THE EFFECT OF A MOLD INHIBITOR ON THE PERFORMANCE OF GROWING TURKEYS

by 553

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B.S., Kansas State University, 1969

A MASTER'S THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Department of Dairy and Poultry Science

KANSAS STATE UNIVERSITY

Manhattan, Kansas

1971

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INTRODUCTION

A major problem associated with today's large scale poultry operations is an increased awareness of the incidence of molds. Interest of medical and agricultural researchers in this problem has been stimulated by reports from Britain, cited by the USDA (Anonymous, 1965a), of a large number of turkeys poisoned from feeding on contaminated peanut meal. Analysis of the peanut meal showed that it contained a toxic by-product of a common mold, *Aspergillus flavus*. The mycotoxin, as mold-caused toxins are called, was given the name aflatoxin.

There are several significant reasons for an increasing mold problem in poultry production units. One is the trend toward mechanical harvesting of high moisture grains. High moisture content is the single most important condition contributing to microbial deterioration of grain, according to Olafson as quoted by Simon (1970). The second is an increase in multiple flock brooding and rearing facilities with short intervals for clean-up between flocks. Under such conditions if proper management and sanitation practices are not undertaken, a buildup of fungal organisms can occur followed by invasion of secondary infections. A third and vital reason that may contribute to increasing contamination by molds is an increase in the use of antibiotics in poultry flocks. According to Pomeroy as quoted in Squibbs' Technical Bulletin on Mycosis (Anonymous,____b), prolonged use of antibiotics alters the bacterial flora of the intestinal tract allowing the yeast-like organisms to take over and become established in the lining of the intestinal tract. Since mold organism are not susceptible to the commonly used antibiotics and other drugs, they thrive under these conditions.

A method that has been explored for control of mold infections in poultry is to inhibit the growth of mold in feed by the addition of chemical additives. The purpose of this experiment was to evaluate the effect of a mold inhibiting chemical[1/] on the performance of growing turkeys and mold growth in feed.

1/ Mold Curb (R) — Registered trademark for a product supplied by Kemin Industries, Inc., Des Moines, Iowa.

REVIEW OF LITERATURE

Mold Damage to Grain

According to conservative estimates by Johnson as reported by Semenuik (1954), the activities of storage fungi result in the loss of between one and two percent of the world's grain production. The threat of this loss causes even greater cost in added expenditures for drying and storing and in penalties inflicted by untimely marketing and lower commercial grading.

More than 50 species of fungi and a considerable number of species of bacteria have been isolated from agricultural seeds. Bacteria do not normally appear to be agents in the deterioration of stored seeds because they require free water to grow.

Fungi in seeds may be rather arbitrarily divided into two groups, field fungi and storage fungi. The field fungi are those that invade the developing seed while it is still on the plant. The storage fungi, principally *Aspergillus* and *Penicillium*, are those which develop on and within seeds at moisture contents often encountered in storage.

Christensen as quoted by Wogan (1966) reported that in terms of their distribution, there is virtually no overlap between field and storage fungi. In his examinations of several thousand samples of wheat, corn, and barley from the United States, Canada, Mexico, England, and Germany, storage fungi were found in only 0.1 to 0.2 percent of the samples as they were received from the field. Instead, these storage fungi invaded the seeds only after the moisture content was reduced to a level of 14 to 18 percent.

A. glacus is one of the major fungi that invade stored seeds (Christensen, 1957). This group is divided into nine series, each of which contains a number of subspecies. Four of these series, *A. amstelodami*, *A. ruber*,

A. repens, *A. restrictus*, have been found associated with many cases of deterioration in stored wheat and appear to constitute the major organisms that invade wheat whose moisture content is 13.2 to 15 percent. With increasing moisture levels above 15 percent, *A. candidus*, *A. flavus*, *A. tamarii*, and perhaps a few other species of the *Aspergillus* and *Penicillium* appear. Essentially the same moisture limits prevail for the growth of the fungi in corn as in wheat.

The kinds and abundance of fungi in grain and grain products varies according to moisture content of the stored material. Thom and LeFevre (1921) observed marked multiplication of molds and bacteria in samples of cornmeal with approximately 20 percent moisture, of which the predominant organisms were molds. Species of yeast *Mucor* and *Penicillium* were present while *A. flavus* was the predominant fungus. In cornmeal with approximately 13 percent moisture, lower numbers of active species of molds were found. *A. repens* was the predominant fungus in cornmeal of 13 to 15 percent moisture. *A. flavus* began activity in 16 percent moisture cornmeal. Above this moisture level other molds such as *Actinomyces* sp., *Penicillium* sp., *Fusarium* sp., and *A. candidus*, became active.

The actual contamination and resulting condemnation of the grain and the threat to animal health is not due to the presence of the fungal organism itself but to the poisonous toxin that the mold is capable of producing. Austwick and Ayerest (1963) reported that the most important factor in alfatoxin production by *A. flavus* is the moisture of or relative humidity surrounding a natural substrate. A relative humidity of 85 ± 1 percent at 30° C. for 21 days was the optimum condition for aflatoxin production in peanuts according to Diener and Davis (1967). Calderwood

and Schroeder (1968) found that rough rice with a moisture content of 24-26 percent developed relatively large amounts of aflatoxin in 7-21 days during warm weather storage. The optimum time and temperature for aflatoxin production reported by Diener and Davis as quoted by Goldblatt (1969) were 25-30^{o} C. for 5-14 days.

Mold Damage to Livestock

In 1934, according to Sippel as quoted by Christensen and Kaufmann (1969), veterinarians in Illinois estimated that in the central part of that state alone, in the winter of 1933-34, 5,000 horses died of "moldy corn disease."

Sippel *et al.* (1953), reported the poisoning of 1,000 swine in southwestern United States in 1952, with a mortality rate of 22 percent. The poisoning was attributed to consumption of moldy corn. Burnside *et al.* (1957), published the results of a follow-up investigation of the preceding report. They isolated 13 cultures from the moldy grain suspected of being toxic, of which 12 were A. *flavus* and one P. *rubrum*. All the fungi were grown separately in autoclaved, moist corn and the corn was fed to swine. The swine fed corn invaded by an isolate of A. *flavus* and those fed corn invaded by P. *rubrum* died within a few days with external and internal symptoms similar to those found in the diseased animals reported by Sippel. They found the corn invaded by P. *rubrum* was so toxic that a mixture of only 20 percent of the moldy grain and 80 percent sound corn resulted in death of two 55-to 60-lb. pigs to which it was fed, in less than 32 hours after each pig had consumed less than eight ounces of the moldy feed. The investigators soaked some of the P. *rubrum* invaded corn in water, then administered

the supernatant water to horses. Two gallons of the water extract from 4 kilograms (about 9 lb.) of the moldy corn resulted in the death, within 34 hours, of a 790-lb. horse. An order of susceptibility of mammals to mold-caused toxins has been derived from feeding groundnut meal containing a known aflatoxin content. The order reported by Allcroft as quoted by Goldblatt (1969) is: pigs 3-12 weeks old; pregnant sows; calves; fattening pigs and mature cattle; sheep.

It has been reported by several researchers that poultry are the most susceptible of domestic animals to mycotoxins and related mycotic diseases. The order of susceptibility in poultry is as follows: ducklings; turkey poults and pheasant chicks; chickens and Coturnix quail. It has been reported by Brown and Abrams as quoted by Goldblatt (1969) that the New Hampshire breed of chickens is more susceptible than other breeds such as Cornish Game, White Rock, White Leghorn and Rhode Island Red.

In England in 1960, according to Asplin as quoted by Chute *et al.* (1964), outbreaks of an unknown disease caused high mortality in turkey poults and ducklings. The disease, known as Turkey X disease, was due to a toxigenic strain of *A. flavus* in Brazilian peanut meal. Archibald *et al.* (1962), reported a syndrome occurred in Canada, involving 400,000 birds over a period of several months, that was very similar in pathology to the mycotoxicosis in England. Asplin and Carnaghan (1961) reported the effect of toxic groundnut meal on chickens is of interest because the mortality produced is low compared with that in turkey poults and ducks.

In earlier work done with chickens by Forgacs and Carll (1955), fungi isolated from feed taken from broiler houses where poultry hemorrhagic syndrome was enzootic, induced acute toxic symptoms in chicks. In a later study,

Forgacs *et al.* (1958), reproduced essentially similar toxic conditions by feeding chicks diets containing feedstuffs on which two selected toxic fungi *P. purpurogenum* and *P. rubrum* of varying toxicity had proliferated. Schiemaier *et al.* (1961), reported that two species of *Aspergilli* were found to produce lesions in chicks indistinguishable from typical hemorrhagic syndrome when fed orally. The poultry hemorrhagic syndrome, as observed in the field, usually reaches its highest intensity at four to seven weeks and is absent at the time broilers are marketed. Therefore, the economic significance of the syndrome in such cases has been questioned frequently. However, it must be remembered that the chicks are subject to stress during the course of the toxicosis and are vulnerable to opportune infections.

Richardson *et al.* (1962a) found that poults fed moldy soybean meal had depressed growth. The meal had been adjusted to 19 percent moisture and allowed to mold for six weeks. Significant decreases in the nutritive value of the meal were also observed. In a subsequent study, Richardson *et al.* (1962b) observed growth depression in chicks fed a broiler mash that had been allowed to mold for two to ten weeks.

According to Cottier *et al.* (1969), feeding hens a 1,834 p.p.b. level of aflatoxin resulted in zero hatchability because of embryo mortality during the first six days of incubation; when hens were changed to a zero level of aflatoxin hatchability was normal after six days on the feed.

Hamilton and Harris (1970) found that aflatoxicosis was interacting with *Candida albicans*, a yeast-like fungi associated with mycotic infections involving the crop of growing turkeys. He suggested that many field problems associated with aflatoxin are not true aflatoxicosis but a syndrome caused by interacting stresses. Also, it was suggested in earlier work by Tripathy *et al.*

(1965), that *C. albicans* plays a role in production of some cases of aortic rupture, possibly through interference with nutrition. Aortic rupture is a major economic concern of turkey producers. Hamilton and Garlich (1970), in other work, suggested dietary aflatoxin produces a generalized loss of strength and integrity in tissues of the chicken and this loss results in an increased susceptibility to bruising. Bruising and the resulting condemnations cause an annual loss of several million dollars to the poultry industry.

Choudhury *et al.* (1970) concluded ochratoxin, produced by the mold *A. ochraceus*, was very toxic to adult laying hens. High levels in the diet were highly fatal and the surviving birds produced poor quality eggs unsuitable for human consumption.

Control of the Problem

Various chemical compounds have been tested for their fungicidial properties. Wolford (1945) used sodium propionate to reduce the growth of microorganisms in fruits and vegetables. Milner (1946) found that sulfanilamide showed the greatest fungistatic activity of 11 sulfa drugs tested. In subsequent trials Milner *et al.* (1947) tested eight compounds as mold inhibitors. Relatively few of the compounds tested prevented visible development of molds on moist seed. Some compounds known to be toxic to certain fungi under certain conditions, such as calcium propionate, did not prevent the visible development of molds on wheat seed treated with them. In some cases a given fungicide inhibited certain molds, but not others, or inhibited exterior development and spore production of one or more molds within the seed. Of the eight compounds tested as moldicides

on wheat with a moisture content of 20 percent, 8-hydroxyquinoline sulfate was most effective followed by, in descending order: thiourea, p-amino-benzoic acid, sulfanilamide, benzene sulfonamide, 2-aminothiazole, chloramine-B, and calcium propionate.

Lenhart and Cosens (1949) used propionic acid to inhibit the growth of molds in sugar solutions. Propionic acid and propionic anhydride at a level of 0.1 percent was reported by Richardson and Halick (1957) to inhibit the growth of molds and heating in corn meal containing approximately 15 percent moisture. The same meal without an inhibitor started to heat in seven days and reached a maximum temperature of 120° F. in ten days. Calcium propionate inhibited the growth of molds and heating at a level of 0.3 percent, but was not effective at a level of 0.1 percent. Sodium propionate did not inhibit the growth of molds and heating at a level of 0.6 percent. Further tests were carried out to determine whether calcium propionate and sorbic acid prevented heating in a variety of feed ingredients. Their data revealed calcium propionate and sorbic acid (hexadienoic acid) prevented heating in every ingredient when its moisture content was only slightly above the critical level. When the moisture content of an ingredient was one to three percent above the critical level, the inhibitors were much less effective.

Schroeder (1964) treated samples of Century Patna rough rice with sodium propionate (5,000 ppm). Samples were air-dried at room temperature and dried with infrared radiation to moisture contents of 12.7 to 13 percent and stored in relative humidities of approximately 75 and 85 percent at room temperature for one to six months. Infrared drying and the sodium propionate treatment reduced field fungi infection prior to storing the seed. The rate

of infection by storage fungi was reduced significantly for six months by treatment with sodium propionate in the samples at 75 percent relative humidity and for four months in the samples stored at 85 percent relative humidity. Only samples stored at 85 percent relative humidity and not treated with sodium propionate showed a significant increase in discolored kernels after six months storage.

Simon (1970) reported that acetic acid and propionic acid were the most effective mold inhibitors under storage conditions. Grain sorghum harvested at 26.5 and 29 percent moisture contents was treated with various levels of acetic acid and propionic acid and stored at 80-82° F. No mold was detected in the grain treated with 0.7 to 0.9 percent propionic acid and stored at 29 percent moisture content for 135 days. Grain containing 26.5 percent moisture treated with 0.5 percent propionic acid remained mold free during 127 days of storage; whereas, mold growth appeared within 40-42 days on grain treated with 0.8 percent acetic acid.

Data compiled by Deyoe and Quadri (1970) indicated that Mold Curb[1/] added to mixed feed at one and two pounds per ton resulted in a significant reduction in mold counts. Samples were analyzed by plating techniques using potato dextrose agar. The effect of pelleting and conditioning also resulted in a marked reduction in mold incidence in the tested samples. The greatest effect occurred when Mold Curb was added at two pounds per ton and when conditioning temperature for pelleting was 70° C. A separate study to evaluate comparative effects of Mold Curb, sodium propionate and calcium propionate did not indicate statistically significant reductions

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[1/] Registered trademark for a product supplied by Kemin Industries, Inc., Des Moines, Iowa.

in mold incidence, although the data showed a trend towards lower mold counts when these materials were added to a mash feed.

A few reports appear in the literature concerning the effect of mold inhibitors on animal performance. Coles *et al.* (1970) compared the performance of individually fed pigs from weaning to 200 lb. weight when fed 0.8 percent propionic acid treated moist barley versus untreated moist and dry barley. They found no significant differences in the performance of pigs fed propionic acid treated ground barley versus untreated ground barley. There were no significant differences between the treatments in carcass measurements or in apparent digestibility coefficients as measured by the chromic-oxide method. According to Mickie, Anonymous (1968c), the addition of propionic acid to moist barley fed to turkeys produced no adverse effects on performance.

EXPERIMENTAL METHODS

Three hundred and twenty day-old Nicholas Broad Breasted White sexed male poults, hatched on February 9, 1970, were wingbanded and distributed randomly into eight pens. Each pen (6.1 by 6.1-meter) contained 40 turkeys. The pens were situated on each side of an aisle which extended north and south the length of a naturally ventilated brooding and rearing house. Each of the four treatments was allotted two replicate pens for the experiment.

The pens were equipped with gas hovers, gallon water fountains and metal trough feeders. At the end of the second week the poults were switched gradually to automatic plastic cup waterers. At three weeks of age the birds were switched to tube-type self-feeders. All pens had concrete floors which were covered with 7.7 to 10.3 cm. of wood shavings. Wet litter was removed and fresh litter added as necessary. The turkeys were debeaked at four weeks of age to prevent cannibalism. Artificial and natural lighting were used to supply 14 hours of light per day during the course of the experiment.

The turkeys were weighed at the end of each four week period through the duration of the 24-week experiment. Records of mortality, feed consumption and body weight were maintained. Feed consumption data were adjusted for mortality. Necessary management practices were performed and management conditions were maintained as nearly alike as possible for all pens. At nine weeks of age infectious sinusitis was diagnosed in the turkeys in some pens. At ten weeks all birds were fed Terramycin[1/] in the diet for one week

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1/Registered trademark for a (oxytetracycline) product supplied by Chas. Pfizer, Inc., New York, N.Y.

at a level of 200 gm. per ton and injected with Tylan.[1/] The latter treatment was repeated at 20 weeks.

All poults were fed from day-old to 24 weeks the K.S.U. standard turkey diets (Appendix, Tables 1, 2, 3). Levels of 0, 454, 908, and 1,816 gm. per ton of the chemical were included in the diets. According to the manufacturer the chemical composition of the product is as follows: Active ingredients—propionic acid 80%; inert ingredients—sodium chloride, propyl-p-hydroxybenzoate, lactic acid, ethyl methylphenylglycidate, calcium silicate, ethyl orthoamino-benzoate, ascorbic palmitate. The poults were fed the starter from day-old to eight weeks, the grower from 8 to 16 weeks, and the finisher from 16 to 24 weeks.

All diets were mixed by a local commercial feed mill. The chemical was added to the diets at the K.S.U. Poultry Research Center feed mixing facility. A 9.1 kg. batch of the diet plus the required level of the chemical was mixed first in a Hobart mixer for 5 min. and then added to 36.3 kg. of diet and mixed for an additional 5 min. in a horizontal mixer. The resulting 45.4 kg. premix was then added to 317.8 kg. of diet and mixed a final time in a 454-kg. Davis horizontal mixer. This procedure was repeated for each treatment level.

The procedure outlined by Sauer and Christensen (1966) was used to determine the effectiveness of the chemical in inhibiting mold in the diets. Five gm. of each diet was placed in 500 ml. of 0.12 percent sterile agar solution and 5 ml. of the resulting suspension were removed and placed in 45 ml. of the same suspension medium. This was shaken briskly 100 times.

[1/] Tylan - registered trademark of Tylosin, Elanco Products Company, Indianapolis, Indiana.

Further dilutions were made in the same manner and 1 ml. of one or more final dilutions was put in each of two sterile petri dishes. Melted potato dextrose agar (PDA) was treated with 1 ml. of sterile 10 percent tartaric acid per liter to lower the pH to approximately 3.2 to inhibit bacterial growth. The agar was cooled to about 52° C. and poured into 100 x 15 mm. plastic petri dishes. The dishes were swirled to distribute the suspended material uniformly and the agar was allowed to harden. The dishes then were incubated at room temperature until the colonies could be counted. Counting was done using a Spencer colony counter. An unequal sub-class analysis of variance (Snedecor, 1956) was used to analyze the weight gain, feed conversion and mortality data.

RESULTS AND DISCUSSION

The average weight gain of the birds for each treatment is shown in Table 1. Analysis of variance (Table 2) shows no significant treatment difference for weight gain over the entire period. It does show, however, a significant ($P < .05$) pen effect as shown in Table 2. Although the data indicate some depression of weight gain at the four pound level, the lack of significance may be due to the large pen effect (error) mean square.

Table 1. Effect of the chemical on average weight gain[1/]

Level of chemical	Age (wk.)					
	4	8	12	16	20	24
(kg./ton)	(kg.)					
0.00	0.71	2.91	5.66	8.62	10.71	12.71
0.45	0.71	2.91	5.72	8.29	10.71	12.86
0.91	0.71	2.95	5.72	8.39	10.71	12.90
1.82	0.69	2.90	5.55	8.15	10.44	12.55

[1/] Each value represents the average of two replicates.

These results are in agreement with data reported earlier, Anonymous (1968c). More than 800 tons of propionic acid treated grain was fed to all classes of livestock in those trials. Data from those reports indicated no significant differences in the performance of livestock fed six times the level of propionic acid used in this experiment. Also no incidence of feed refusal was reported.

Table 2. Analysis of variance of average weight gain of turkeys fed the mold inhibiting chemical

Source of variation	d.f.	F-value	Mean Squares		
			Wt. gain	Feed conv.	Mort.
a. Treatment	4	a/b	83,028.50	0.04	74.13
b. Pens	5	b/e	52,189.47*	0.03**	77.06**
c. Periods	5	c/e	210,115,839.99**	9.62**	142.89**
d. Trt x pens	20	d/e	11,255.39	0.01	10.95
e. Error	25		14,189.27	0.01	5.78
Total	59				

* Significant at ($P < .05$)

** Significant at ($P < .01$)

Feed conversion data in Table 3 show a similar trend as weight gain. There was no significance between treatments but there were significant ($P < .01$) pen differences. An LSD analysis was performed on the means in an attempt to determine where the differences were. The data (Table 4) show no consistency in pen differences, therefore offering no explanation for the significant pen effect. Lack of an effect of the chemical on feed utilization is in agreement with data presented by Anonymous (1968c).

Table 3. Effect of the chemical on feed conversion[1/]

Level of chemical	Age (wk.)					
	4	8	12	16	20	24
(gm./ton)	(gm. feed/gm. wt.)					
0	1.63	2.06	2.70	3.17	3.63	4.11
454	1.69	1.95	2.54	3.14	3.64	4.08
908	1.65	2.11	2.72	3.31	3.86	4.25
1,816	1.67	2.09	2.73	3.23	3.82	4.21

[1/]Each value represents the average of two replicates.

Table 4. LSD analysis of feed conversion and weight gain means

Level of chemical	Replicate	Feed conv. means	Wt. gain means
(gm./ton)			
0	1	2.97[b 1/]	6,910[c]
	2	2.80[a]	6,709[a]
454	1	2.83[a]	6,830[b]
	2	2.86[a]	6,901[c]
908	1	3.01[b]	6,989[d]
	2	2.96[b]	6,802[b]
1,816	1	2.99[b]	6,730[a]
	2	2.97[b]	6,700[a]

1/ Values with different superscripts differ significantly at the ($P < .05$) level.

Although mortality was significant ($P < .01$) for treatments and pens, no consistent relationship between mortality and level of the chemical was observed (Table 5).

Table 5. Effect of chemical on mortality[1/]

Level of chemical	Age (wk.)					
	4	8	12	16	20	24
(gm./ton)			(%)			
0	3.5	4.6	6.9	9.2	10.4	11.5
454	1.3	2.5	5.0	7.5	7.5	11.1
908	1.2	3.9	13.1	13.1	16.9	19.8
1,816	3.9	6.4	9.0	10.1	10.1	15.1

[1/] Each value represents the average of two replicates.

The effectiveness of the chemical on mold growth in the diets was evaluated. The data appear in Table 6. The mold counts in the feed samples were low at all levels including the control. The data show a trend toward declining mold counts in the diets as the level of chemical was increased. These data are in agreement with the findings of Deyoe and Quadri (1970) who found the product (Mold Curb) added to mixed feed at 454 and 908 gm. per ton resulted in a significant ($P < .01$) reduction in mold counts.

Table 6. Effect of the chemical on mold growth in diet and litter samples

Level of chemical	Diet	Litter
(gm./ton)	(No. of organisms/gm.)[1/]	
0	32,000	78,000
454	12,000	48,000
908	4,000	82,000
1,816	2,000	10,000

[1/] Each value represents the average of 5 replicates.

Litter samples (Table 6) show a similar trend of reduced mold counts as the level of chemical was increased except for the 908 gm. level. The higher count at the 908 gm. level could be due to an error in sampling procedure. Another explanation may be that the litter was not kept as dry as litter in other pens due to a water main break just a few weeks before the samples were taken.

SUMMARY

Three hundred twenty large type market turkeys were fed the standard K.S.U. turkey diets containing a mold inhibitor (Mold Curb) at 0, 454, 908, or 1,816 gm. per ton from 0 through 24 weeks of age. The turkeys were weighed each four-week period to determine their performance at each treatment level. Mold counts were made on samples of the diets and litter to determine the effect of the chemical on mold growth. Under the conditions of this investigation the results show:

1. There was no significant difference in average weight gain or feed consumption over the 24-week period of the turkeys fed the various levels of the additive.
2. There was a significant ($P < .01$) pen difference for feed conversion and weight gain over the 24-week period but an LSD analysis failed to show any consistent relationships.
3. There was a significant difference ($P < .01$) in mortality between pens, but no significant relationships to the treatments were observed.
4. There was a trend toward lower mold counts in the diet and litter samples as the level of the chemical was increased.

Under the conditions of this study, the chemical neither enhanced nor depressed performance of growing turkeys.

ACKNOWLEDGMENT

The author wishes to express his appreciation to Dr. A. W. Adams of the Department of Dairy and Poultry Science for his suggestions and constructive criticism in developing this experiment and in preparing this thesis. Gratitude is expressed to the members of my committee: Drs. C. W. Deyoe of the Department of Grain Science and Industry, and P. E. Sanford and C. L. Norton of the Department of Dairy and Poultry Science. Appreciation is extended to Kemin Industries, Inc., Des Moines, Iowa, for partial support of this experiment. Also thanks is extended to A. J. Kahrs, Poultry Farm Superintendent, for his assistance and to my fiancee, Janalee, for her typing of this thesis.

REFERENCES

Anonymous, 1965a. Preventing mold-caused toxins in farm commodities. U.S.D.A. ARS 22-92, pp. 1-2.

Anonymous, ——b. Mycosis - Technical bulletin on crop mycosis and mycotic diarrhea. E. R. Squibb & Sons, Inc., East Brunswick, N.J., pp. 1-2.

Anonymous, 1968c. Tech. (Research) Bull., B. P. Chemicals (U.K.) Ltd., Wallington Surrey, England.

Archibald, R. McG., H. T. Smith, and J. D. Smith, 1962. Brazilian groundnut toxicosis in Canadian broiler chickens. Canadian Vet. J. 3:322-325.

Asplin, F. D., and R. B. A. Carnaghan, 1961. The toxicity of certain groundnut meals for poultry with special reference to their effect on ducklings and chickens. Vet. Record 73:1215-1218.

Austwick, P. K. C., and G. Ayerst, 1963. Toxic products in groundnuts. Groundnut microflora and toxicity. Chem. Industry 2:55-61.

Burnside, J. E., W. L. Sippel, J. Forgacs, W. T. Carll, M. B. Atwood, and A. R. Doll, 1957. A disease of swine and cattle caused by eating moldy corn. II: Experimental production with pure cultures of molds. Am. J. Vet. Research 18:817-824.

Calderwood, D. L., and H. W. Schroeder, 1968. Aflatoxin development and grade of undried rough rice following prolonged storage in aerated bins. U. S. Dept. Agr., Agr. Res. Serv. Rep. 52-25, 32p.

Choudhury, H., C. W. Carlson, and G. Semeniuk, 1970. Toxic effects of ochratoxin on laying hens. Abstract of a paper presented at the 59th Annual Poultry Sci. Assoc., University of Tennessee, Knoxville, Tennessee.

Christensen, C. M., 1957. Deterioration of stored grain by fungi. Botanical Rev. 23:108-134.

Christensen, C. M., and H. H. Kaufmann, 1969. *Grain Storage*. The University of Minnesota Press, Minneapolis, Minnesota.

Chute, H. L., S. L. Hollander, E. S. Barden, and D. C. O'Meara, 1964. The pathology of mycotoxicosis of certain fungi in chickens. Avian Dis. 9:57-66.

Coles, D. J. A., G. W. Dean, and J. R. Luscombe, 1970. Single cereal diets for bacon pigs. II: The effect of methods of storage and preparation of barley on performance and carcass quality. Animal Prod. Rep. 12:1-6.

Cottier, C. R., C. H. Moore, U. L. Diener, and N. D. Davis, 1969. The effect of feeding four levels of aflatoxin on hatchability and subsequent performance of broilers. Poultry Sci. 48:1797.

Deyoe, C. W., and S. F. Quadri, 1970. Effects of mold inhibitors in various types of feeds. Feedstuffs 42, 31:58-59.

Diener, U. L., and N. D. Davis, 1967. Limiting temperature and relative humidity for growth and production of aflatoxin and free fatty acids by *Aspergillus flavus* in sterile peanuts. J. Am. Oil Chemists' Soc. 44:22-28.

Forgacs, J., and W. T. Carll, 1955. Preliminary mycotoxic studies on hemorrhagic disease in poultry. Vet. Med. 50:172.

Forgacs, J., H. Koch, W. T. Carll, and R. H. White-Stevens, 1958. Additional studies on the relationship of mycotoxicosis to the poultry hemorrhagic syndrome. Am. J. Vet. Research 19:744-753.

Goldblatt, L. A., 1969. *Aflatoxin*. Academic Press, Inc., New York, N.Y., London, England.

Hamilton, P. B., and J. D. Garlich, 1970. Aflatoxin as a cause of fatty liver syndrome in laying hens. Abstract of a paper presented at the 59th Annual Poultry Sci. Assoc., University of Tennessee, Knoxville, Tennessee.

Hamilton, P. B., and J. R. Harris, 1970. Interaction of aflatoxicosis with *Candida albicans* infections and other stresses. Abstract of a paper presented at the 59th Annual Poultry Sci. Assoc., University of Tennessee, Knoxville, Tennessee.

Lenhart, J., and K. W. Cosens, 1949. Molds and bacteria curbed in immersion freezing. Food Ind. 21:442.

Milner, M., 1946. Utility of sulfa drugs for the inhibition of mold respiration in grain. Science 104:463-464.

Milner, M., C. M. Christensen, and W. F. Geddes, 1947. Grain storage studies. VII. Influence of certain mold inhibitors on respiration of moist wheat. Cereal Chem. 24:507-517.

Richardson, L. R., and J. V. Halick, 1957. Studies on feed spoilage. (Texas) Agr. Exp. Sta. (Research) Bull. 879.

Richardson, L. R., S. Wilkes, J. Godwin, and K. R. Pierce, 1962a. Effect of moldy diet and moldy soybean on the growth of chicks and poults. J. Nutrition 78:301-306.

Richardson, L. R., and B. D. Webb, 1962b. Effect of moldy diet on the growth and mortality of chicks and poults. Texas Feed Service Report No. 23, Texas A & M College, pp. 3-4.

Sauer, D. B., and C. M. Christensen, 1966. Comparison of hard red spring and winter wheats in storage. Phytopathology 56, 6:619-623.

Schiemaier, G., Panda Bhagabat, H. M. DeVolt, N. C. Laffer, and R. D. Creek, 1961. Hemorrhagic lesions in chickens resembling naturally occurring "hemorrhagic syndrome" produced experimentally by mycotoxins. Poultry Sci. 40:1132-1133.

Schroeder, H. W., 1964. Sodium propionate and infrared drying for control of fungi infecting rough rice. Phytopathology 54, 7:858-862.

Semeniuk, G., 1954. Microflora. *Storage of Cereal Grains and their Products.* American Association of Cereal Chemists, St. Paul, Minnesota.

Simon, J. A., 1970. Chemical control of microorganisms in stored grains. PhD Dissertation. Kansas State University, Manhattan, Kansas.

Sippel, W. L., J. E. Burnside, and B. F. Atwood, 1953. A disease of swine and cattle caused by eating moldy corn. Proc. of the Vet. Assoc., pp. 174-181.

Snedecor, G. W., 1956. *Statistical Methods,* 5th Edition. Iowa State College Press, Ames, Iowa.

Thom, C., and E. LeFevre, 1921. Flora of corn meal. J. Agr. Research 22:179-188.

Tripathy, S. B., W. J. Mathey, and S. G. Kenzy, 1965. Study of aortic changes associated with candidiasis of turkeys. Avian Dis. 9:520-530.

Wogan, G. N., 1966. *Mycotoxins in Feedstuffs.* The M.I.T. Press, Cambridge, Massachusetts, pp. 14.

Wolford, E. R., 1945. The effect of sodium propionate on microorganisms. J. Bact. 50:235.

APPENDIX

Table A-1. K.S.U. 26% protein complete turkey starter

Ingredients	Percentage of ration
Corn, yellow, ground	20.0
Sorghum grain, ground	19.0
Middlings, wheat standard	10.0
Alfalfa meal, 17% prot., dehy.	5.0
Soybean meal, 44% prot. solv. extr.	30.0
Fish meal	2.5
Fish solubles	2.5
Meat and bone scraps, 50% prot.	5.0
Fermentation solubles (DDS)	2.5
Calcium carbonate (Ground limestone)	2.0
Dicalcium phosphate	1.0
Salt (Sodium chloride)	0.5
	Added per 1,000 gm.
	mg.
Trace mineral mix[1/]	506
Methionine	1,012
Coccidiostat[2/]	506
nf-180(R)	1,012
Antibiotics supplement[3/]	3,520
Vitamin A (10,000 U.S.P. units/gm.)	550
Vitamin D_3 (15,000 I.C.U./gm.)	110
Vitamin E (Myvamix (R) 20,000 I.U./lb.)	220
Vitamin K (Sodium Bisulfite (R))	4
Vitamin B_{12} (20 mg./lb.)	506
B-complex vitamin mix[4/]	1,012
Blackheadstat[5/]	506

[1/] Trace mineral premix supplying by %: Mn 10; Fe 10; Cu 1; Zn 5; I_2 0.3; Co 0.1.

[2/] Amprol - 25% or equivalent coccidiostat.

[3/] Combination of 2 antibiotics: Aureomycin 50 gm./T and Zinc Bacitracin 50 gm./T (45 gm. Aurofac-25 and 115 gm. Baciferm-10 (R)).

[4/] B-complex vitamin mix supplying in mg./lb.: riboflavin 2,000; pantothenic acid 2,680; niacin 6,000; choline chloride 20,000.

[5/] Histostat - 50% or equivalent blackheadstat.

(R) Registered trademark.

Table A-2. K.S.U. 20% protein complete turkey grower

Ingredients	Percentage of ration
Corn, yellow, ground	25.0
Sorghum grain, ground (milo)	30.0
Oats, ground	5.0
Soybean meal, 44% prot. solv. extr.	25.0
Fish meal, 60% prot.	2.0
Meat and bone meal, 50% prot.	2.0
Alfalfa meal, 17% prot., dehy.	5.0
Fermentation solubles (DDS)	1.0
Calcium carbonate (Ground limestone)	2.5
Dicalcium phosphate	2.0
Salt (Sodium chloride)	0.5
	Added per 1,000 gm.
	mg.
Trace mineral premix[1/]	1,012
Vitamin A (10,000 units/gm.)	440
Vitamin D_3 (15,000 I.C.U./gm.)	110
B-complex vitamin mix[2/]	1,012
Vitamin B_{12} (20 mg./lb.)	506
Methoinine	506
Antibiotic supplement[3/]	352
Coccidiostat[4/]	506
Blackheadstat[5/]	506
nf-180 (R)	1,012

[1/] Trace mineral premix supplying by %: Mn 10; Fe 10; Ca, max. 14, min. 12; Cu 1; Zn 5; 0.3; Co 0.1.

[2/] B-complex vitamin mix supplying in mg./lb.: riboflavin 2,000; pantothenic acid 2,680; niacin 6,000; choline chloride 20,000.

[3/] Combination of 2 antibiotics: Aureomycin 5 gm./T and Zinc Bacitracin 5 gm./T (4.5 gm. Aurofac-25 (R) and 11.5 gm. Baciferm-10 (R)).

[4/] Amprol-25%(R) at rate of 1 lb./T.

[5/] Histostat-50(R) at rate of 1 lb./T.

(R) Registered trademark.

Table A-3. K.S.U. 16% protein complete turkey finisher

Ingredients	Percentage of ration
Corn, yellow, ground	30.0
Sorghum grain, ground (milo)	40.0
Alfalfa meal, 17% prot., dehy.	4.0
Soybean meal, 44% prot. solv. extr.	15.0
Meat and bone scraps	5.0
Fermentation solubles (DDS)	1.0
Calcium carbonate (Ground limestone)	2.5
Dicalcium phosphate	2.0
Salt (Sodium chloride)	0.5
	Added per 1,000 gm.
	mg.
Trace mineral premix[1/]	506
Vitamin A (10,000 units/gm.)	330
Vitamin D_3 (15,000 I.C.U./gm.)	110
B-complex vitamin mix[2/]	506
Vitamin B_{12} (20 mg./lb.)	506
Methionine	506
Antibiotic supplement[3/]	352
nf-180(R) (Furazolidone)	506

[1/] Trace mineral premix supplying by %: Mn 10; Fe 10; Ca, max. 14; min. 12; Cu 1; Zn 5; I_2 0.3; Co 0.1.

[2/] B-complex vitamin mix supplying in mg./lb.: riboflavin 2,000; pantothenic acid 2,680; niacin 6,000; choline chloride 20,000.

[3/] Combination of 2 antibiotics: Aureomycin 5 gm./T and Zinc Bacitracin 5 gm./T (4.5 gm. Aurofac-25 (R) and 11.5 gm. Baciferm-10 (R)).

(R) Registered trademark.

THE EFFECT OF A MOLD INHIBITOR ON THE PERFORMANCE OF GROWING TURKEYS

by

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B.S., Kansas State University, 1969

AN ABSTRACT OF A MASTER'S THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Department of Dairy and Poultry Science

KANSAS STATE UNIVERSITY

Manhattan, Kansas

1971

Large-type market turkeys were fed the standard K.S.U. turkey diets containing a mold inhibitor at 0, 454, 908, or 1,816 gm. per ton from 0 through 24 weeks of age. Two pens (40 poults per pen) were fed each level of the mold inhibitor. Records were maintained on rate of gain, feed consumption and mortality. Mold counts were made on samples of the diets and litter to determine the effect of the chemical on mold growth.

Under the conditions of this experiment it was found that the levels of mold inhibitor used had no significant effect on final weight gain, feed conversion or mortality. There were significant pen x weight gain ($P < .05$) and pen x feed conversion ($P < .01$) interactions. An LSD analysis performed on the means failed to show any consistent relationships. A significant ($P < .01$) difference in mortality between pens was observed, but no significant correlations to the treatments were established. Although mold counts were not high in any of the diets, a trend toward lower mold counts was observed as the level of mold inhibitor was increased. A similar trend occurred in the litter samples.

The results of this investigation indicate that the mold inhibitor in the diet neither enhanced nor depressed performance of growing turkeys.