuing experiments on the other animals will be required, because the histopathologic lesions on the liver of ducklings after administration with aflatoxins are severer than those on the liver of chickens after administration of aflatoxins.[48]

Conclusion

This report describes the comparison of the histopathologic lesions induced in ducklings by a single oral administration or a single feeding of afatoxin produced by Korean Industrial Strain of the *Aspergillus flavus* and feed mixed with the aflatoxin and standard aflatoxin.

The basic histopathologic lesions associated with administration of the aflatoxins consisted of hemorrhages, hepatic parenchyma necroses, and fat changes and proliferation of bile ductule cells. Variations occurred in doses of the aflatoxins and the duration of the experiment.

It is suggested that Korean aflatoxin may be slightly severer than standard aflatoxin, and feed mixed with Korean aflatoxin may be slightly severer than Korean aflatoxin.

Legends for Figures

Fig. 3. The liver of duckling 3 day after intubation of 30 μg Korean aflatoxin. Extravasation of erythrocytes shows throughout the lievr. H & E stain. × 150.

Fig. 4. The liver of a duckling 2 days after intubatoin of 20 μg Korean aflatoxin. Focal hemorrhages are observed in some areas and slight fat changes are shown throughout the liver. H & E stain. ×150.

Fig. 5. The liver of a duckling 3 days after intubatoin of 20 μg Korean aflatoxin. Sinusoids of the liver are distended by erythrocytes and moderate fat changes are shown throughout the liver. H & E stain. ×150.

Fig. 6. The liver of a ducking 3 days after feeding with the feed mixed with 20 μg Korean aflatoxin. Note moderate proliberating bile ductule cells with erythroytes and slight fat changes. H & E stan. ×450.

Fig. 7. The liver of a duckling 3 days after administration of 30 μg Korean aflatoxin. Marked pyknosis of the hepatic cells and slight hemorrhages are shown in this section, and

marked fat changes are observed in the center of this photograph. H & E stain. ×450.

Fig. 8. The liver of a duckling after 3 days intubation of 30 μg Korean aflatoxin. Pyknosis, karyorrhexis and karyolisis of hepatic cells with fat changes are shown. H & E stain. ×450.

Fig. 9. The liver of a duckling after 3 days intubation of 20 μg Korean aflatoxin. proliferating oval cells and single nucleoli of hepatic are observed. H & E stain. ×450.

Fig. 10. The liver of a duckling after 7 days feeding with the feed mixed with 20 μg Korean aflatoxin. Mitotic figures of bile ductule cells are shown and proliferating oval cells attempted to form duct-like structures. H & E stain. ×450.

Fig. 11. The liver of a duckling after 7 days intubation of 10 μg standard aflatoxin. Moderate proliferation of bile ductule cells is shown. H & E stain. ×150.

Fig. 12. The liver of a duckling after 7 days feeding with the feed mixed witih 20 μg Korean aflatoxin. Marked proliferation of bile ductule cells is observed. H & E stain. ×150.

Fig. 13. The liver of a duckling 7 days after administration of 20 μg Korean aflatoxin. Marked proliferation of bile ductule cells is observed. H & E stain. ×150.

Fig. 14. The liver of a duckling 7 days after administration of 10 μg Korean aflatoxin. A mitotic figure of a hepatic cell is observed in the center of this photograph. H & E stain. ×450.

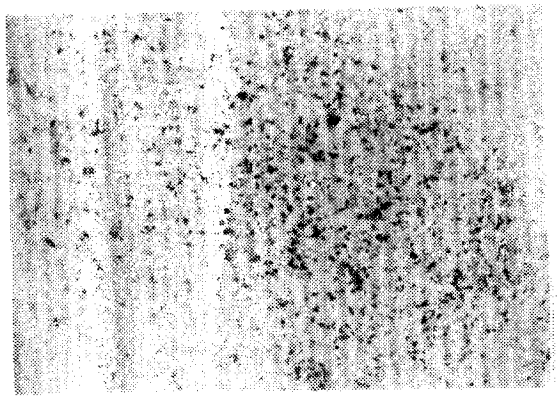


Fig. 3.

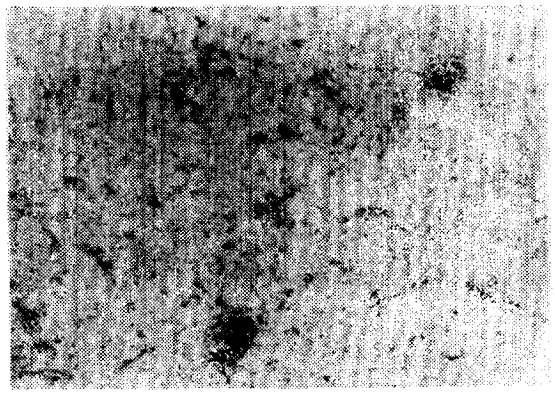
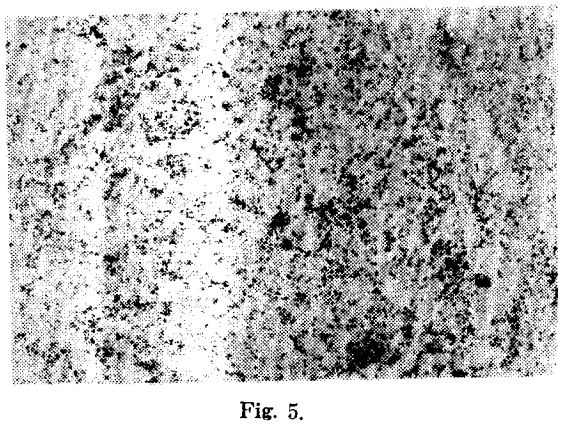


Fig. 4.



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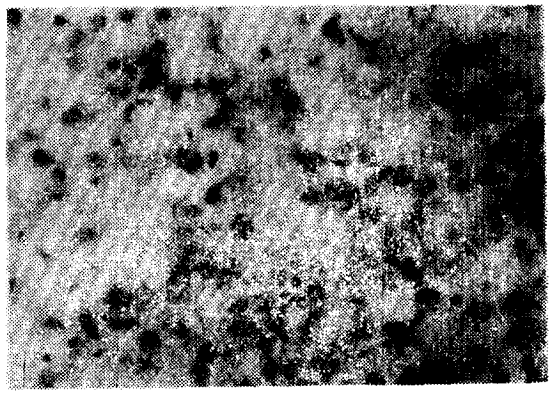

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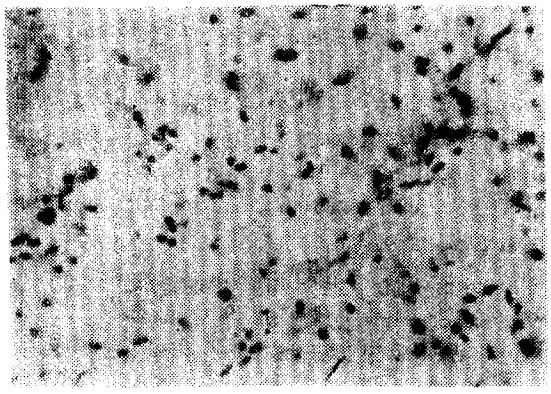

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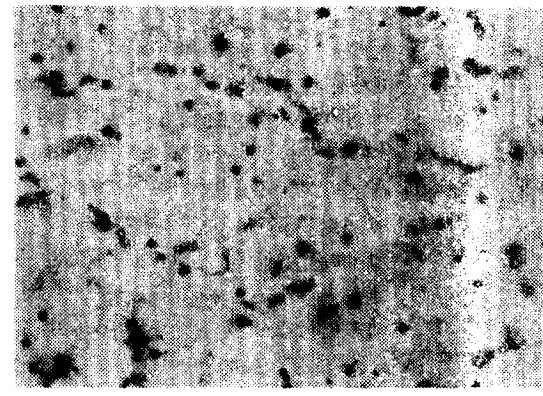

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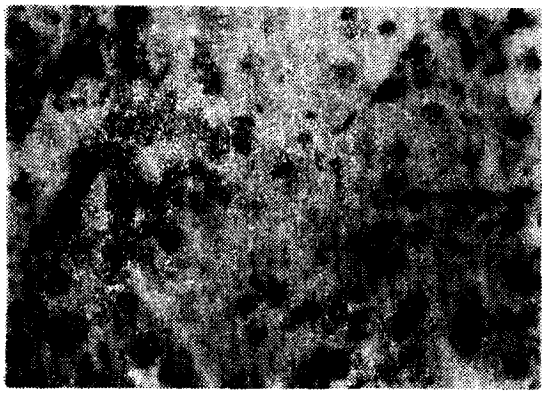

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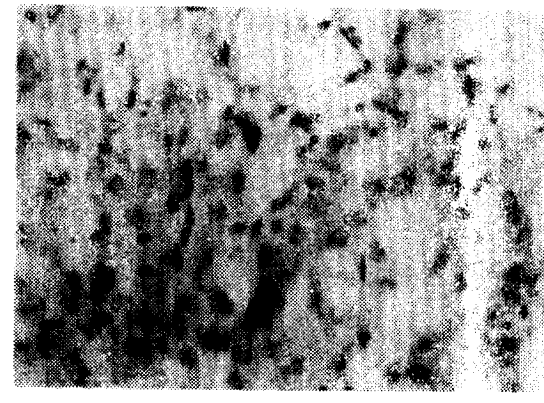

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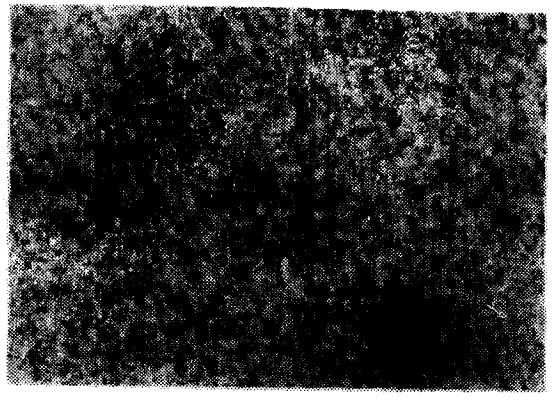

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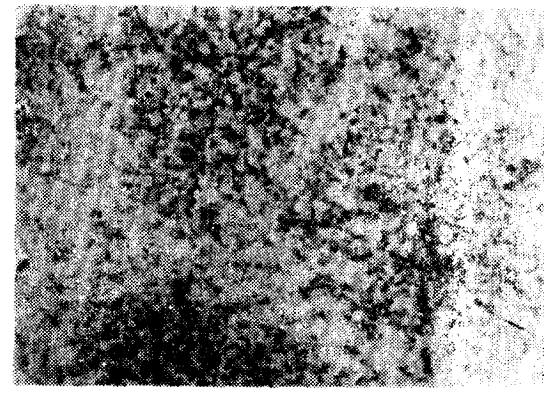

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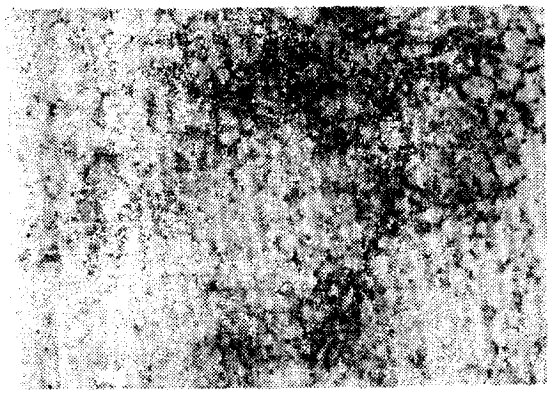

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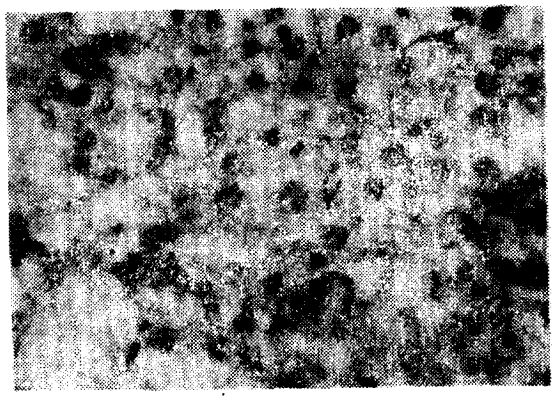

Fig. 14.

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오리병아리의 肝臟에서 한국산 Aflatoxin이 유발시킨 病變에 관한 病理組織學的硏究

윤 화 중

건국대학교 축산대학 수의학과

국 문 초 록

한국산 aflatoxin의 독성을 비교 규명하기 위하여 결정 aflatoxin, 사료배합 aflatoxin 및 표준 aflatoxin을 오리병아리에 각 1회씩 경구투여 또는 급식시켜 얻은 결과는 다음과 같다.

1. 각종 aflatoxin의 독성으로 인하여 유발된 공통된 주요 병리조직학적 병변은 出血, 肝細胞의 壞死, 脂肪變性 및 膽管細胞의 增殖 등이였다.

2. 病變의 程度는 독소의 투여량과 경과시간에 따라 多樣하게 나타났다.

3. 한국산 aflatoxin의 독성은 표준 aflatoxin에 비해 약간 더 심한 病變을 나타냈으며, 한국산 aflatoxin에서도 결정 aflatoxin을 투여한 경우보다 사료에 배합하여 급식시킨 경우가 약간 더 심한 病變을 나타냈다.

4. 닭병아리의 간장에서 aflatoxin이 유발시킨 병리조직학적 소견보다 오리병아리의 간장에서의 경우가 그 정도에 있어 더욱 심하고 뚜렷하였다.