HISTOMORPHOLOGICAL EVALUATION OF THE LIVER OF BROILER CHICKENS SUPPLEMENTED WITH A PREBIOTIC COMPOUND BASED ON MANNANOLIGOSACCHARIDES, FRUCTOOLIGOSACCHARIDES, AND GLUCOOLIGOSACCHARIDES

AVALIAÇÃO HISTOMORFOLÓGICA DO FÍGADO DE FRANGOS DE CORTE SUPLEMENTADOS COM COMPOSTO PREBIÓTICO À BASE DE MANANOLIGOSSACARÍDEOS, FRUTOLIGOSSACARÍDEOS E GLUCOLIGOSSACARÍDEOS

EVALUACIÓN HISTOMORFOLÓGICA DEL HÍGADO DE POLLOS DE ENGORDE SUPLEMENTADOS CON UN COMPUESTO PREBIÓTICO A BASE DE MANANOLIGOSACÁRIDOS, FRUCTOOLIGOSACÁRIDOS Y GLUCOOLIGOSACÁRIDOS

https://doi.org/10.56238/sevened2026.012-022

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ABSTRACT

This study aimed to microscopically evaluate the liver of broiler chickens supplemented with a prebiotic compound containing mannan-oligosaccharides, fructo-oligosaccharides, and gluco-oligosaccharides. A total of 240 female birds were distributed into three experimental treatments with three replicates of four birds each, from 1 to 42 days of age. The treatments were: T1 – 500 g of prebiotic compound per ton of feed, T2 – 16.5 ppm of Flavomycin per ton of feed, and T3 – control (no treatment). Liver samples were collected on days 7, 14, 28, and 42 post-treatment and processed according to histological routine. Histopathological alterations were analyzed using frequency distribution and the Kruskal-Wallis test ($p<0.05$) in GraphPad Prism 7.0. The results showed a lower frequency of lesions in T1 at 7, 14, and 28 days, but by day 42, the lesions were similar among groups. These findings suggest that

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the prebiotic compound effectively contributed to liver protection against aflatoxin-induced damage, reducing hepatic lesions.

Keywords: Aflatoxin. Histopathology. Liver. Poultry.

RESUMO

Objetivou-se avaliar microscopicamente o fígado de frangos de corte suplementados com composto prebiótico a base de mananoligossacarídeos, frutoligossacarídeos e glucoligossacarídeos. Foram utilizadas 240 aves fêmeas distribuídas em três tratamentos experimentais com três repetições de quatro aves cada, no período de um a 42 dias de idade. Os tratamentos experimentais foram: Tratamento 1 (T1) 500 g de composto prebiótico por tonelada de ração, Tratamento 2 (T2) 16,5 ppm de Flavomicina por tonelada de ração, Tratamento 3 (T3) As aves receberam a ração sem nenhum tratamento. No Tratamento 1 (T1) o composto prebiótico utilizado como aditivo alimentar e tem como base mananoligossacarídeos (MOS), os glucoligossacarídeos (GOS) e os frutoligossacarídeos (FOS). As amostras de fígado foram coletadas nos dias D7, 14, 28 e 42 pós-tratamento, posteriormente foram processadas conforme rotina histológica. As alterações histopatológicas foram analisadas por distribuição de frequência, utilizando o teste não paramétrico de Kruskal-Wallis, adotando-se nível de significância de 5% e utilizando GraphPad Prism 7,0. Os resultados apresentaram uma frequência de lesões menor do T1 durante as idades de 7, 14, 28, porém aos 42 dias as lesões eram iguais entre os grupos. Desta forma podemos sugerir que o composto prebiótico avaliado atuou de forma efetiva na defesa contra a aflatoxina, contribuindo com a redução das lesões hepáticas.

Palavras-chave: Aflatoxina. Avicultura. Fígado. Histopatologia.

RESUMEN

Se tuvo como objetivo evaluar microscópicamente el hígado de pollos de engorde suplementados con un compuesto prebiótico a base de manano-oligosacáridos, fructo-oligosacáridos y gluco-oligosacáridos. Se utilizaron 240 aves hembras distribuidas en tres tratamientos experimentales con tres repeticiones de cuatro aves cada una, durante el período de uno a 42 días de edad. Los tratamientos experimentales fueron: Tratamiento 1 (T1): 500 g de compuesto prebiótico por tonelada de alimento; Tratamiento 2 (T2): 16,5 ppm de flavomicina por tonelada de alimento; Tratamiento 3 (T3): las aves recibieron el alimento sin ningún tratamiento. En el Tratamiento 1 (T1), el compuesto prebiótico utilizado como aditivo alimentario tuvo como base manano-oligosacáridos (MOS), gluco-oligosacáridos (GOS) y fructo-oligosacáridos (FOS). Las muestras de hígado fueron recolectadas en los días D7, 14, 28 y 42 postratamiento y posteriormente procesadas conforme a la rutina histológica. Las alteraciones histopatológicas se analizaron mediante distribución de frecuencia, utilizando la prueba no paramétrica de Kruskal-Wallis, adoptándose un nivel de significancia del 5% y utilizando el software GraphPad Prism 7.0. Los resultados mostraron una menor frecuencia de lesiones en el T1 a las edades de 7, 14 y 28 días; sin embargo, a los 42 días las lesiones fueron iguales entre los grupos. De esta forma, se puede sugerir que el compuesto prebiótico evaluado actuó de manera efectiva en la defensa contra la aflatoxina, contribuyendo a la reducción de las lesiones hepáticas.

Palabras clave: Aflatoxina. Avicultura. Hígado. Histopatología.

1 INTRODUCTION

The contamination of ingredients used in broiler feed by mycotoxins, especially aflatoxins, represents an important limiting factor for the productive performance and health of the birds. The ingestion of these toxins compromises growth, immune response and hepatic metabolism, since the liver is the main organ involved in biotransformation, presenting relevant morphological and histopathological alterations, in addition to economic impact because it is an edible viscera widely used by industry (Bünzen & Haese, 2006; Monson et al., 2015).

In this context, the use of prebiotics, such as mannan oligosaccharides (MOS), fructooligosaccharides (FOS) and glucoligosaccharides (GOS), has stood out as a nutritional strategy capable of reducing the effects of mycotoxins, as they favor the intestinal microbiota and act on the adsorption and biodegradation of these toxins (Coelho et al., 2014; Hiththatiyage et al., 2025; Nunes et al., 2009). Thus, the objective of this study was to microscopically evaluate the liver of broilers supplemented with a prebiotic compound based on mannan oligosaccharides, fructooligosaccharides and glucoligosaccharides.

2 THEORETICAL FRAMEWORK

The ingestion of feed contaminated with mycotoxins leads to a reduction in the growth and development of animals, making them more susceptible to diseases, especially in the growth and reproduction phases, in addition to the impairments in performance caused by toxins, a point that should be highlighted is the decrease in nutritional value in grains attacked by fungi, especially in energy levels (Bünzen & Haese, 2006).

Poultry feed is mainly composed of corn (60%) and soybeans (20%) and in these ingredients, mycotoxins have been constantly isolated. According to Monson et al. (2015), the ingestion of contaminated feed provides the biotransformation and transmission of aflatoxins.

Among the edible viscera of poultry, the liver is economically more important, being widely used as a raw material for processed foods. The mycotoxin analysis laboratory (Lamic) determines maximum levels of aflatoxin, 0 ppb in the initial phase up to 14 days, 2 ppb up to the age of 35 days and maximum tolerance of 5 ppb in the final phase from 35 days. It is known that aflotoxin poisoning in poultry can generate hepatomegaly, with pale and friable phpilated, with the main histopathological lesions being focal necrosis of hepatocytes, hemorrhages, inflammation and hyperplasia of bile duct cells (Monson et al., 2015)

Hepatic fat synthesis and its transport to other areas of the body are seriously affected by the action of AFB1 (Merkley et al. 1987). Several types of substances such as carbohydrates, peptides, proteins, lipids, fibers, and alcohols can be classified as prebiotics. According to Silva (2012), the carbohydrates called oligosaccharides, which have short chains of polysaccharides of three to ten simple sugars, are the ones with the best prebiotic characteristics.

The prebiotics most studied as feed additives in poultry nutrition are oligosaccharides, especially mannan oligosaccharides (MOS), glycoligosaccharides (GOS) and fructooligosaccharides (FOS). GOS and FOS are substrates for beneficial bacteria widely used in various species, as they are easy to produce and present good results (Coelho et al., 2014; Nunes et al., 2009). MOS also have the ability to be used as adsorbents for aflatoxins, as they reduce the problems caused by them. mycotoxins, commonly present in low-quality maize (Stringhini et al. 2000). With the use of these drugs, postbiotics are produced, which can biodegrade mycotoxins through mechanisms such as epoxidation, hydroxylation, dehydrogenation, and reduction by extracellular or intracellular enzymes (Hiththatiyage et al., 2025).

The use of probiotics and prebiotics in animal feed emerges as a viable alternative that meets the demand of a more demanding consumer market, since it can provide more health to animals without leaving residues (Dong et al., 2024). They replace antibiotics as growth promoters and immunity enhancers, especially by forming an intestinal microflora that is more resistant to pathogens and can provide more carbohydrates to birds, increasing their energy level (Yang et al., 2025).

Thus, the objective of this study was to evaluate the liver histology of broilers supplemented with a prebiotic compound based on mannan oligosaccharides, fructooligosaccharides and glucoligosaccharides.

3 METHODOLOGY

This study was conducted under the terms and conditions of the Ethics Committee on the Use of Animals of the Federal Rural University of Pernambuco, approved under number 135/2018. A total of 240 female birds of the Coob lineage were distributed in three experimental treatments with three replicates of four birds each, in the period from one to 42 days of age. When housed, the birds received water and feed ad libitum. The experimental treatments were: Treatment 1 500 g of prebiotic compound per ton of feed, Treatment 2 16.5 ppm of Flavomycin per ton of feed, Treatment 3 feed without any treatment. In Treatment 1, the prebiotic compound used is considered a food additive and is based on mannan

oligosaccharides (MOS), glucoligosaccharides (GOS) and fructooligosaccharides (FOS). The diets were prepared according to recommendations recommended for the lineage.

The corn used in the manufacture of poultry feed contained an average of 3.5 ppb for Aflatoxin and no adsorbent was added for mycotoxins. When housed, the birds received water and feed ad libitum. Liver samples were collected at 7, 14, 28 and 42 days after lodging, fixed in 10% buffered formaldehyde in 0.1M phosphate buffer and pH 7.2. After 24 hours in the fixator, the fragments were cleaved and kept in the same fixator for another 24 hours, and then placed in a 70% alcohol solution until the beginning of histological processing. Following the routine protocol of the Histology Laboratory of the Department of Animal Morphology and Physiology of UFRPE, the liver fragments were dehydrated in increasing concentrations of ethyl alcohol, diaphanized in xylol, impregnated and embedded in paraffin. Then, the blocks were cut into 3 µm thickness in a rotating microtome, Leica® RM2125RT model, and the sections obtained were with hematoxylin and eosin.

In the histopathological evaluation, the lesions were analyzed for the degree of intensity as Absent, Discrete, Moderate or Severe. And as for its distribution, in Absent, Focal, Foclavly extensive, Multifocal, Multifocal to coalescent or diffuse.

The experimental design was completely randomized. Histopathological changes were analyzed by frequency distribution and statistical inference, using the non-parametric Kruskal-Wallis test, adopting a significance level of 5% and using GraphPad Prism 7.0.

4 RESULTS AND DISCUSSIONS

At 7 days of age, Treatment 1 (T1) had a lower frequency of vacuolar degeneration, hepatitis, and liver cell hypertrophy compared to Treatment 2 (T2), although it showed a higher frequency of hepatic steatosis. In relation to Treatment 3 (T3), T1 showed less hepatic congestion, vacuolar degeneration and Kupffer cell hypertrophy, but a higher occurrence of hepatic steatosis and hepatocyte necrosis (Table 1). The main histopathological changes visualized are illustrated in Figure 1.

These alterations are compatible with the classic pattern of aflatoxin-induced hepatotoxicity, characterized by vacuolar degeneration, steatosis, necrosis, and biliary proliferation. The literature describes that aflatoxin promotes direct hepatocellular damage through reactive metabolites formed in the liver, resulting in structural and functional changes proportional to the dose ingested. Patterson (1983) states that aflatoxin induces the formation of liver tumors in several animal species, evidencing its hepatotoxic potential.

At 14 days, T1 showed a lower frequency of vacuolar degeneration and bile duct proliferation when compared to T2, in addition to lower vacuolar degeneration in relation to

T3 (Table 1). The lower intensity of these lesions at early ages suggests a possible modulating effect of the prebiotic compound on the bioavailability or absorption of the toxin. Considering that the liver is the main target organ of aflatoxin, alterations such as vacuolar degeneration reflect early metabolic impairment (Clifford and Rees, 1996), especially in young birds, whose hepatic system is still in functional maturation (Quezada et al., 2000).

At 28 days, T1 had a lower frequency of hepatic steatosis than T2, but a higher occurrence of hepatic congestion, vacuolar degeneration, and hepatitis. When compared to T3, less hepatic steatosis was observed, but greater vacuolar degeneration, hemorrhage, liver cell hypertrophy, and hepatitis (Table 1). This pattern suggests that, although initial protection may have occurred, the toxic challenge remained throughout the production cycle.

At the age of 42 days, there was a general improvement in all treatments, although T1 maintained greater vacuolar degeneration and hepatitis when compared to T3 (Table 1). This progressive reduction of lesions with advancing age may be related to both metabolic adaptation and a decrease in the effective load of circulating toxin. Chen et al. (1984) demonstrated that aflatoxin can be detected in tissues a few hours after ingestion, but is no longer identifiable four days after removal of the contaminated feed, showing relatively rapid clearance capacity after cessation of exposure.

The analysis of the distribution and intensity of the lesions (Table 2) reinforces the dynamic character of the lesion process. At 7 days, there was a statistical difference in hemorrhage (distribution and intensity) and in the intensity of liver cell micronodules. At 14 days, hepatic congestion, hemorrhage, hepatic cell hypertrophy, micronodules, and hepatocyte necrosis showed statistical differences in distribution, while congestion, hemorrhage, hypertrophy, micronodules, necrosis, and bile duct proliferation differed in intensity. At 28 days, necrosis of hepatocytes and proliferation of bile ducts maintained statistical differences in both distribution and intensity, and at 42 days differences persisted for congestion, hemorrhage, hypertrophy, micronodules, necrosis, and biliary proliferation.

Bile duct proliferation and hepatocyte necrosis are alterations often associated with chronic or subacute toxic processes. Bintvihok et al. (2002) demonstrated that aflatoxin levels in the liver are approximately ten times higher than those found in muscle, confirming the liver as the main site of accumulation and biotransformation of the toxin. In addition, they observed a reduction in liver levels after removal of contaminated food, which corroborates the progressive improvement observed at 42 days.

Table 1

Relative (%) and absolute frequency of histopathological changes in the liver of birds supplemented with prebiotic compound based on mannan oligosaccharides, fructooligosaccharides and glucoligosaccharides at 7, 14, 28 and 42 days

Alterações	07 dias			14 dias			28 dias			42 dias		
	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3
	n=12	n=12	n=12	n=12	n=12	n=12	n=12	n=12	n=12	n=12	n=12	n=12
Congestão hepática	8,33 (1/12)	0,00 (0/12)	25,00 (3/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	8,33 (1/12)	0,00 (0/12)	8,33 (1/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)
Esteatose hepática	41,67 (5/12)	16,67 (2/12)	8,33 (1/12)	16,67 (2/12)	16,67 (2/12)	16,67 (2/12)	0,00 (0/12)	8,33 (1/12)	16,67 (2/12)	0,00 (0/12)	0,00 (0/12)	16,67 (2/12)
Degeneração vacuolar	33,33 (4/12)	50,00 (6/12)	75,00 (9/12)	25,00 (3/12)	33,33 (4/12)	41,67 (5/12)	66,67 (8/12)	50,00 (6/12)	58,33 (7/12)	58,33 (7/12)	41,67 (5/12)	41,67 (5/12)
Hemorragia	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	8,33 (1/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)
Hepatite	0,00 (0/12)	25,00 (3/12)	16,67 (2/12)	41,67 (5/12)	41,67 (5/12)	41,67 (5/12)	50,00 (6/12)	33,33 (4/12)	41,67 (5/12)	16,67 (2/12)	8,33 (1/12)	8,33 (1/12)
Hipertrofia de células de Hepática	8,33 (1/12)	16,67 (2/12)	16,67 (2/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	8,33 (1/12)	8,33 (1/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)
Micro nódulo células de Hepática	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	8,33 (1/12)	8,33 (1/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)
Necrose de hepatócitos	8,33 (1/12)	8,33 (1/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)
Proliferação de ductos biliares	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	8,33 (1/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)	0,00 (0/12)

T1 – Treatment 1: 500 g of prebiotic compound per ton of feed; T2 – Treatment 2: 16.5 ppm of Flavomycinper ton of feed; T3 – Treatment 3: feed without any treatment. Kruskal-Wallis test at the level of 5% significance.
Source: Authors.

Table 2

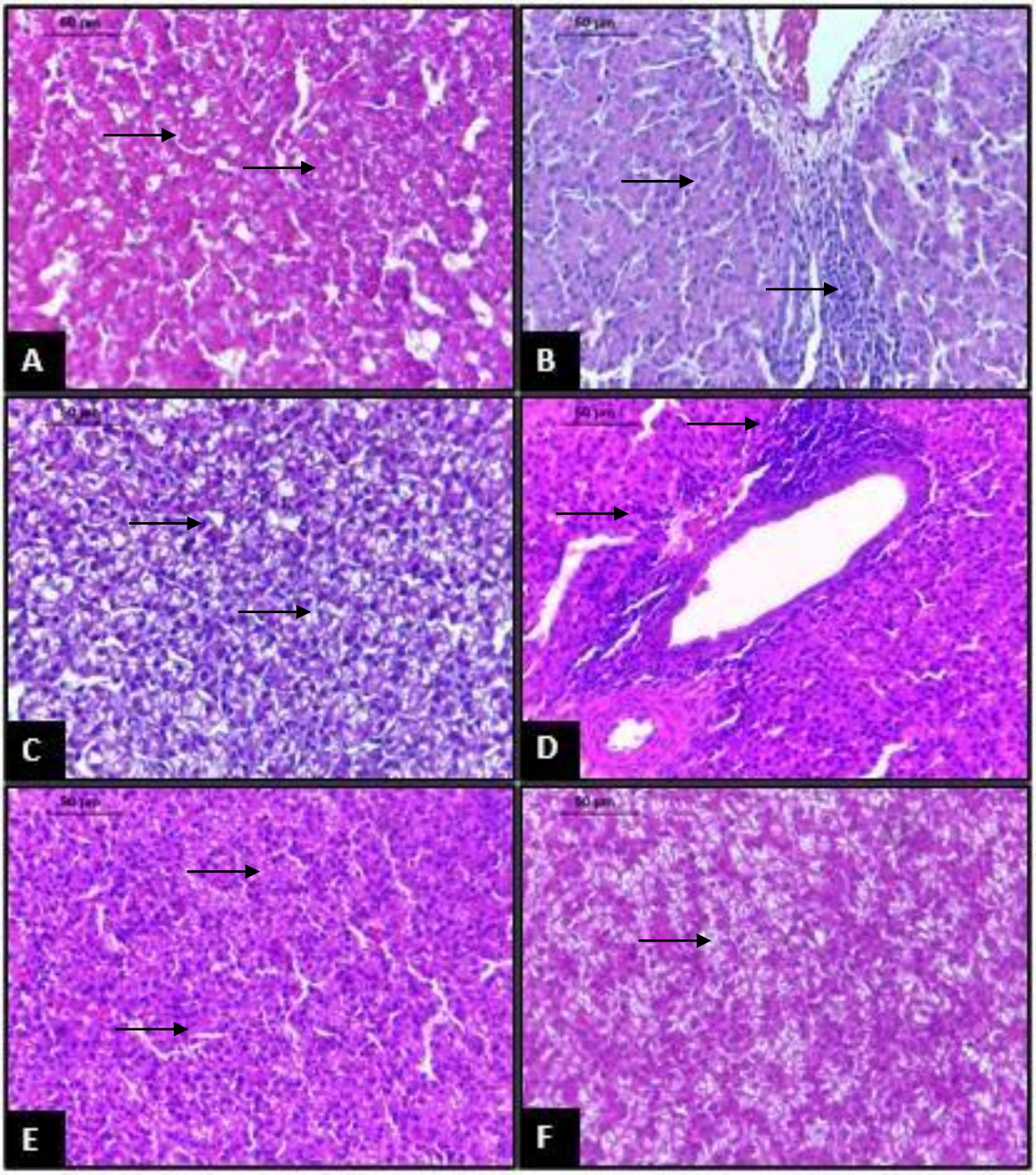
Medians of the distribution and intensity of histopathological changes in the liver of birds supplemented with prebiotic compound based on mannan oligosaccharides, fructooligosaccharides and glucoligosaccharides at 7, 14, 28 and 42 days

Alterações	07 dias				14 dias				28 dias				42 dias			
	T1	T2	T3	Valor de P*	T1	T2	T3	Valor de P*	T1	T2	T3	Valor de P*	T1	T2	T3	Valor de P*
	n=12	n=12	n=12		n=12	n=12	n=12		n=12	n=12	n=12		n=12	n=12	n=12	
Distribuição																
Congestão hepática	0,00	0,00	0,00	0,147	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,597	0,00	0,00	0,00	0,0001
Esteatose hepática	0,00	0,00	0,00	0,335	0,00	0,00	0,00	0,751	0,00	0,00	0,00	0,356	0,00	0,00	0,00	0,127
Degeneração vacuolar	0,00	0,00	5,00	0,068	0,00	0,00	0,00	0,563	5,00	1,50	2,00	0,591	5,00	0,00	0,00	0,648
Hemorragia	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,367	0,00	0,00	0,00	0,0001
Hepatite	0,00	0,00	0,00	0,191	0,00	0,00	0,00	0,982	0,00	0,00	0,00	0,951	0,00	0,00	0,00	0,795
Hipertrofia de células de Hepática	0,00	0,00	0,00	0,750	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,342	0,00	0,00	0,00	0,0001
Micro nódulo células de Hepática	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,597	0,00	0,00	0,00	0,0001
Necrose de hepatócitos	0,00	0,00	0,00	0,597	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,0001
Proliferação de ductos biliares	0,00	0,00	0,00	0,597	0,00	0,00	0,00	0,367	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,0001
Intensidade																
Congestão hepática	0,00	0,00	0,00	0,162	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,326	0,00	0,00	0,00	0,0001
Esteatose hepática	0,00	0,00	0,00	0,331	0,00	0,00	0,00	0,978	0,00	0,00	0,00	0,366	0,00	0,00	0,00	0,127
Degeneração vacuolar	0,00	0,00	1,00	0,076	0,00	0,00	0,00	0,651	1,00	0,50	0,50	0,548	1,50	0,00	0,00	0,687
Hemorragia	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,367	0,00	0,00	0,00	0,0001
Hepatite	0,00	0,00	0,00	0,205	0,00	0,00	0,00	0,981	0,00	0,00	0,00	0,973	0,00	0,00	0,00	0,793
Hipertrofia de células de Hepática	0,00	0,00	0,00	0,797	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,367	0,00	0,00	0,00	0,0001
Micro nódulo células de Hepática	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,597	0,00	0,00	0,00	0,0001
Necrose de hepatócitos	0,00	0,00	0,00	0,597	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,0001
Proliferação de ductos biliares	0,00	0,00	0,00	0,367	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,0001	0,00	0,00	0,00	0,0001

T1 – Treatment 1: 500 g of prebiotic compound per ton of feed; T2 – Treatment 2: 16.5 ppm of Flavomycin per ton of feed; T3 – Treatment 3: feed without any treatment. Kruskal-Wallis test at the level of 5% significance.
Source: Authors.

Figure 1

Photomicrograph of poultry liver. (A) Microvesicular fat degeneration at 7 days at T1, HE, =50 µm bar; (B) Periportal lymphohistiocytic infiltrate at 14 days on T1, HE, = 50 µm bar; (C) Vacuolar degeneration at 14 days in T1, HE, =50 µm bar; (D) Perivascular lymphohistiocytic infiltrate at 28 days on T1-weighted sequences, HE, =50 µm bar; (E) Vacuolar degeneration at 28 days at T1, HE, =50 µm bar; (F) Vacuolar degeneration at 42 days at T1, HE, =50 µm bar



Source: Authors.

Hussain et al. (2016) reported that birds exposed to high levels of aflatoxin had a higher number of liver lesions and reduced food and water intake. This finding is relevant, because the decrease in intake can alter the dynamics of exposure to the toxin and influence

the intensity of the lesions observed. Hepatic impairment can reduce appetite and affect metabolic capacity, establishing a cycle of aggression and adaptive response.

Considering that this study reflects real farm conditions, it is unlikely that all birds ingested the same amount of toxin simultaneously. Thus, individual variations in hepatic intake and metabolic capacity may explain differences in the frequency and intensity of lesions between treatments. In general, there was a trend of reduction in liver alterations with advancing age, suggesting an inverse relationship between lesion intensity and maturity of the birds. Patterson (1983) points out that, although aflatoxin has carcinogenic potential, the short production cycle of broiler chickens limits the manifestation of neoplastic alterations, being restricted predominantly to degenerative and inflammatory lesions.

The results obtained indicate that the prebiotic compound may have exerted a modulating effect mainly in the initial phases, evidenced by the lower frequency of vacuolar degeneration and hepatic congestion at 14 days. However, the persistence of lesions at 28 days demonstrates that the toxic challenge was not completely neutralized, although there was an overall improvement at the end of the experimental period.

5 CONCLUSION

The prebiotic compound evaluated showed a suggestive effect of liver protection against exposure to aflatoxin, especially in the early stages of rearing, reducing the frequency of lesions such as vacuolar degeneration and hepatic congestion. A trend of regression of the changes was observed with advancing age, although the toxic challenge persisted during the productive cycle. The findings indicate a potential modulatory effect of the prebiotic on aflatoxin-induced liver lesions in broilers, especially in the early development period.

ACKNOWLEDGMENTS

The authors thank the team at Rancho Brejo Santo, the Department of Animal Morphology and Physiology, Federal Rural University of Pernambuco (UFRPE) and the Coordination for the Improvement of Higher Education Personnel (CAPES).

REFERENCES

Bintvihok, A., Thiengnin, S., Doi, K., & Kumagai, S. (2002). Residues of aflatoxins in the liver, muscle and eggs of domestic fowls. Journal of Veterinary Medical Science, 64(11), 1037–1039.

Büzen, S., & Haese, D. (2006). Controle de micotoxinas na alimentação de aves e suínos. Revista Eletrônica Nutritime, 3(1), 304–309.

Chen, C., Pearson, A. M., Coleman, T. H., Gray, J. I., Pestka, J. J., & Aust, S. D. (1984). Tissue deposition and clearance of aflatoxins from broiler chickens fed a contaminated diet. Food and Chemical Toxicology, 22(6), 447–451.

Clifford, J., & Rees, K. (1966). Aflatoxin: A site of action in the rat liver cell. Nature, 209, 312–313. https://doi.org/10.1038/209312a0

Hussain, Z., Khan, M. Z., Saleemi, M. K., Khan, A., & Rafique, S. (2016). Clinicopathological effects of prolonged intoxication of aflatoxin B1 in broiler chicken. Pakistan Veterinary Journal, 36(4), 477–481.

Merkley, J. W., Maxwell, R. J., Phillips, J. G., & Huff, W. E. (1987). Hepatic fatty acid profiles in aflatoxin-exposed broiler chickens. Poultry Science, 66(1), 59–67.

Monson, M. S., Coulombe, R. A., & Reed, K. M. (2015). Aflatoxicosis: Lessons from toxicity and responses to aflatoxin B1 in poultry. Agriculture, 5, 742–777.

Patterson, D. S. P. (1983). Aflatoxin in farm animals. Veterinary Research Communications, 7, 135–145.

Quezada, T., Cuéllar, H., Jaramillo-Juárez, F., Valdivia, A. G., & Reyes, J. (2000). Effects of aflatoxin B1 on the liver and kidney of broiler chickens during development. Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology, 125(3), 265–272. https://doi.org/10.1016/S0742-8413(99)00107-3

Silva, W. T. M., Borsatti, L., Nunes, R. V., Pozza, P. C., Pozza, M. S. S., & Giusti, L. D. B. (2012). Inulina na produção de frangos de corte. Scientia Agraria Paranaensis, 11(3), 16–24.

Stringhini, J. H., Mogyca, N. S., & Andrade, M. A. (2000). Efeito da qualidade do milho no desempenho de frangos de corte. Revista Brasileira de Zootecnia, 29(1), 191–198.