EFFECT OF ESTERIFIED GLUCOMANNAN ON ALLEVIATION OF AFLATOXICOSIS IN BROILER CHICKEN

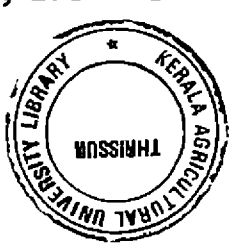
MALDHURE NITEEN ARVIND

Thesis submitted in partial fulfilment of the requirement for the degree of

Master of Veterinary Science

Faculty of Veterinary and Animal Sciences
Kerala Agricultural University, Thrissur

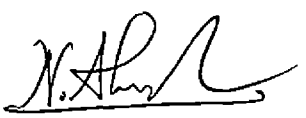
2007



Department of Poultry Science
COLLEGE OF VETERINARY AND ANIMAL SCIENCES
MANNUTHY, THRISSUR-680651
KERALA, INDIA

DECLARATION

I hereby declare that this thesis, entitled **"EFFECT OF ESTERIFIED GLUCOMANNAN ON ALLEVIATION OF AFLATOXICOSIS IN BROILER CHICKEN"** is a bonafide record of research work done by me during the course of research and that the thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship or other similar title, of any other University or Society.



Mannuthy,

MALDHURE NITEEN ARVIND

14.06.07

CERTIFICATE

Certified that this thesis, entitled **"EFFECT OF ESTERIFIED GLUCOMANNAN ON ALLEVIATION OF AFLATOXICOSIS IN BROILER CHICKEN"** is a record of research work done independently by **Dr. Maldhure Niteen Arvind,** under my guidance and supervision and it has not previously formed the basis for the award of any degree, diploma, fellowship or associateship to him.

Mannuthy

14.06.07

Dr. R. Richard Churchil,
(Chairman, Advisory Committee)
Assistant Professor,
AICRP on Poultry for Eggs
Department of Poultry Science,
College of Veterinary and Animal Sciences,
Mannuthy.

CERTIFICATE

We, the undersigned members of the Advisory Committee of **Dr. MALDHURE NITEEN ARVIND.**, a candidate for the degree of Master of Veterinary Science in Poultry Science, agree that this thesis entitled **"EFFECT OF ESTERIFIED GLUCOMANNAN ON ALLEVIATION OF AFLATOXICOSIS IN BROILER CHICKEN"** may be submitted by **Dr. MALDHURE NITEEN ARVIND.**, in partial fulfillment of the requirement for the degree.

Dr. R. Richard Churchil,
(Chairman, Advisory Committee)
Assistant Professor,
AICRP on Poultry for Eggs
College of Veterinary and Animal Sciences, Mannuthy.

Dr. A. Jalaludeen,
Associate Professor and Director i/c,
Centre for Advanced Studies in Poultry Science
College of Veterinary and Animal Sciences, Mannuthy.
(Member)

Dr. Leo Joseph
Associate Professor and Head,
University Poultry Farm,
College of Veterinary and Animal Sciences, Mannuthy.
(Member)

Dr. Stephen Mathew,
Associate Professor,
Department of Animal Genetics And Breeding,
College of Veterinary and Animal Sciences, Mannuthy
(Member)

19/09/08
EXTERNAL EXAMINER (S.C. EDWIN)
Associate Professor & Head,
Dept. of Poultry Science
Veterinary College & Res. Institute
Namakkal - 637 002.

ACKNOWLEDGEMENT

Today I met the finale of my endeavor but the search for suitable thanks is still not over as it is beyond the power of my expression for my esteemed advisor ***Dr. R. Richard Churchil,*** *Assistant Professor, AICRP on Poultry for eggs, Department of Poultry Science. It was indeed a pleasure for me to work under his superb guidance. His valuable guidance, creative suggestions, constructive criticism and constant encouragement during the course are not only praiseworthy but also unforgettable. I am particularly grateful for his emphasis on simplicity and elegance in life.*

It was indeed a fortunate to have ***Dr. A. Jalaludeen,*** *Associate Professor and Director i/c, Centre for Advanced Studies in Poultry Science, as a member of the advisory committee. He provided a persistent, gentle push to wrap the thing up all along during my entire course of work. He has remained extremely approachable and friendly. I thank him for his words of wisdom and for his feedback.*

To the greatest degree, I am grateful to ***Dr. Leo Joseph,*** *Associate professor & Head, University Poultry Farm, member of the Advisory Committee, for his generous encouragement, inspiration, kindness and personal guidance in the pursuit of this work.*

I am sincerely thankful to ***Dr. Stephen Mathew,*** *Associate Professor, Department of Animal Genetics and Breeding, as a member of the advisory committee, for his whole-hearted co-operation during the period of research work.*

As such it is imperative to thank ***Dr. Narayanankutty,*** *Senior Scientist, AICRP on Poultry for Eggs and* ***Dr. Peethambaran,*** *Associate Professor, Department of Poultry Science, as part of the work would not have been possible without a great deal of support from him. He was quite but always insightful. His approach deserves the highest respect I can give.*

I owe my sincere gratitude to Department of Poultry Science ***Dr. Amritha Viswanath,*** *Associate Professor, Department of Poultry Science,* ***Dr. P. Anitha,*** *Assistant Professor Department of Poultry Science for their valuable guidance, timely help and moral support.*

My two years at COVAS were made much more enjoyable by the companionship of my colleagues, ***Drs. Simi G.*** *and* ***Preeta Raghvan,*** *I feel lucky to share my postgraduate studies with them.*

I sincerely acknowledge the staff of our department ***Mrs. Vilasini*** *and* ***Mr. Paul*** *for their timely help.*

I thank farm workers AICRP on Poultry for eggs, for the help rendered in my research work.

I gratefully acknowledge ***Dr. Mercy,*** *Associate Professor and Head, Department of Nutrition and* ***Dr. Lalithakunjamma,*** *Associate Professor and Head, Department of Pathology, for providing the laboratory facilities for my work.*

I gratefully acknowledge ***Dr.Shamsudeen,*** *Assistant professor, TANUVAS and Phd. Scholar, CARI for providing the work materials.*

I take great pleasure in thanking ***Dr. E. Nanu,*** *Dean i/c, Faculty of Veterianry and Animal Sciences, for providing me the facilities for my research.*

I gratefully acknowledge the wholehearted support and help rendered by ***Drs. Binoj, Bipin, Sanjeev kumar, Kishor (K.J.), Dinkar, Upendra, Sujith, Seji and internship students (2001 batch)*** *in assisting me during my research work.*

The invaluable help rendered by my beloved seniors ***Drs. Raseena*** *and* ***Preethymol*** *and juniors* ***Drs. Balagi, Chandni, Bhadra*** *and* ***Shamna*** *are duly acknowledged.*

I am in short of words to express my deep sense of gratefulness for the understanding, love and encouragement of ***Dr. Binoj chacko, M. Sasikumar*** *and* ***Kishor kumar (K.J.)*** *who remains my confident and very good friends.*

My friends have served as a support network in many ways over these years and this journey was no different. I wish to thank my friends ***Drs. Hamza, Tamppan, Kallu, Vivek (TA), Albert, Bibu, Jeenesh, Ranjith, Roy, Biju, Jhotish, Nishant, Rojan, Ganesh, Shaiby, Prince, Darshana, Ann, Tassi, Asha.*** *Their companionship has been invaluable in all facets of my life.*

I was particularly fortunate to have had respectful seniors ***Drs. Lu, Shanmugam, Vikram, Poulson, Rajugopal, Vivek, Sunilji, Rana, Senthil (P.K.), Shekar, Rajaganapathy, Anoop, Rishi, Prejith, Deepak, Babu,*** *in the PG hostel, with whom I have shared many hours of interesting discussions. They helped my stay in hostel as an enjoyable and a memorable one.*

With great fondness, I express my heartfelt thanks to ***Drs. Hamza, Tamppan*** *and* ***Deepak,*** *for their help and co-operation. Thank you very much for being friendly with me.*

I would like to thank ***Dr. Praveena Churchil*** *for her care and kindliness showed on me forever.*

No words of thanks could be enough to those ***mute creatures,*** *which have laid down their invaluable life for my work. I am deeply indepted from bottom of my heart to those creatures.*

A very big thanks goes of course to my ***loving parents*** *for instilling the love of learning in me and to my brother and sister for accompanying me through the ups and downs of my life. They helped me not only in charting a great course but also in providing me with the skills I need to conquer the road ahead.*

Without the help of these nothing would have been possible. I bow before the ***almighty,*** *for the interminable blessings that have helped me in every stage of my life.*

Dr. Niteen

Dedicated To
My Parents And Teachers

CONTENTS

LIST OF TABLES

LIST OF FIGURES

Introduction

1. INTRODUCTION

Indian poultry industry has registered a growth rate of 15 to 20 per cent annually during the last two decades. Chicken meat production in India has increased from 1.08 million metric tones in 2000 to 1.60 million metric tones during 2004 (FAO, 2005). Broiler industry in India has gained spectacular progress and has transformed into imperative industrial status. This accomplishment is attributed to scientific breeding and management ordained through tenacious research. India contributes 2.39 per cent of world's total poultry meat production. However, poultry meat consumption in India is only 1.6 kg per year, which is far below the world's per capita poultry meat consumption of 11 kg per year. To meet the gap between production and requirement the Indian broiler industry has to expand at least by five times. This can only be achieved by systematic expansion based on scientific research and aptitude. Breeding and nutrition will continue to be the two major areas of future research. Nutritional research focusing on maximizing feed utilization is a good proposition to utilize the available feed resources to the maximum extent for poultry production.

The poultry industry is concerned about mycotoxins because its presence in the feed of poultry results in great economic losses. It must be remembered that the word 'toxin' means 'poison' and poison causes losses in productivity. Aflatoxins, a group of extremely toxic chemicals are produced by certain species of fungi belonging to the genus Aspergillus. Aflatoxin B1 and three structurally similar aflatoxin compounds (B2, G1 and G2) have been detected as contaminants of crops before harvest, between harvesting and drying, during storage and after processing or manufacturing (Council for Agricultural Science and Technology, 1989).

Scientists based in the related fields of animal nutrition and various research institutions focused their attention towards aflatoxin only after the outbreak of Turkey 'X' disease which caused the mortality of over 1,00,000 turkey poults in the United Kingdom during 1960. Poor growth rate (Smith and Hamilton, 1970), poor feed conversion efficiency (Reddy *et al.*, 1982) and immuno-suppression (Devurkar *et al.*, 1995) are the most perilous hazards of aflatoxin causing great economic losses to the broiler farmers. Aflatoxin B1, the most toxic of all aflatoxins is immunosuppressant, hepatocarcinogenic, teratogenic and mutagenic. Aflatoxins affect the commercial broiler production by reducing growth rate, downgrading of carcass, increasing disease susceptibility and mortality and affecting nutrient metabolism.

The fungal contamination of feeds and feedstuffs can be reduced to certain extent by adopting proper pre-harvest, harvest and post-harvest technologies. A variety of physical, chemical and biological methods of detoxification have been reported. But practical, cost effective and large-scale methods for detoxifying aflatoxin in the feeds and feed ingredients are currently not available with the exception of natural and synthetic zeolites. Zeolitic compounds may have some adverse effects including increased incidence of tibial dyschondroplasia and reduced bone calcification in broiler chicks (Edwards, 1988). However, rapid progress in the field of biotechnology has opened an avenue for biological means of detoxification.

Esterified glucomannan (E-GM), a new generation biological toxin binder is enzymatically extracted from the cell wall of certain strains of yeast *Saccharomyces cereviciae* has been identified by the modern biotechnology as promising substance for mycotoxin neutralization. It has been successfully shown to alleviate the toxic effects of aflatoxin and other mycotoxins in broilers by few

research workers (Raju and Devegowda, 2000; Basmacioglu *et al.* 2005). The porous nature of the E-GM provides more surface area for adsorption at low inclusion levels with none of the negative side effects attributed to mineral clay binders. E-GM binds aflatoxins and reduces the harmful effects of aflatoxins in chicken (Devegowda *et al.*, 1998). This effect of E-GM might be attributed to mycotoxin adsorption. The evidence currently seems to suggest that the E-GM can fulfill the necessary criteria for a safe and cost-effective solution to mycotoxin contamination. In addition, E-GM has the ability to block colonization of pathogens in the gastrointestinal tract. However, the effect of E-GM in aflatoxin free diets has not been studied yet by the earlier researchers. Further, the benefit of adding E-GM on economics has not been investigated by any of the earlier worker. Therefore a composite study throwing light on these neglected areas was designed with the following objective-

- To study the effect of Esterified Glucomannan as feed additive in counteracting the severity of aflatoxicosis in broilers.

Review of Literature

2. REVIEW OF LITERATURE

The aflatoxicosis was first reported in turkeys (Turkey 'X' disease) in the year 1961 (Blount, 1961). Since then several studies on aflatoxicosis had shown that aflatoxin is a potent growth depressant (Smith and Hamilton 1970; Chattopadhyay *et al.* 1985; Johri *et al.* 1990), teratogenic, carcinogenic and mutagenic (Oguz and Kurtoglu, 2000) in broiler chicken. Among aflatoxins, aflatoxin B1, a metabolite of *Aspergillus flavus* and *Aspergillus parasiticus*, is extremely hepatotoxic to birds (Rizzi *et al.*, 1998). Aflatoxin causes a variety of effects in poultry including poor performance, altered organ morphology and altered serum biochemistry and haematology (Churchil, 1996; Raju and Devegowda, 2000; Afzal and Saleem, 2004). Aflatoxin also causes severe gross and microscopic changes in the liver, kidney, bursa and spleen (Espada *et al.*, 1992; Ledoux *et al.*, 1999). Aflatoxins impair the humoral and cellular immune responses and increase susceptibility to some environmental and infectious agents (Gabal and Azzam, 1998). Relevant scientific reports on the effect of aflatoxin on broilers have been reviewed in the following section.

2.1 EFFECT OF AFLATOXIN

2.1.1 Body Weight

The first observation of growth depression due to aflatoxicosis was recorded by Blount (1961).

Kratzer *et al.* (1969) studied the effect of dietary aflatoxin at 1600, 800 and 400 ppb levels on body weight of broiler chicken. At 1600 ppb, the growth retardation was evident but not significant. Whereas at 800 ppb, the mean body weight of aflatoxin fed chicks exceeded that of control group. No adverse effects were noted on body weight at 400 ppb aflatoxin level.

Smith and Hamilton (1970) reported a threshold level of 1.25 ppm aflatoxin for broilers, above which the growth depression was more evident.

In a study on commercial broilers, feeding aflatoxin at 10 ppm lowered the growth rate by about half at four weeks of age (Smith *et al.*, 1971).

Chattopadhyay *et al.* (1985) observed a considerable decrease in body weight in aflatoxin treated chicks during initial five to six weeks of the experiment. However, this effect was diminished during the later part of the study.

Churchil (1996) reported poor growth rate and significant reduction in mean body weight from second week onwards in aflatoxin (1 ppm) treated broiler birds during his study for eight weeks period.

Mani *et al.* (2000a) studied the performance of three commercial broiler strains under aflatoxicosis. Three commercial broiler strains were fed with diet containing either 0.00 or 0.20 ppm aflatoxin B1 for a period of eight weeks and found that aflatoxin fed groups in all the strains recorded decreased body weight on 5, 27, 42 and 56 days of age.

Raju and Devegowda (2000) reported that the 35 days body weight of broiler chicks in aflatoxin fed group (0·3 mg/kg) was significantly ($P<0·01$) lowered.

Mani *et al.* (2001) studied the effect of aflatoxin B1 (200 ppb) on the performance of broilers and reported significantly lower body weight at eight weeks of age in aflatoxin B1 fed group.

Girish and Devegowda (2006) reported that the body weight of broiler chickens receiving 2 mg per kg aflatoxin was significantly ($P<0.05$) reduced at 35 days age.

2.1.2 Weight Gain

A significant reduction in body weight gain was observed by San-Gabriel (1971) when aflatoxin was added at 450 ppb level to the feed in broiler chicken.

Chicken fed with ration containing 2500 or 5000 ppb aflatoxin for a period of three weeks showed depressed body weight gain (Lanza *et al.*, 1980; Doerr and Huff 1981; Huff *et al.* 1986 and Giroir *et al.* 1991).

Hubbard broiler strain when fed with 50 and 100 ppb aflatoxin in the feed for a period of five weeks showed a reduction in body weight gain (Mashaly *et al.*, 1986).

A progressive decrease in body weight gain in broilers was observed by Johri *et al.* (1990) when the level of aflatoxin was increased from 0.0 to 0.75 ppm. However the difference in weight gain was statistically insignificant up to 0.2 ppm aflatoxin.

Dose related reduction in body weight gain was observed by Vasan *et al.* (1998), when day-old Vencobb broiler chicks were maintained on diets contaminated with 0, 0.25, 0.5, and 1 ppm aflatoxin B1 for a period of four weeks.

Rosa *et al.* (2001) reported significant ($P<0.05$) reduction in body weight gain in broilers receiving aflatoxin B1 (5 mg/kg) in diet from 30 to 52 days of age.

Shivachandra *et al.* (2003a) studied the effect of aflatoxin (1 ppm) on broiler chicks from seventh day to seventh week of age and found lowered mean body weight gain up to 42 days post feeding.

Pimpukdee *et al.* (2004) reported that aflatoxin B1 at 5 mg per kg diet significantly affected the broiler health and production. The reduction in body weight gain was significant after first week and at the end of the study (3 weeks), there was 19 per cent reduction in weight gain.

Basmacioglu *et al.* (2005) studied the effect of dietary aflatoxin (2 mg /kg) in broilers for a period of 21 days of age and found significant ($P<0.05$) decrease in body weight gain (11.71 %) from second week onwards compared with the control.

Bintvihok and Kositcharoenkul (2006) reported reduced body weight gain in broilers receiving 50 ppb and 100 ppb aflatoxin B1 for a period of six weeks.

2.1.3 Feed Intake

Reddy *et al.* (1982) observed that the feed consumption was adversely affected in commercial broiler chicks fed a diet contaminated with 0.5 ppm aflatoxin.

On the contrary, Maurice *et al.* (1982) noticed no significant change in feed intake of broilers subjected to aflatoxin treatment at 350 and 650 ppb levels.

Ghosh and Chauhan (1991) observed that the feed consumption decreased ($P<0.05$) in chicks fed 0.3 and 1 ppm aflatoxin B1 from day old to six weeks of age.

Churchil (1996) reported that the weekly cumulative feed consumption of broilers receiving aflatoxin (1 ppm) treated feed was lesser than control throughout the experimental period (8 weeks).

Dose related reduction in feed intake was observed by Vasan *et al.* (1998) when day old Vencobb broiler chicks were maintained on diets contaminated with 0, 0.25, 0.5, and 1 ppm aflatoxin B1 for four weeks period.

Mani *et al.* (2000a) observed a considerable decrease in feed consumption in chicks treated with aflatoxin B1 (0.2 ppm) from day-old to eight weeks of age.

Raju and Devegowda (2000) studied the effect of dietary aflatoxin at 0.3 mg per kg level in broilers from day-old to 35 days of age and noticed significant ($P < 0.01$) decrease in feed consumption in aflatoxin fed group from second week onwards. They also found that the cumulative feed intake was depressed by 10 per cent in aflatoxin treated group at 35 days of age compared to control.

Mani *et al.* (2001) reported less feed consumption in eight weeks old broilers fed aflatoxin B1 at 200 ppb level.

Reduction in feed intake was observed by Shivachandra *et al.* (2003a) when broiler chicks were maintained on aflatoxin (1 ppm) contaminated diet from seventh day to seventh week of age.

Tedesco *et al.* (2004) noticed that the mean feed intake was decreased significantly ($P<0.05$) in the last 2 weeks of experimental period when broiler chicks were fed with 0.8 mg per kg aflatoxin B1 for 35 days.

Basmacioglu *et al.* (2005) reported a reduction of 8 to 11 per cent feed consumption in aflatoxin (2 mg/kg) treated group during an experiment on broilers for 21 days; however, the decrease was not statistically significant.

Bintvihok and Kositcharoenkul (2006) observed that the feed consumption was adversely affected in broilers receiving aflatoxin B1 contaminated diets (50 ppb and 100 ppb) for a period of six weeks.

Girish and Devegowda (2006) reported that the feed intake was adversely affected at 35 days of age in commercial broiler chicks fed a diet contaminated with 2 mg aflatoxin per kg of feed.

2.1.4 Feed Efficiency

Smith *et al.* (1971) recorded decreased efficiency of feed conversion at the dose of 10 ppm aflatoxin in a commercial broiler diet.

Reddy *et al.* (1982) observed that the feed efficiency was adversely affected in commercial broiler chicks fed a diet contaminated with 1.25 ppm aflatoxin.

On the contrary, Shen *et al.* (1987) reported an improved feed efficiency at lower levels of aflatoxin (15 to 450 ppb) in broiler chickens.

According to Ghosh and Chauhan (1991), the feed efficiency decreased significantly ($P<0.05$) in the chicks fed 0.3 and 1 ppm aflatoxin B1 from day old to six weeks of age.

Mani (1995) fed broiler chickens with graded levels of aflatoxin and found that 0.2 ppm and above caused poor feed efficiency in eight weeks old broilers.

Sudarshan *et al.* (1995) reported that the feed efficiency of broilers was significantly ($P<0.05$) decreased (30 per cent) on feeding 0.5 ppm aflatoxin when compared to control.

Churchil (1996) reported poor weekly feed efficiency up to eight weeks period in broiler chicken fed 1 ppm aflatoxin.

Swamy and Devegowda (1998) reported that the feed efficiency of broilers was significantly ($P<0.05$) decreased on feeding of 200 and 400 ppb aflatoxin for a period of six weeks compared to control group.

Vasan *et al.* (1998) reported significantly ($P<0.05$) lower feed efficiency in dose dependant manner when day-old Vencobb broiler chicks were maintained on diets contaminated with 0.25, 0.5, and 1 ppm aflatoxin B1 for four weeks.

In a study conducted on broiler chicks for eight weeks period, Mani *et al.* (2000a) observed that the efficiency of feed utilization was decreased at 0.2 ppm dietary aflatoxin level.

Raju and Devegowda (2000) observed that the feed conversion efficiency value was increased in broiler chicks receiving aflatoxin contaminated diet (0.3 mg/kg) for a period of 35 days.

Mani *et al.* (2001) studied the effect of aflatoxin (200 ppb) on the performance of broilers and reported poor feed efficiency at eight weeks in aflatoxin fed group.

Bintvihok and Kositcharoenkul (2006) reported decreased efficiency of feed conversion in hybrid Arbor Acor broiler chickens fed aflatoxin B1 (50 and 100 ppb) for a period of six weeks.

Girish and Devegowda (2006) reported increased value of feed conversion ratio indicating poor efficiency in 35 days old broiler chicks on feeding aflatoxin at 2 mg per kg level.

2.1.5 Serum Biochemistry

2.1.5.1 Total Protein

Lanza *et al.* (1980) from their study identified that the plasma proteins as the most sensitive criteria for detecting the susceptibility of broilers to aflatoxin. They found that the level of total protein was depressed by aflatoxin (2.5 and 5.0 ppm) in all age groups of broilers from zero to six weeks of age.

Similarly, Reddy *et al.* (1982) found decreased serum proteins concentration when commercial broiler chicks were fed 0.25 ppm aflatoxin.

Mani *et al.* (1993) reported significant reduction in serum protein levels in broiler chickens fed with 0, 0.75 and 1.50 mg aflatoxin B1 per kg of feed up to eight weeks of age.

Stanley *et al.* (1993) and Nath *et al.* (1995) reported significant reduction in serum total proteins concentration in broiler chickens fed 5 and 2 ppm aflatoxin respectively.

Ali *et al.* (1994) studied the effect of aflatoxin at the level of 1.5 and 3 ppm in broiler chicken from fifth week onward for a period of three weeks and found that serum protein was significantly lowered in aflatoxin treated group.

Churchil (1996) reported significant reduction in serum total protein level in broiler chicken treated with 1 ppm dietary aflatoxin for a period of eight weeks.

In a trial with broiler chickens for a period of six weeks, Swamy and Devegowda (1998) found reduction in total serum proteins at 200 and 400 ppb aflatoxin.

Among the broiler chicks maintained on diets contaminated with 0, 0.25, 0.5, and 1 ppm aflatoxin B1 for four weeks, Vasan *et al.* (1998) reported significant reduction in serum protein in 0.5 and 1 ppm aflatoxin fed groups.

In a study on graded levels of aflatoxicosis (0, 20, 40, 60, 80 and 100 ppb), Arulmozhi *et al.* (2000) recorded a dose-dependant decrease of total serum protein in six week old broiler chicken.

Mani *et al.* (2000a) reported that the serum protein level was lowered in broiler chicken receiving 0.2 ppm of aflatoxin B1 from day-old to eight weeks of age.

Mani *et al.* (2000b) studied the effect of different levels of dietary aflatoxin B1 (0, 100, 200, 300, 400 and 500 ppb) on the blood constituents in commercial broilers and found significant ($P<0.01$) decrease in the serum protein level in aflatoxin treated birds than that of control group. He also reported that the reduction in serum protein level was in gradual fashion with increasing level of aflatoxin B1 in feed.

Raju and Devegowda (2000) reported a decrease of serum protein concentration by 25.4 per cent in broiler chicken fed aflatoxin (0.3 mg/kg) for a period of 35 days. He also reported that the decrease was more pronounced at 21 days of age.

Rosa *et al.* (2001) found reduced levels of serum protein during an experiment on aflatoxicosis (5 mg aflatoxin B1/kg) in broilers from 30 to 52 days of age.

Chaturvedi and Singh (2002) found that the total serum protein level was significantly ($P<0.05$) decreased (30 per cent) in commercial broiler chickens fed a diet containing aflatoxin (3 ppm) for a period of 21 days.

Shivachandra *et al.* (2003b) observed lowered levels of serum protein in broiler chicks fed aflatoxin at 1 ppm level from seventh day to seventh week of age.

Basmacioglu *et al.* (2005) studied the effect of dietary aflatoxin (2 mg/kg) in broilers from zero to 21 days of age and found that the serum total protein level was significantly decreased in aflatoxin treated birds.

2.1.5.2 Albumin

At 0.5 and 2.5 ppm levels of aflatoxin, Chattopadhyay *et al.* (1985) observed no significant difference in serum albumin levels between control and treatment groups.

However, significant reduction in serum albumin concentration was observed by Huff *et al.* (1992) and Smith *et al.* (1992) when the broiler chickens were fed with 3.5 ppm aflatoxin from zero to three weeks of age.

Similar reduction in serum albumin was also recorded by Stanley *et al.* (1993) and Nath *et al.* (1995) when broiler chickens were fed 5 and 2 ppm aflatoxin respectively.

Ali *et al.* (1994) studied the effect of aflatoxin at the levels of 1.5 and 3 ppm in broiler chicken from fifth week onwards for a period of three weeks and reported significant reduction in serum albumin in aflatoxin treated groups.

Vasan *et al.* (1998) studied the effect of aflatoxin at the levels of 0, 0.25, 0.5, and 1 ppm on day-old Vencobb broiler chicks for four weeks period and reported depression in serum albumin even in 0.25 ppm aflatoxin fed group. The author also found that the albumin-globulin ratio was widened with increasing level of toxin in the feed.

Arulmozhi *et al.* (2000) reported dose-dependant decrease in the serum albumin throughout the experiment when diets contaminated with aflatoxin B1 (0, 20, 40, 60, 80 and 100 ppb) were fed to the broiler chicken for a period of 45 days. The decrease was significant in all the aflatoxin B1 treated groups on 45^{th} day of the experiment.

Rosa *et al.* (2001) reported significant decrease ($P<0.05$) in serum albumin level in broilers fed aflatoxin B1 (5 mg/kg) from 30 to 52 days of age.

Chaturvedi and Singh (2002) found significantly ($P<0.05$) lower serum albumin (35.7 %) than control in commercial broiler chickens fed a diet containing 3 ppm aflatoxin for a period of 21 days.

Shivachandra *et al.* (2003b) observed lowered level of serum albumin in broiler chicks fed 1 ppm aflatoxin from seventh day to seventh week of age.

Basmacioglu *et al.* (2005) found that the serum albumin level was significantly decreased in broilers fed aflatoxin (2 mg/kg) for a period of 21 days.

2.1.5.3 Glucose

Maurice *et al.* (1982) reported that the plasma glucose level was significantly elevated in broiler chickens dosed with 650 ppb dietary aflatoxin from day-old to three weeks of age.

In contrast, significant decrease in blood glucose was observed by Raina *et al.* (1991) at 0.1 ppm aflatoxin in all age groups between seven and 49 days.

Similar reduction in blood glucose was also observed by Kumar (1995) in aflatoxin (1 ppm) treated broilers at four weeks of age.

Churchil (1996) reported significant reduction in serum glucose level in broiler chicken treated with aflatoxin (1 ppm) for a period of eight weeks.

According to Mani (1995), the serum glucose level was increased during aflatoxin (0.5 ppm) feeding for a period of eight weeks.

According to Mani *et al.* (2000a), the serum glucose level was elevated in broiler chickens dosed with 0.2 ppm dietary aflatoxin B1 from day old to eight weeks of age.

Mani *et al.* (2000b) studied the effect of various levels of dietary aflatoxin B1 (0, 100, 200, 300, 400 and 500 ppb) on the blood constituents in commercial broilers for eight weeks period and found that the increase in serum glucose level was directly proportional to aflatoxin level in the feed. The highest serum glucose level was observed in 500 ppb toxin fed groups.

Whereas, in contrast to the above finding, lower serum glucose level was reported in aflatoxin (3 ppm) fed commercial broiler chickens treated for 21 days by Chaturvedi and Singh (2002).

By feeding 2 mg total aflatoxin per kg feed for a period of 21 days, Basmacioglu *et al.* (2005) observed significant decrease in serum glucose level in broilers.

2.1.5.4 Cholesterol

At 2.5 ppm level, aflatoxin had no significant effect on plasma cholesterol when fed from three to six weeks of age; whereas, at 5 ppm level, significant reduction was recorded in all age groups. Further, the effect of aflatoxin on plasma cholesterol diminished as age advanced (Lanza *et al.*1980).

Maurice *et al.* (1982) reported that the plasma cholesterol level was significantly elevated in broiler chickens receiving 650 ppb aflatoxin from day old to three weeks of age.

Raina *et al.* (1991), Huff *et al.* (1992), Kumar (1995) and Nath *et al.* (1995) recorded significant reduction in serum cholesterol level in broiler chickens at dietary levels of 0.1, 3.5, 1 and 2 ppm aflatoxin respectively.

Mani *et al.* (1993) reported that the feed containing 0.75 and 1.50 mg aflatoxin B1 per kg caused significant reduction in cholesterol level at eight weeks of age.

A dose-related reduction in serum cholesterol was observed by Mani (1995) in eight weeks old broiler chicken fed graded levels (0.1, 0.2, 0.3, 0.4 and 0.5 ppm) of aflatoxin.

Churchil (1996) reported significant reduction in serum cholesterol level in broiler birds fed 1 ppm aflatoxin for a period of eight weeks.

Vasan *et al.* (1998) reported dose-related reduction in serum cholesterol in Vencobb broiler chicks fed aflatoxin B1 (0, 0.25, 0.5, and 1 ppm) for a period of four weeks.

Mani *et al.* (2000a) reported that serum cholesterol level was lowered in broiler chicken receiving 0.2 ppm dietary aflatoxin B1 from day old to eight weeks of age.

Mani *et al.* (2000b) studied the effect of different levels of dietary aflatoxin B1 (0, 100, 200, 300, 400 and 500 ppb) on the blood constituents in commercial broilers and found significant ($P<0.01$) decrease in the serum cholesterol level in aflatoxin B1 treated birds than control. Further, they noticed that the serum cholesterol level was negatively associated with the dose of aflatoxin B1 in the feed.

According to Raju and Devegowda (2000), the serum cholesterol concentration was decreased by 31·7 per cent in a group of broiler chicks fed aflatoxin (0.3 mg/kg) for a period of 35 days.

In a study on broilers for a period of 21 days, Basmacioglu *et al.* (2005) reported significant decrease in serum cholesterol level at 2 mg total aflatoxin per kg diet.

2.1.6 Relative organ weight and carcass yield

According to Carnaghan *et al.* (1966), the liver-body weight ratio was increased when Rhode Island Red chicks were fed from hatching to eight weeks of age, a commercial ration to which a highly toxic groundnut meal was added.

During aflatoxicosis, enlargement of liver and spleen and regression of bursa of Fabricius were noticed by Smith and Hamilton (1970).

Chen *et al.* (1985) observed enlargement of kidney, liver and heart when broiler chickens were fed a diet containing 2057 and 1323 μg of aflatoxin B1 and B2 respectively per kg of feed.

In contrast to the above reports, Shen *et al.* (1987) observed that the liver-body weight ratio in broilers was unchanged by dietary aflatoxin (25, 50, 100, 200 and 400 μg per kg).

Huff *et al.* (1992) reported increased relative weights of the liver, kidney, proventriculus and heart of chicken, which received a diet consisting 3.5 μg per g of aflatoxin.

Similarly, Stanley *et al.* (1993) observed significant increase in relative weights of liver, heart and proventriculus with the addition of 5 ppm aflatoxin in a broiler diet.

Churchil (1996) reported significantly ($P<0.05$) lower eviscerated carcass yield and higher relative weights of liver and kidney in broiler chicken treated with aflatoxin (1 ppm) for a period of eight weeks.

Significant increase in the weight of liver and gizzard was noticed by Devegowda *et al.* (1998) by feeding 400 ppb aflatoxin to broiler chickens for a period of six weeks.

Mani *et al.* (2000a) reported enlargement of liver, atrophy of bursa and reduced eviscerated yields due to aflatoxicosis (0.2 ppm) in eight weeks old broiler chicken.

Raju and Devegowda (2000) found increased weights of liver and kidney (17.8 and 26·5 per cent respectively) in broiler chicks fed aflatoxin (0.3 mg/kg) for a period of 35 days.

Ortatatli and Oguz (2001) reported increase in the weights of liver and kidney and reduction in the sizes of thymus and bursa of Fabricius at the end of three weeks in aflatoxin (2 mg total aflatoxin/kg) fed group.

According to Rosa *et al.* (2001), the relative weights of liver, kidney, spleen and pancreas were increased in broilers fed 5 mg aflatoxin B1 per kg from 30 to 52 days of age.

Mani *et al.* (2001) reported reduced carcass yield (61.09 to 62.40 %) in the aflatoxin B1 (200 ppb) fed group than control group (63.58 to 64.51%) at eight weeks of age.

Chaturvedi and Singh (2002) found that the weight of liver of broilers on a diet containing three ppm aflatoxin was significantly higher (21.91%) than that of broilers fed with control diet.

Raju and Devegowda (2002) reported significant reduction in the size of bursa in commercial broilers fed a diet containing 300 ppb aflatoxin from day-old to five weeks of age; however the spleen weight remained unaffected by aflatoxin.

Shivachandra *et al.* (2003a) studied the effect of aflatoxin (1 ppm) on broiler chicks from seventh day to seventh week of age and reported enlargement of liver, spleen and kidney and progressive atrophy of bursa and thymus up to 42 days post feeding.

Girish and Devegowda (2006) reported enlargement of liver (21.7 %), spleen (51 %), kidney (26.4 %) and gizzard (16.8 %), and significant depression in the weights of thymus (23.3 %) and bursa of Fabricius (30 %) in broilers at thirty five days of age fed a diet containing 2 mg aflatoxin per kg of feed.

2.1.7 Livability

Smith *et al.* (1971) observed that the broiler chicks fed a contaminated diet with 10 ppm aflatoxin for four weeks period caused death of about one fourth of the stock.

Reddy *et al.* (1982) observed that the livability was adversely affected in commercial broiler chicks fed aflatoxin (1 ppm) contaminated diet.

On the contrary, Giambrone *et al.* (1985) fed broiler chicks with diets containing aflatoxin B1 at the levels of 100, 200, 400 or 800 µg per kg from two to five weeks of age and reported neither morbidity nor mortality.

Similarly, Chattopadhyay *et al.* (1985) also recorded no mortality attributable to aflatoxin during zero to ten weeks in broiler chickens fed commercial based rations contaminated with aflatoxin (0.5 and 2.5 ppm).

Mani *et al.* (1992) observed a dose related mortality fashion in commercial broiler chicks when fed from 0 to 2 ppm aflatoxin B1 from zero to eight weeks of age.

Churchil (1996) reported higher mortality rate (51.67 per cent) in broiler chicken treated with dietary aflatoxin (1 ppm) from zero to eight weeks of age.

Prabaharan and George (1999) recorded a mortality rate of four per cent in aflatoxin (1 ppm) fed broiler chicks from day old to 32 days of age.

Mani *et al.* (2000a) studied the effect of aflatoxin (0.2 ppm) on three different commercial broiler strains for a period of eight weeks and reported 20 per cent mortality in strain one and three and 6.7 per cent mortality in strains two and three respectively.

Shivachandra *et al.* (2003a) studied the effect of aflatoxin (1 ppm) on broiler chicks from seventh day to seventh week of age and reported 3.33 per cent mortality in the aflatoxin fed group.

2.1.8 Gross pathology

Carnaghan *et al.* (1966) reported that in first three weeks the liver of Rhode Island Red chicks when fed a commercial ration to which 15 per cent of highly toxic groundnut meal was added with aflatoxin B1 content of approximately 10 ppm, were enlarged and putty colored with a reticulated network showing Moroccan leather appearance. The consistency was soft and petechial haemorrhages were frequently present. Thereafter there was an apparent reduction in size of the organ with increasing firmness of texture which was observed until seventh week when well defined, raised, nodular areas were seen on the surface. Diffuse white pin head sized foci were visible from the sixth week. Throughout the experimental period the liver of birds fed aflatoxin remained pale and yellow ochre coloured with petechial haemorrhage.

Balachandran (1983) in his study revealed that the liver from birds fed 1 ppm aflatoxin were enlarged, pale or discolored with Moroccan leather appearance in the first two weeks and yellow colored in the next two weeks. The kidneys were enlarged, pale and congested with few petechial haemorrhage at 1 ppm and were marked at 3 ppm level. Thigh and leg musculatures revealed petechial haemorrhages both at 1 and 3 ppm levels of aflatoxin. Spleen showed slight enlargement at 1 ppm and was pronounced at 3 ppm aflatoxin.

At slaughter Chen *et al.* (1985) noticed haemorrhagic spots on the surface of some of the liver while most other liver were pale coloured. Distended gall bladder and the presence of haemorrhagic spots on the muscle surfaces, especially on the breast were also evident in considerable number of aflatoxin fed birds.

Kumar (1995) fed broilers with 1 ppm aflatoxin and noticed gross changes like enlargement and yellowish discolouration of liver and enlargements of kidneys.

Bakshi *et al.* (1995) recorded hepatomegaly with petechial haemmorhages and spleenomegaly when broiler chickens were fed aflatoxin at 0.75, 1.5 and 3 ppm levels for a period of six weeks.

Rosa *et al.* (2001) studied the effect of aflatoxin B1 (5 mg/kg) from 30 to 52 days of age in male broiler chickens and found that the livers of broilers fed diets containing aflatoxin were friable and pale in appearance.

Shivachandra *et al.* (2003a) studied the gross lesions of aflatoxin (1 ppm) on 15 days post feeding in broiler chicken. The livers were fatty, friable, pale and enlarged with a few haemorrhagic foci on the surface. The authors also observed swollen and congested kidneys, spleenomegaly and progressive atrophy of bursa and thymus.

Karaman *et al.* (2005) reported that the liver and kidney of 21 days old broiler chicks from aflatoxin (2mg/kg) treated group were swollen and pale yellowish red. The spleen were enlarged and congested and bursa of Fabricius had no visible morphological changes.

2.1.9 Histopathology

San-Gabriel (1971) recorded diffuse fatty degeneration, nodular hyperplasia, discrete hyperplasia of portal spaces, occlusion of hepatic sinusoids and enlarged nuclei in the liver of broiler chicken fed aflatoxin at 450 ppb in the feed.

Mohiuddin *et al.* (1986) reported periportal fatty changes and necrosis, loss of normal hepatic architecture and an increased kupffer cell reaction in chicken maintained on a feed containing 20000 ppb of aflatoxin.

Hepatic lesions reported by Espada *et al.* (1992) in chicken dosed with 0.2 and 3 μg of aflatoxin per gram body weight had vacuolations of hepatocytes due to fatty metamorphosis and bile duct proliferation.

Mundas and Rao (2001) studied the histopathological changes due to aflatoxin (0.5 ppm) feeding on liver of broiler chicken from day ten to the end of eighth week. From fifth week onwards they found hyperplasia of bile duct epithelium, increase in number of bile ducts and individualization of hepatocytes and by eighth week, in addition to these changes periportal infiltration of mononuclear cells, vacuolated appearance of hepatocytes and condensed chromatin material in nucleus were observed in the liver of aflatoxin treated birds.

Ortatatli and Oguz (2001) found that liver of broiler chickens fed aflatoxin diet for a period of 21 days had moderate to severe fatty changes. In some cases the

liver showed hepatocellular regeneration, periportal fibrosis, bile duct hyperplasia, nodular lymphoid cell accumulation and occasional heterophil and mononuclear cell infiltrations or both in portal areas.

Rosa *et al.* (2001) studied the effect of aflatoxin B1 (5 mg/kg) on liver of male broilers from 30 to 52 days of age by histopathology and noticed moderate to severe diffuse hepatic vacuolization characterized by severe and diffuse hepatic steastosis in liver of aflatoxin fed group.

The microscopic changes like congestion, haemorrhage with varying degrees of degenerative changes in hepatocytes, necrosis and bile duct proliferation were noticed on ten days post feeding in the liver of aflatoxin fed (600 ppb) broiler chicks by Gupta and Singh (2003).

On microscopic examination, Shivachandra *et al.* (2003a) reported mild degenerative changes in hepatocytes at 3 days post feeding and diffuse areas of fatty changes up to 42 days post feeding in liver of broiler chicken fed aflatoxin at 1 ppm level.

Karaman *et al.* (2005) found that the liver of broiler chicks fed an aflatoxin-containing diet (2 mg/kg) for 21 days revealed moderate to severe hydropic degeneration and fatty changes in hepatocytes, bile duct proliferation and periportal fibrosis in the portal areas and accumulation of nodular lymphoid cell within the hepatic lobules.

2.1.10 Economics

Arulmozhi (1999) studied the effect of six different levels of aflatoxin (0, 20, 40, 60, 80, 100 ppb) on performance of broilers from zero to 45 days and reported that net returns per bird from group one to six were Rupees 16.82, 15.58,

13.33, 13.63, 5.25 and 3.24 respectively. The economic loss in profit compared to control in group two to six were 7.3, 20.7, 18.9, 68.8 and 80.7 per cent respectively. The economic loss was highest at 100 ppb level and minimum at 20 ppb level.

2.2 EFFECT OF ESTERIFIED GLUCOMANNAN

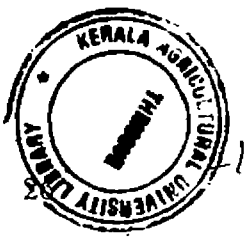
Esterified Glucomannan (E-GM) is a new generation mycotoxin neutralizing agent, evolved out of modern biotechnological innovations. Its effect on alleviation of toxic effects of mycotoxins has been successfully tested by various workers during last one decade (Khajarern and Khajarern, 1999; Raju and Devegowda, 2000; Basmacioglu *et al.*, 20005; Karaman *et al.*, 2005). However, its effects on growth and other production parameters in the absence of mycotoxin have not been investigated thoroughly; hence the scientific information on this aspect is scarce. The very little information available in the literature on the effect of E-GM on growth rate, feed intake and feed efficiency in aflatoxin free diet is compiled below.

2.2.1 Growth Rate

Pavicic and Nemanic (2001) reported that the supplementation of esterified glucomannan (1 g/kg) to basal diet up to 42 days in broiler chickens had increased the body weight by six per cent and live weight gain by seven per cent when compared to control.

Aravind *et al.* (2003) found that the broiler chickens fed esterified glucomannan (0.05 %) performed significantly better (3.48 %) than those on the basal diet alone during his study for 35 days.

Karaman *et al.* (2005) reported that the addition of yeast glucomannan (both 0.5 and 1 g/kg) to the basal diet did not produce any significant ($P<0.05$) changes in growth rate compared to the control during his 21 days study.



Basmacioglu *et al.* (2005) found that the addition of esterified glucomannan (both 0.5 and 1 g/kg) to the basal diet did not produce any significant effect on growth of broilers at 21 days of age, when compared to the control.

2.2.2 Feed Intake

Rizzi *et al.* (2003) studied the effect of esterified glucomannan (0.11 %) in Warren hens for a period of 28 days and reported non significant effect of esterified glucomannan on feed intake compared to control.

2.2.3 Feed Efficiency

Aravind *et al.* (2003) reported that the supplementation of esterified glucomannnan at 0.05 per cent to basal diet had resulted in improvement of feed conversion ratio in 35 days old broilers than control.

2.3 EFFECT OF ESTERIFIED GLUCOMANNAN ON AFLATOXICOSIS

The beneficial effects of *Saccharomyces cerevisiae* in alleviating the toxic effects of aflatoxin have been confirmed by earlier workers (Stanley *et al.*, 1993; Churchil, 1996). Mannan from the glucan-rich inner cell wall of this yeast is thought to be the prime component possessing anti-mycotoxin properties. Esterified Glucomannan (E-GM) had shown considerable binding ability with several commonly occurring mycotoxins (Devegowda *et al.*, 1998). Yeast glucomannan also showed markedly high binding ability *in vitro* (75 to 90 %) and *in vivo* with aflatoxin (Murthy and Devegowda, 2004), and it has been preferred for detoxification of aflatoxin in poultry species. A few studies confirmed the ability of yeast glucomannan in reversing the adverse effects of aflatoxin on performance, serum biochemistry, haematology and immune responses of birds (Raju and

Devegowda, 2000; Arvind *et al.,* 2003; Girish and Devegowda, 2006). The literature on the aflatoxin counteracting effects of Esterified Glucomannan in broiler chicken is reviewed hereunder.

2.3.1 Body weight and weight gain

Khajarern and Khajarern (1999) reported that esterified glucomannan at 1 g per kg level for a period of six weeks was more effective than 0.5 g per kg level on growth performance of broilers fed diets containing aflatoxin (0.1 to 0.3 mg/kg).

Raju and Devegowda (2000) found that the supplementation of esterified-glucomannan (1 g/kg) to the aflatoxin (0.3 mg/kg) contaminated diet increased body weight (2·26 %) in broiler chickens at 35 days of age indicating its possible beneficial effect on aflatoxicosis. The improvement in body weight was significant from second week onwards.

Aravind *et al.* (2003) found that the addition of esterified glucomannan (0.05 %) significantly countered (8.84 %) the growth depressing effect of aflatoxin-contaminated diet (168 ppb) in 35 days old broiler chicken.

Basmacioglu *et al.* (2005) found that the addition of esterified glucomannan (1 g/kg) to an aflatoxin containing diet (2 mg total aflatoxin/kg) partially countered the adverse effect of aflatoxin on body weight gain in broilers of 21 days of age. The ameliorating effect was greater at 1 g per kg than 0.5 g per kg level.

Karaman *et al.* (2005) reported that the addition of 1 g per kg yeast glucomannan to the feeds contaminated with aflatoxin (2 mg/kg) can reduce the toxicity of aflatoxin in whole body of broilers of 21 days age. The higher concentration of yeast glucomannan (1 g/kg) was more effective than the lower

concentration (0.5 g/kg) in counteracting the adverse effect of aflatoxin on broiler chicken.

Girish and Devegowda (2006) reported that the addition of a glucomannan containing yeast product (Mycosorb®) at the level of 1 kg per ton to the aflatoxin contaminated diet (2 mg/kg) significantly alleviated the growth depression due to aflatoxin and significantly improved the fifth week body weight by 6.6 per cent than aflatoxin alone fed group.

2.3.2 Feed Intake

Raju and Devegowda (2000) noticed that the feed intake was improved in aflatoxin (0.3 mg/kg) plus esterified glucomannan (1 g/kg) supplemented group compared to aflatoxin alone fed group of broiler chickens at 35 days of age indicating its possible beneficial effect on aflatoxicosis.

Aravind *et al.* (2003) found that the supplementation of esterified glucomannan (0.05%) to a mycotoxin contaminated diet (aflatoxin 168 ppb, ochratoxin 8.4 ppb, zearalenone 54 ppb and T-2 toxin 32 ppb) effectively improved feed intake in 35 days old broilers.

Basmacioglu *et al.* (2005) found that the addition of esterified glucomannan (1 g/kg) to an aflatoxin containing diet (2 ppm) partially alleviated the adverse effect of aflatoxin on feed consumption in broiler chicken at 21 days of age.

Girish and Devegowda (2006) reported that the supplementation of a glucomannan containing yeast product (Mycosorb®) at the level of 1 kg per ton in aflatoxin (2 mg/kg) contaminated feed for a period of 35 days significantly improved feed consumption of broilers.

2.3.3 Feed Efficiency

According to Raju and Devegowda (2000), the feed efficiency remained unaffected in aflatoxin (0.3 mg/kg) plus esterified glucomanan (1 g/kg) supplemented group of broiler chicken compared to aflatoxin alone fed group at 35 days of age.

Aravind *et al.* (2003) found that the supplementation of esterified glucomannan (0.05%) to a mycotoxin contaminated diet (aflatoxin 168 ppb, ochratoxin 8.4 ppb, zearalenone 54 ppb and T-2 toxin 32 ppb) effectively improved feed efficiency in 35 days old broilers.

The adverse effect of aflatoxin (2 mg total aflatoxin/kg) on feed efficiency of broiler chicks was partially alleviated by the addition of esterified glucomannan (1 g/kg) for a period of 21 days (Basmacioglu *et al.* 2005).

In a study on broilers for a period of 35 days, Girish and Devegowda (2006) observed that the toxic effects of aflatoxin (2 mg/ kg) on feed efficiency were reversed with the supplementation of a glucomannan containing yeast product (Mycosorb®) at the rate of 1 kg per ton of feed.

2.3.4 Serum Biochemistry

Khajarern and Khajarern (1999) reported significant improvements in serum biochemical parameters by the addition of esterified glucomannan (1 g/kg). They also found that the supplementation of esterified glucomannan at 1 g per kg level was more effective than 0.5 g per kg level on biochemistry of broilers fed a diet containing aflatoxin (0.1 to 0.3 mg/kg) for a period of six weeks.

Raju and Devegowda (2000) found that the supplementation of esterified-glucomannan decreased serum protein, cholesterol, BUN and blood haemoglobin contents at 35th day, indicating its possible beneficial effect on mycotoxicosis (aflatoxin 0.3 mg/kg, ochratoxin 2 mg/kg and T-2 toxin 3 mg/kg) in broiler chickens.

Aravind *et al.* (2003) found that the supplementation of esterified glucomannan (0.05%) to a mycotoxin contaminated diet (aflatoxin 168 ppb, ochratoxin 8.4 ppb, zearalenone 54 ppb and T-2 toxin 32 ppb) effectively improved serum parameters in broilers of 35 days old.

Basmacioglu *et al.* (2005) found that the addition of esterified glucomannan (1g/kg) to an aflatoxin (2 mg total aflatoxin/kg) containing diet between one and 21 days of age significantly counteracted the adverse effect of aflatoxin on serum protein, cholesterol, albumin and glucose levels in broilers.

2.3.5 Relative organ weight and carcass yield

Raju and Devegowda (2000) found that the supplementation of esterified-glucomannan decreased the weights of liver and adrenals indicating its possible beneficial effect on mycotoxicosis (aflatoxin 0.3 mg/kg, ochratoxin 2 mg/kg and T-2 toxin 3 mg/kg) in broiler chickens of 35 days old.

Raju and Devegowda (2002) found that the addition of esterified glucomannan (0.1%) in a diet containing aflatoxin 300 ppb, ochratoxin 2 ppm and T-2 toxin 3 ppm significantly improved the weights of bursa of Fabricius and thymus in five weeks old broilers.

Aravind *et al.* (2003) reported that esterified glucomannan supplementation (0.05%) to a mycotoxin contaminated diet (aflatoxin 168 ppb, ochratoxin 8.4 ppb, zearalenone 54 ppb and T-2 toxin 32 ppb) did not significantly diminish the effects of toxins on the relative weight of liver and gizzard at 35 days of age in broilers.

Girish and Devegowda (2006) reported that the toxic effects of aflatoxin (2 mg/kg) such as increased weights of liver, spleen, kidney, gizzard, thymus and bursa of Fabricius in broilers of 35 days old were alleviated with the supplementation of 1 kg per ton of a glucomannan containing yeast product (Mycosorb®).

2.3.6 Livability

Basmacioglu *et al.* (2005) reported that mortality was not statistically significant between aflatoxin (2 mg/kg) alone, esterified glucomannan alone (0.5 g/kg), aflatoxin (2mg/kg) plus esterified glucomannan (0.5 g/kg), esterified glucomannan alone (1 g/kg) and aflatoxin (2mg/kg) plus esterified glucomannan (1 g/kg) groups in an experiment with broilers up to 21 days of age.

2.3.7 Gross Pathology

Karaman *et al.* (2005) reported reduction in the severity of macroscopic lesions in livers of 21 days old broilers and found that the number of affected livers was only three and one in the aflatoxin (2 mg/kg) plus esterified glucomannan (0.5 g/kg) and aflatoxin (2 mg/kg) plus esterified glucomannan (1 g/kg) groups respectively compared to aflatoxin alone treated group in which the number of affected livers was seven.

2.3. 8 Histopathology of Liver

Karaman *et al.* (2005) reported that the supplementation of yeast glucomannan (0.5 g/kg) in a aflatoxin (2 mg/kg) contaminated diet showed slight improvement on the histopathological changes in the liver of broiler chicks of 21 days old when compared to that of aflatoxin alone treated group. The authors also found that the number of chicks displaying histopathological changes in the liver was significantly decreased in aflatoxin plus yeast glucomannan (1 g/kg) compared to aflatoxin alone fed group.

Materials and Methods

3. MATERIALS AND METHODS

An experiment was conducted in the Department of Poultry Science, College of Veterinary and Animal Sciences, Mannuthy to study the effect of Esterified Glucomannan (E-GM) on alleviation of aflatoxicosis in broiler chicken. The study was conducted for a period of six weeks during December 2006 and January 2007.

3.1 PRODUCTION OF AFLATOXIN

3.1.1 Maintenance of fungi

*Aspergillus parasiticus var.globosus**411 obtained from Microbial Type Culture Collection and Gene Bank, Institute of Microbial Technology (IMTEC), Chandigarh, India was used for the production of aflatoxin. The fungus was maintained by sub culturing it on potato dextrose agar at 10 days interval (Shotwell *et al.*, 1966).

3.1.2 Production of fungal rice culture

Aflatoxin B1 was produced in rice (Shotwell *et al.*, 1966). Fifty grams of rice, free from any adulteration, was taken in 250 ml conical flask, plugged with cotton, autoclaved at 50 lb psi for 15 min (so as to reach half cooked form) and cooled. Eight to ten ml distilled water was added into each flask and were shaken for uniform distribution of water. Inoculation was done by transferring some fresh spores of *A. parasiticus var.globosus**411 into the individual flask with the help of platinum loop under sterile conditions. The flasks were kept at room temperature and hand shaken vigorously for six to ten times a day to avoid clumping and facilitate easy growth of the fungus. The mold growth was confirmed by the

appearance of whitish growth on rice; gradually changing to greenish colour showing extensive mycelial growth at the end of the incubation period. After 10 days post-inoculation the contaminated rice was again autoclaved. The autoclaved culture rice was dried, ground to powder form and used in the experimental rations.

3.2 ESTIMATION OF AFLATOXIN B1

3.2.1 Standard aflatoxin B1

Pure aflatoxin B1 obtained from M/s Sigma chemicals, USA, was used as standard for the estimation of aflatoxin B1 in fungal rice culture as well as in experimental diets.

3.2.2 Mouldy rice culture

Quantitative measurement of aflatoxin B1 in the mouldy rice was done as per Romer (1975). Slurry was prepared by blending 50 g of mouldy rice powder with 250 ml of water for three minutes. The extraction was done by blending 150 g of slurry with 250 ml of acetone for two minutes and filtering through fluted Whatman No.1 filter paper. The extract was purified by taking 150 ml of the extract in 500 ml conical flask and swirling it after adding 3 g of cupric carbonate. In another 500 ml conical flask ferric gel was prepared by adding 30 ml of ferric chloride solution (0.41M) to 170 ml of sodium hydroxide (0.2M) and swirling. The ferric gel was immediately transferred to the flask containing the extract and the contents were mixed thoroughly and allowed to stand for 2 minutes with occasional swirling. This mixture was filtered through Whatman No.1 filter paper and 100 ml of the filtrate was mixed with 100 ml of sulphuric acid (0.03%) in 500 ml separating funnel and the extraction was done thrice using 20 ml, 20 ml and 10 ml of chloroform; each time collecting the lower chloroform layer. The combined

of chloroform; each time collecting the lower chloroform layer. The combined extract was transferred to a 250 ml separating funnel containing 100 ml of potassium hydroxide (0.02M) per potassium chloride (1 percent) mixture and gently swirled for 10 seconds. The lower chloroform layer was collected in a 100 ml beaker through a funnel containing a bed of anhydrous sodium sulphate. The final extract was evaporated to near dryness in a water bath at 40^0C and transferred to a vial and dried completely. Known quantity of chloroform was added to dissolve the extract and taken to Thin Layer Chromatography (TLC) for quantification. The TLC plates were prepared with 0.25 mm thickness by using Silica gel G - Water (2:1) slurry and activated at 110^0C for an hour. The samples and standards were spotted on the TLC plates and developed in Chloroform - Acetone (90:10) mixture for 45 minutes. The developed plates were compared under long wave UV lamp in a chromatoview cabinet. The aflatoxin (B1) content was calculated according to AOAC (1990) specifications.

3.2.3 Screening of feed ingredients for Aflatoxin

Feed ingredients (maize, wheat bran, soyabean meal, unsalted dried fish and gingelly oil cake) used for the preparation of basal diet were screened for the presence of aflatoxins in the same method as described by Romer (1975). The feed ingredients free of aflatoxins were only used for the preparation of experimental diets.

3.3 PREPARATION OF EXPERIMENTAL DIETS

The ingredient composition of basal starter and finisher rations is presented in Table 1. The proximate analysis of the feed ingredients and rations was carried out according to the procedure described by AOAC (1990). The chemical composition of the starter and finisher rations are shown in Table 2.

Table 1. Per cent ingredient composition of starter and finisher experimental rations

Ingredients	Inclusion level (%)	
	Starter (0-4 weeks)	Finisher (5-6weeks)
Maize	55.00	62.00
Wheat bran	3.00	3.00
Soyabean meal	26.00	21.00
Gingelly oil cake	4.00	-
Unsalted dried fish	10.00	12.00
Mineral mixture[1]	1.75	1.75
Salt	0.25	0.25
Total	100.00	100.00
Feed Supplements (g per 100 kg feed)		
Vitamin mixture[2]	10	10
DL Methionine	100	50
Lysine hydrochloride	140	100
Choline chloride[3]	120	120
Coccidiostat[4]	50	50
B-Complex powder[5]	7.5	7.5

Note:

[1] Mineral mixture composition: Calcium 32%, Phosphorus 6%, Magnesium 1000 ppm, Cobalt 60 ppm, Zinc 2600 ppm, Iron 0.1%, Iodine 100 ppm, Copper 100 ppm and Manganese 2700 ppm.

[2] Vitamin mixtures: (Nicomix A, B_2, D_3, K powder® from Nicholas Primal India Ltd., Mumbai). Each gram containing Vit. A 82,500 IU, Vit. B_2 50 mg, Vit. D_3 1200 IU and Vit. K 10 mg.

[3] Choline Chloride: (Anichol® from Jubilant Organosys Ltd., Gujrat) containing Choline chloride 50% dry, Corncob & Rice husk powder 40%.

[4] Coccidiostat: (Wocox® from Wockhardt Ltd., Wockhardt Towers, Bandra Kurla Complex, Mumbai-400 051) containing Diclazuril 0.5% w/w.

[5] B-Complex powder: (Meriplex FDS® from Wockhardt Ltd., Wockhardt Towers, Bandra Kurla Complex, Mumbai-400 051). Each gram containing Vit.B_1 8 mg, Vit.B_6 16 mg, Vit.B_{12} 80 mg, Vit.E 80 mg, Niacin 120 mg, Folic acid 8 mg and Pantothenate 80 mg.

Table 2. Per cent chemical composition of starter and finisher experimental rations (On dry matter basis)

Sl. No.	Nutrients	Starter Ration	Finisher Ration
1.	Dry matter	88.33	88.07
2.	Crude protein	23.17	20.16
3.	Ether extract	3.05	3.78
4.	Crude fibre	4.37	3.78
5.	NFE	60.77	63.26
6.	Total ash	8.64	9.02
7.	Acid insoluble ash	3.04	3.96
Calculated values			
1.	ME Kcal/Kg	2801	2909
2.	Lysine	1.45	1.3
3.	Methionine	0.51	0.44
4.	Calcium	1.27	1.22
5.	Total Phosphorus	0.54	0.58

The basal rations were made isocaloric and isonitrogenous as given in Table 2. The mouldy rice containing known quantity of aflatoxin B1 and an E-GM containing commercial product (Mycosorb® Alltech inc., Bangalore) were incorporated either alone or in combination, so as to prepare the following experimental diets.

Treatment	Number of replications	Number of birds in each replicate	Type of diet
T1	5	10	Control diet
T2	5	10	Control diet + 1.0 ppm Aflatoxin
T3	5	10	Control diet + 0.1per cent E-GM
T4	5	10	Control diet + 1.0 ppm Aflatoxin + 0.1 per cent E-GM

3.4 BIOLOGICAL TRIAL

Two hundred, day-old straight run commercial broiler chicks (Vencobb) procured from Venkateshwara Hatcheries Ltd., Palakkad, Kerala formed the experimental subjects. The chicks were wing banded and weighed individually. The chicks were allotted randomly to four treatment groups with five replicates each containing ten birds.

The chicks were reared under deep litter system of management. The experimental shed was cleaned and disinfected one week prior to the commencement of the experiment. Litter material was spread to a thickness of 6 cm in each pen. A floor space of 925 sq cm per chick was allotted. Feeders, waterers and other equipment were cleaned, disinfected and sun dried before use.

The chicks were brooded till they attained four weeks of age. Thereafter, light was provided only during night hours to enhance feed intake. Standard managemental procedures were followed during the course of experiment.

All the chicks were vaccinated as per the following vaccination schedule

Age	Vaccine	Route of Administration
5 days	Ranikhet disease (F1)	Eye drops
14 days	IBD intermediate (Live)	Drinking water
24 days	IBD intermediate (Live)	Drinking water
28 days	Ranikhet Disease (LaSota)	Drinking water

The birds were provided with respective treatment feed and water *ad libitum* throughout the experimental period. Broiler starter diet was fed up to four weeks of age and then switched over to broiler finisher diet till the end of six weeks of age.

Both the diets were formulated as per BIS (1992) specification of nutrients for broiler chicken.

3.5 METEOROLOGICAL PARAMETERS

The wet and dry bulb thermometer readings were taken at forenoon (8.0 am) and afternoon (2.0 pm) daily. The maximum and minimum temperatures were recorded at forenoon on all days throughout the experimental period. The weekly mean for maximum and minimum temperatures, dry bulb temperatures and relative humidity calculated from this data are given in Table 3.

3.6 BODY WEIGHT AND WEIGHT GAIN

The body weight of individual bird was recorded at weekly interval and the average weekly body weight and weight gain per bird were calculated for various treatment groups.

3.7 FEED INTAKE

Feed intake of birds was recorded for each replicate at weekly interval. From this data the average weekly feed intake was calculated for various treatment groups.

3.8 FEED EFFICIENCY

Feed efficiency (kg of feed per kg weight gain) was calculated for each replicate based on the data on body weight gain and feed intake.

3.9 SERUM PARAMETERS

At six weeks of age, blood samples of two birds from each replicate were collected from the jugular vein in a clean dry labeled glass tube and were kept in slanted position at room temperature to facilitate the separation of serum. The parameters like total protein, albumin, glucose and cholesterol were estimated from serum using following protocols.

The serum total protein was estimated colorimetrically by Biuret method utilizing the kit supplied by Beacon Diagnostics Pvt. Ltd., 424, New GIDC, Kabilpore, Navasari – 396 424, India.

The serum albumin was estimated by Bromocresol green method utilizing the kit supplied by Agappe Diagnostics Pvt. Ltd., Agappe hills, Dist. Ernakulam, Kerala 683562, India.

The serum glucose was estimated by GOD-PAP method utilizing the kit supplied by Agappe Diagnostics Pvt. Ltd., Agappe hills, Dist. Ernakulam, Kerala 683562, India.

The serum total cholesterol was estimated by CHOD-PAP method utilizing the kit supplied by Agappe Diagnostics Pvt. Ltd., Agappe hills, Dist. Ernakulam, Kerala 683562, India.

3.10 PROCESSING YIELDS AND LOSSES

Two birds (1 male and 1 female) randomly selected from each replicate were sacrificed at the end of the experiment to study carcass characteristics like

dressing percentage, processing yields and losses and the relative weights (g per 100 g body weight) of liver, spleen, kidney and bursa of Fabricius.

3.11 LIVABILITY

The mortality of birds from different treatment groups was recorded and the livability was worked out. The dead birds were subjected to post mortem examination and the lesions were recorded.

3.12 GROSS LESION

During the evisceration stage of slaughter, organs like liver, spleen, kidney and bursa of Fabricius were examined from two birds of each replicate of treatment to study the gross lesions.

3.13 HISTOPATHOLOGY OF LIVER

At the time of slaughter the liver samples from two birds of each treatment were collected in 10 per cent neutral buffered formalin for the histopathology.

3.14 ECONOMICS

Cost of feed for different dietary treatments was calculated based on the cost of ingredients and Mycosorb®. Cost of feed per kg live weight for different dietary treatments was calculated based on body weight attained and recurring expenditure at six weeks of age.

3.15 STATISTICAL ANALYSIS

Data collected on various parameters was statistically analyzed as per the methods described by Snedecor and Cochran (1994).

Results

4. RESULTS

The results of the experiment conducted to study the effect of Esterified Glucomannan (E-GM) on alleviation of aflatoxicosis in broiler chicken are presented in this chapter.

4.1 METEOROLOGICAL PARAMETERS

The data pertaining to microclimate viz., the mean weekly maximum and minimum temperatures (°C) and mean dry bulb temperature and per cent relative humidity recorded daily at 8 am and 2 pm inside the experimental house during the study period, (18th December 2006 to 29th January 2007) are presented in Table.3. During the course of experiment the mean maximum temperature ranged from 31.14 to 32.57°C with overall mean of 31.71°C, while the mean minimum temperature ranged from 24.28 to 26.50°C with overall mean of 25.44°C. The mean dry bulb temperature at 8 am in the forenoon ranged from 24.86 to 28°C with overall mean of 27.1°C, whereas, the range was from 29 to 31.14°C at 2 pm in the afternoon with overall mean of 31°C. The per cent relative humidity varied from 60 to 68.71 in the forenoon (8.0 am) with overall mean of 63.14 and from 42 to 55.57 at 2 pm in the afternoon with overall mean of 48.

4.2 BODY WEIGHT

The mean body weight of broiler chicken recorded at weekly interval as influenced by the supplementation of aflatoxin B1 and esterified glucomannan (E-GM) is given in Table 4 and graphically represented in Fig.1. The statistical analysis of the data on day-old body weight of chicks did not reveal any significant difference while, data on body weight from first to sixth week of age revealed significant ($P<0.05$) differences among the treatment groups.

Table 3. Mean weekly meteorological parameters inside the experimental house during the period from 18th December 2006 to 29th January 2007

Age in week	Temperature (°C)		Dry bulb temperature (°C)		Relative humidity (%)	
	Maximum	Minimum	Forenoon (8 am)	Afternoon (2 pm)	Forenoon (8 am)	Afternoon (2 pm)
1	31.32	25.68	26.06	29.10	60.00	48.86
2	31.28	25.28	25.00	30.00	65.00	47.14
3	31.14	24.28	24.86	29.00	68.71	49.14
4	32.28	26.50	28.00	30.00	61.00	55.57
5	32.57	24.85	26.00	31.14	61.71	42.00
6	32.07	25.85	27.10	31.00	62.43	45.29
Overall mean	31.71	25.44	27.10	31.00	63.14	48.00

Table 4. Mean weekly body weight (g) of broiler chicken as influenced by aflatoxin B1 and Esterified Glucomannan supplementation in experimental diets

Treatment	Age (Weeks)						
	0	1	2	3	4	5	6
T1 E-GM=0.0% Aflatoxin B1=0 ppm	41.68 ± 0.25	120.50^{a} ± 2.32	320.80^{a} ± 5.60	640.50^{a} ± 18.23	1070.40^{a} ± 7.03	1649.20^{a} ± 11.68	2218.94^{a} ± 29.95
T2 E-GM=0.0% Aflatoxin B1=1 ppm	41.25 ± 0.35	102.14^{b} ± 3.67	189.88^{d} ± 7.82	307.39^{c} ± 6.65	561.20^{c} ± 7.58	1008.02^{c} ± 5.83	1483.20^{c} ± 10.78
T3 E-GM=0.1% Aflatoxin B1=0 ppm	42.42 ± 0.69	121.50^{a} ± 1.44	299.10^{b} ± 6.73	609.03^{a} ± 27.67	1061.50^{a} ± 39.29	1633.00^{a} ± 20.60	2198.29^{a} ± 34.76
T4 E-GM=0.1% Aflatoxin B1=1 ppm	41.78 ± 0.33	108.80^{b} ± 1.55	223.80^{c} ± 4.01	359.73^{b} ± 6.13	694.14^{b} ± 6.54	1214.00^{b} ± 1.97	1765.40^{b} ± 7.65
Overall mean	41.78 ± 0.22	113.23 ± 2.17	258.39 ±12.60	479.16 ± 34.68	846.81 ± 52.28	1376.05 ± 63.32	1916.45 ± 71.68

Means bearing the different superscripts within the same column differ significantly ($P<0.05$)

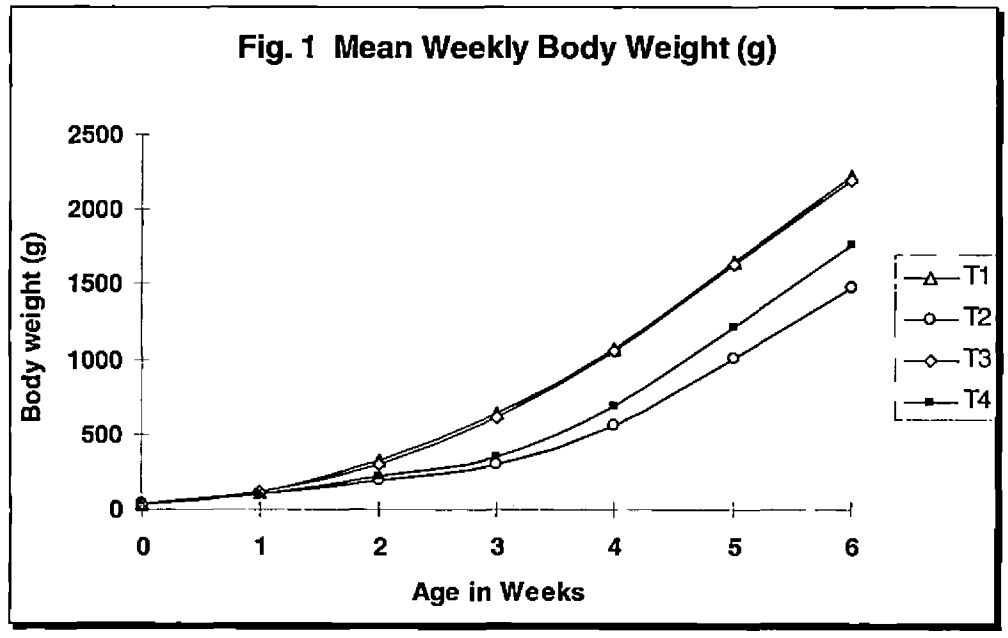


Fig. 1 Mean Weekly Body Weight (g)
Body weight (g)
2500
2000
1500
1000
500
0
0
1
2
3
4
5
6
Age in Weeks
T1
T2
T3
T4

The day-old body weight of chicks revealed no significance among treatments signifying proper randomization of experimental units.

At the first week, birds fed control diet (T1) and E-GM alone (T3) had statistically comparable but significantly ($P<0.05$) higher body weight than aflatoxin B1 alone (T2) and aflatoxin B1 plus E-GM supplemented (T4) groups. Between aflatoxin treated groups (T2 and T4), the body weight in E-GM supplemented birds (T4) was numerically superior to aflatoxin alone fed birds (T2); however, the difference was not statistically significant.

The second week mean body weight was highest in T1 (control) and lowest in T2 (aflatoxin B1 alone) with T3 (E-GM alone) and T4 (Aflatoxin B1 and E-GM) falling in between them. The mean body weights from all the four treatments were significantly ($P<0.05$) different from each other.

The third week mean body weights of control (T1) and E-GM alone (T3) fed groups were statistically similar and significantly ($P<0.05$) higher than the body weights of aflatoxin B1 treated birds either alone or in combination with E-GM (T2 and T4). However, between aflatoxin treated groups either alone (T2) or in combination with E-GM (T4), T4 registered significantly ($P<0.05$) superior body weight than T2.

At fourth week of age, higher body weight was recorded in treatment T1 (1070.40 g) followed by the treatments T3 (1061.50 g), T4 (694.14 g) and T2 (561.20 g). The birds fed control diet (T1) and E-GM alone (T3) had statistically similar body weights but weighed significantly ($P<0.05$) higher than the birds fed aflatoxin containing feed either with or without E-GM (T4 and T2). The birds fed

aflatoxin B1 alone (T2) showed significantly ($P<0.05$) inferior body weight than the birds fed aflatoxin B1 along with E-GM (T4).

The body weight at fifth week of age followed similar trend to that of previous two weeks.

At the end of sixth week of age, the mean body weights recorded were 2218.94, 1483.20, 2198.29 and 1765.40 g for treatments T1, T2, T3 and T4 respectively with overall mean of 1916.45g. The trend in body weight among the treatments was similar to that of third, fourth and fifth weeks. The sixth week body weights in T1 (control) and T3 (E-GM alone) were statistically similar and significantly ($P<0.05$) higher than that of other treatments (T2 and T4). The mean body weight in aflatoxin B1 plus E-GM fed birds (T4) was significantly ($P<0.05$) higher than their counterparts fed aflatoxin B1 alone (T2).

4.3 BODY WEIGHT GAIN

The mean body weight gain of broiler chicken at weekly intervals as influenced by the supplementation of aflatoxin B1 and E-GM is given in Table 5 and graphically depicted in Figures. 2 and 3. Statistical analysis of the data on mean body weight gain from first to sixth week and cumulative body weight gain from zero to four and from zero to six weeks of age revealed significant ($P<0.05$) differences among the treatments.

The birds fed aflatoxin B1 alone (T2) had significantly ($P<0.05$) lower first week weight gain than the birds maintained on control diet (T1) and E-GM alone (T3). Although the birds fed combination of aflatoxin B1 and E-GM (T4) had numerically superior weight gain than aflatoxin B1 alone fed group (T2), the difference was statistically insignificant.

Table 5. Mean weekly and cumulative weight gains (g) of broiler chicken as influenced by aflatoxin B1 and Esterified Glucomannan supplementation in experimental diets.

Treatment	Age (Weeks)						Cumulative (Period)	
	1	2	3	4	5	6	0-4	0-6
T1 E-GM=0.0% Aflatoxin B1=0 ppm	68.42^{a} ± 1.71	210.70^{a} ± 4.49	327.30^{a} ± 23.79	429.90^{a} ± 15.15	578.80^{a} ± 6.88	567.74^{a} ± 18.97	1036.31^{a} ± 13.82	2182.86^{a} ± 33.83
T2 E-GM=0.0% Aflatoxin B1=1 ppm	56.89^{b} ± 1.49	91.74^{d} ± 6.58	117.51^{b} ± 1.25	253.81^{c} ± 4.48	446.82^{c} ± 10.28	475.18^{b} ± 7.33	519.95^{c} ± 7.85	1441.95^{c} ± 10.56
T3 E-GM=0.1% Aflatoxin B1=0 ppm	66.68^{a} ± 3.35	190.00^{b} ± 5.14	309.93^{a} ± 25.69	452.47^{a} ± 18.20	571.50^{a} ± 23.44	565.29^{a} ± 14.33	1019.08^{a} ± 39.34	2155.87^{a} ± 34.88
T4 E-GM=0.1% Aflatoxin B1=1 ppm	62.22^{ab} ± 1.07	119.50^{c} ± 3.72	133.23^{b} ± 1.90	334.41^{b} ± 8.79	519.86^{b} ± 5.97	551.40^{a} ± 8.00	649.36^{b} ± 7.88	1720.62^{b} ± 9.10
Overall mean	63.55 ± 1.40	152.98 ± 11.46	221.99 ± 23.66	367.65 ± 19.11	529.25 ± 13.61	539.90 ± 10.79	806.18 ± 52.85	1875.32 ± 72.12

Means bearing the different superscripts within the same column differ significantly ($P<0.05$)

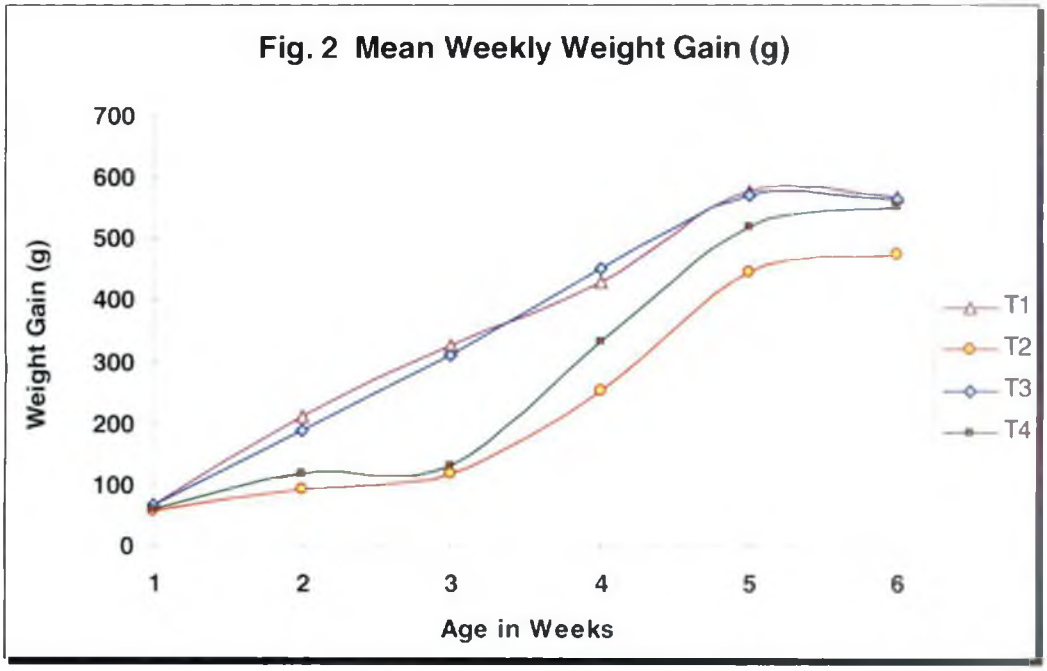


Fig. 2 Mean Weekly Weight Gain (g)
700
600
500
400
300
200
100
0
Weight Gain (g)
1
2
3
4
5
6
Age in Weeks
T1
T2
T3
T4

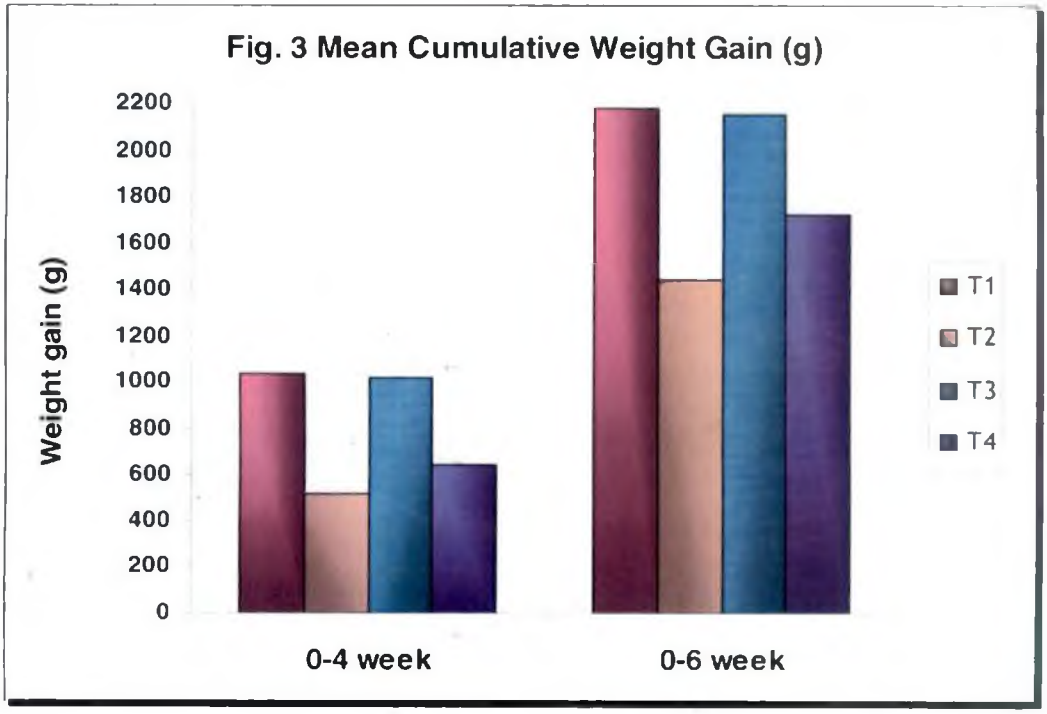


Fig. 3 Mean Cumulative Weight Gain (g)
2200
2000
1800
1600
1400
1200
1000
800
600
400
200
0
Weight gain (g)
0-4 week
0-6 week
T1
T2
T3
T4

The second week weight gain was highest in T1 (control) and lowest in T2 (aflatoxin B1 alone) with T3 (E-GM alone) and T4 (aflatoxin B1 plus E-GM) falling in between them. The weight gain at this age from all the four treatment groups differed significantly ($P<0.05$) from each other.

At third week, weight gain in aflatoxin B1 fed treatment groups (T4 and T2) recorded significantly ($P<0.05$) lower weight gain than non-aflatoxin treatment groups (T1 and T3) irrespective of E-GM supplementation. Treatments T4 and T2 were statistically comparable but the value in T4 was numerically higher than T2.

At fourth week, the birds maintained on aflatoxin B1 alone (T2) had significantly ($P<0.05$) lower weight gain than the birds in other treatment groups. However, non-aflatoxin fed birds with or without E-GM supplementation (T1 and T3) were statistically similar in their weight gain. Between aflatoxin B1 fed birds (T2 and T4), weight gain in E-GM supplemented group (T4) was significantly ($P<0.05$) higher than in non-supplemented group (T2).

The mean weight gains at five weeks of age of non-aflatoxin fed treatment groups irrespective of E-GM supplementation (T1 and T3) were statistically comparable and significantly ($P<0.05$) higher than their aflatoxin B1 fed counterparts in T2 and T4. Between aflatoxin B1 fed birds (T2 and T4), weight gain in E-GM supplemented group (T4) was significantly ($P<0.05$) higher than in non-supplemented group (T2).

At sixth week, the birds fed with aflatoxin B1 alone (T2) had significantly ($P<0.05$) lower weight gain than the birds maintained on control diet (T1), 0.1 per cent E-GM alone (T3) and combination of aflatoxin B1 and E-GM (T4).

Treatments T1, T3 and T4 were statistically comparable and formed homogenous group.

The treatment groups fed control (T1) and E-GM alone supplemented (T3) diets had statistically similar but significantly ($P<0.05$) higher fourth week cumulative weight gain than aflatoxin B1 (T2) and aflatoxin B1 with E-GM (T4) treated groups. The treatment T4 had significantly ($P<0.05$) higher cumulative weight gain than T2 at fourth week of age.

The cumulative weight gain (g) at sixth week of age was 2182.86, 1441.95, 2155.87 and 1720.62 for T1, T2, T3 and T4 respectively with overall mean of 1875.32. At sixth week the cumulative weight gain in non-aflatoxin treated birds (T1 and T3) were statistically similar but significantly ($P<0.05$) higher than their aflatoxin B1 treated counterparts (T2 and T4). Between the aflatoxin B1 treated groups (T2 and T4), E-GM supplemented group (T4) gained significantly ($P<0.05$) higher weight than aflatoxin B1 alone treated birds (T2).

4.4 FEED INTAKE

The mean weekly and cumulative feed intake as influenced by dietary inclusion of aflatoxin B1 and E-GM is given in Table 6 and graphically represented in Figures. 4 and 5. Statistical analysis of the data on weekly feed intake from first to six weeks of age and cumulative feed intake from zero to four and zero to six weeks of age revealed significant ($P<0.05$) differences among the treatments.

At the end of first week of age, feed intake recorded in birds fed aflatoxin B1 alone (T2) was significantly ($P<0.05$) lower than the birds maintained on control diet (T1) and 0.1 percent E-GM alone (T3). The treatments T1 and T3 were statistically comparable and formed homogenous group.

Table 6. Mean weekly and cumulative feed intake (g) of broiler chicken as influenced by aflatoxin B1 and Esterified Glucomannan supplementation in experimental diets.

Treatment	Age (Weeks)						Cumulative (Period)	
	1	2	3	4	5	6	0-4	0-6
T1 E-GM=0.0% Aflatoxin B1=0 ppm	105.51^{a} ± 1.52	344.70^{a} ± 5.21	557.81^{a} ± 35.43	765.21^{a} ± 15.71	1108.92^{a} ± 28.77	1177.26^{a} ± 16.95	1773.23^{a} ± 29.57	4059.41^{a} ± 59.37
T2 E-GM=0.0% Aflatoxin B1=1 ppm	92.59^{b} ± 1.31	163.00^{d} ± 12.41	214.60^{b} ± 4.84	495.60^{c} ± 11.60	904.00^{b} ± 14.27	1102.40^{b} ± 6.77	965.79^{c} ± 4.96	2972.19^{c} ± 23.06
T3 E-GM=0.1% Aflatoxin B1=0 ppm	103.35^{a} ± 3.74	311.00^{b} ± 9.59	529.44^{a} ± 30.78	785.80^{a} ± 27.88	1075.00^{a} ± 45.45	1173.82^{a} ± 22.16	1729.59^{a} ± 53.57	3978.41^{a} ± 62.25
T4 E-GM=0.1% Aflatoxin B1=1 ppm	98.73^{ab} ± 1.01	206.84^{c} ± 4.58	238.20^{b} ± 6.50	630.67^{b} ± 15.67	1027.02^{a} ± 7.69	1208.20^{a} ± 4.80	1174.45^{b} ± 16.42	3409.67^{b} ± 20.03
Overall mean	100.05 ± 1.51	256.39 ± 17.45	385.01 ± 38.11	669.32 ± 28.10	1028.74 ± 21.99	1165.42 ± 11.12	1410.77 ± 81.36	3604.92 ± 103.74

Means bearing the different superscripts within the same column differ significantly ($P<0.05$)

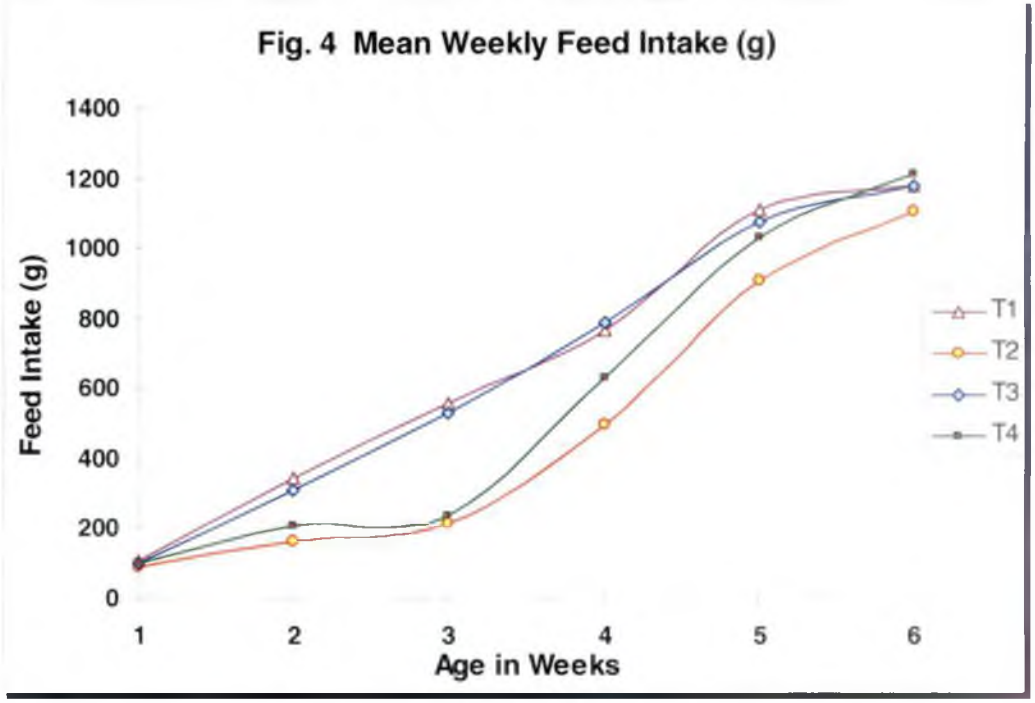


Fig. 4 Mean Weekly Feed Intake (g)
1400
1200
1000
800
600
400
200
0
Feed Intake (g)
1
2
3
4
5
6
Age in Weeks
T1
T2
T3
T4

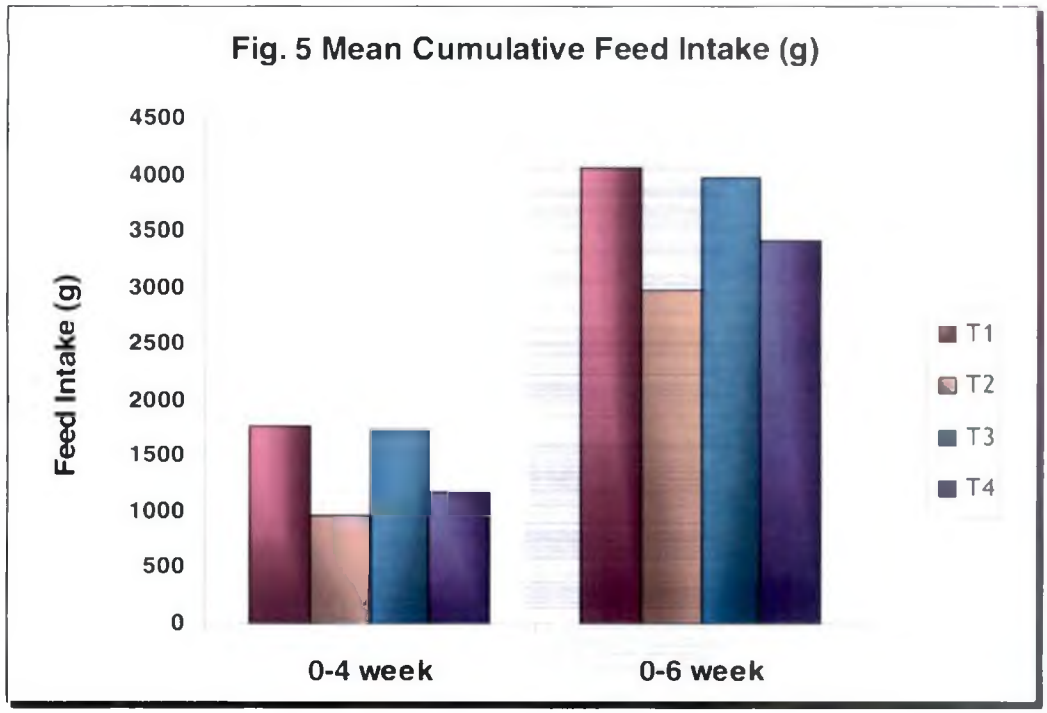


Fig. 5 Mean Cumulative Feed Intake (g)
4500
4000
3500
3000
2500
2000
1500
1000
500
0
Feed Intake (g)
0-4 week
0-6 week
T1
T2
T3
T4

The treatment T4 (aflatoxin B1 and E-GM combination) had numerically higher feed intake than T2 (aflatoxin B1 alone), however, the difference was statistically insignificant.

The second week feed intake was highest in T1 (control group) and lowest in T2 (aflatoxin B1alone) with T3 (E-GM alone) and T4 (combination of aflatoxin B1 and E-GM) falling in between them. The feed intake in all the four treatments were significantly ($P<0.05$) different from each other.

The groups fed treatment diets containing no aflatoxin B1 irrespective of E-GM supplementation (T1 and T3) had statistically similar third week feed intake which was significantly ($P<0.05$) higher than that of aflatoxin treated birds either alone (T2) or in combination with E-GM (T4). Aflatoxin B1 treated birds with E-GM supplementation (T4) had numerically higher feed intake than their non-E-GM supplemented counterparts (T2). However, the difference was statistically insignificant.

The birds maintained on aflatoxin B1 alone (T2) had significantly ($P<0.05$) lower fourth week feed intake than the birds in other treatment groups. However non- aflatoxin fed birds irrespective of E-GM supplementation (T1 and T3) were statistically similar in their feed intake. Between aflatoxin B1 fed birds (T2 and T4), feed intake in E-GM supplemented group (T4) was significantly ($P<0.05$) higher than that of non-supplemented group (T2).

The mean weekly feed intake recorded at fifth week of age in control (T1), E-GM alone (T3) and combination of aflatoxin B1 and E-GM (T4) was significantly ($P<0.05$) higher than that of aflatoxin B1 alone (T2) fed birds. The feed intake in

treatments T1, T3 and T4 was statistically comparable and form homogenous group.

The statistical analysis of the data on feed intake recorded at the end of sixth week also revealed similar results to that of fifth week. Feed intake in aflatoxin B1 alone fed group (T2) was significantly lower than all the other treatment groups. The birds fed control diet (T1), E-GM alone (T3) and aflatoxin B1 plus E-GM (T4) had statistically similar sixth week feed intake.

The mean cumulative feed intake up to fourth week in T1 (control) and T3 (E-GM alone) was statistically similar and significantly ($P<0.05$) higher than that of T2 (aflatoxin B1 alone) and T4 (aflatoxin B1 plus E-GM). The birds offered a diet containing aflatoxin B1 alone (T2) consumed significantly ($P<0.05$) less feed than those supplemented with E-GM along with aflatoxin B1 (T4).

The mean cumulative feed intake of birds up to sixth week for T1, T2, T3 and T4 (4059.41, 2972.19, 3978.41 and 3409.67 respectively) followed similar trend to that of cumulative feed intake upto fourth week. Statistically similar feed intake was recorded in control (T1) and E-GM alone (T3) fed groups, which were significantly ($P<0.05$) higher than that of other treatments (T2 and T4). Between aflatoxin B1 fed groups (T2 and T4), E-GM supplemented group (T4) had significantly ($P<0.05$) higher feed intake than the non-supplemented group (T2).

4.5 FEED EFFICIENCY

The mean weekly feed efficiency of birds maintained on different dietary treatments is presented in Table 7 and graphically represented in Figures. 6 and 7. Data on feed efficiency revealed significant ($P<0.05$) differences among the dietary groups during second, third, fourth, fifth and sixth weeks of age.

Table 7. Mean weekly and cumulative feed efficiency (kg feed / kg body weight gain) of broiler chicken as influenced by aflatoxin B1 and Esterified Glucomannan supplementation in experimental diets

Treatment	Age (Weeks)						Cumulative (period)	
	1	2	3	4	5	6	0-4	0-6
T1 E-GM=0.0% Aflatoxin B1=0 ppm	1.54 ± 0.04	1.64^{b} ± 0.01	1.71^{b} ± 0.02	1.79^{bc} ± 0.03	1.91^{b} ± 0.05	2.08^{b} ± 0.05	1.71^{b} ± 0.01	1.86^{c} ± 0.02
T2 E-GM=0.0% Aflatoxin B1=1 ppm	1.63 ± 0.05	1.77^{a} ± 0.04	1.82^{a} ± 0.03	1.95^{a} ± 0.05	2.02^{a} ± 0.04	2.32^{a} ± 0.02	1.86^{a} ± 0.03	2.06^{a} ± 0.01
T3 E-GM=0.1% Aflatoxin B1=0 ppm	1.56 ± 0.03	1.64^{b} ± 0.01	1.72^{b} ± 0.04	1.74^{c} ± 0.03	1.88^{b} ± 0.02	2.08^{b} ± 0.09	1.70^{b} ± 0.02	1.85^{c} ± 0.03
T4 E-GM=0.1% Aflatoxin B1=1 ppm	1.59 ± 0.04	1.74^{ab} ± 0.05	1.79^{ab} ± 0.03	1.89^{ab} ± 0.03	1.98^{ab} ± 0.02	2.19^{ab} ± 0.03	1.81^{a} ± 0.02	1.98^{b} ± 0.01
Overall mean	1.58 ± 0.02	1.70 ± 0.02	1.76 ± 0.02	1.84 ± 0.02	1.95 ± 0.02	2.17 ± 0.02	1.77 ± 0.02	1.94 ± 0.02

Means bearing the different superscripts within the same column differ significantly ($P<0.05$)


Fig. 6 Mean Weekly Feed Efficiency
Feed Efficiency
2.4
2.2
2
1.8
1.6
1.4
1.2
1
2
3
4
5
6
Age in Weeks
T1
T2
T3
T4

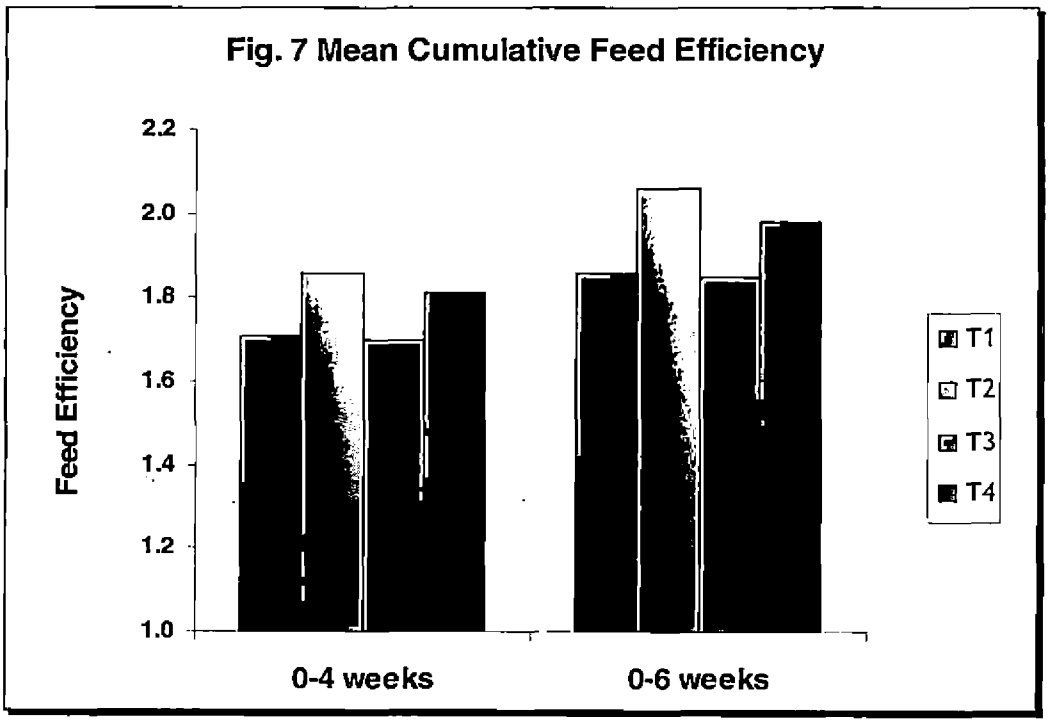


Fig. 7 Mean Cumulative Feed Efficiency
Feed Efficiency
2.2
2.0
1.8
1.6
1.4
1.2
1.0
0-4 weeks
0-6 weeks
T1
T2
T3
T4

The data revealed that there was no significant effect on feeding aflatoxin B1 and/or E-GM on the first week feed efficiency. The analysis of data on cumulative feed efficiency (0-4 and 0-6 weeks of age) revealed significant differences ($P<0.05$) among the treatments.

Feeding of aflatoxin B1 and /or E-GM had no effect on mean feed efficiency at first week of age. At second week of age, the feed efficiency was statistically similar in control (T1), E-GM alone (T3) and aflatoxin B1 plus E-GM (T4) fed groups. Although the feed efficiency of aflatoxin B1 alone fed birds (T2) was statistically comparable with aflatoxin B1 plus E-GM (T4) fed group, which was significantly ($P<0.05$) inferior to T1 and T3.

The statistical analysis of the data on feed efficiency recorded at the end of third week revealed the same trend to that of second week.

At fourth week of age, superior and statistically comparable weekly feed efficiency was noted in non-aflatoxin treatment groups (T1 and T3). Among the four treatments, significantly ($P<0.05$) inferior feed efficiency was observed in aflatoxin B1 alone fed birds (T2). Although feed efficiency in aflatoxin B1 plus E-GM treated birds (T4) was statistically comparable to T2, the numeric value of feed efficiency was higher in T2.

At fifth week, superior weekly feed efficiencies were recorded in T1 (control), T3 (E-GM alone) and T4 (aflatoxin B1 plus E-GM), which were statistically comparable also. Aflatoxin B1 fed birds (T2) had significantly ($P<0.05$) inferior feed efficiency than aflatoxin free treatment groups (T1 and T3). Eventhough the aflatoxin alone treated birds (T2) had numerically higher feed efficiency value than aflatoxin B1 plus E-GM treated birds (T4), statistical analysis did not reveal any significant difference.

At sixth week of age, the feed efficiency for the dietary groups T1, T2, T3 and T4 were 2.08, 2.32, 2.08 and 2.19 respectively with an overall mean of 2.17. Statistical analysis of the data revealed same trend to that of fifth week. Superior weekly feed efficiencies were recorded in T1 (control), T3 (E-GM alone) and T4 (aflatoxin B1 plus E-GM), which were statistically comparable also. Aflatoxin B1 fed birds (T2) had significantly ($P<0.05$) inferior feed efficiency than aflatoxin free treatment groups (T1 and T3). Between the aflatoxin treated birds (T2 and T4), the E-GM supplemented group (T4) had numerically lower feed efficiency value than aflatoxin B1 alone treated birds (T2). However, statistical analysis did not reveal any significant difference between them.

The mean cumulative feed efficiency values from zero to four weeks in treatment groups receiving aflatoxin B1 either alone (T2) or in combination with E-GM (T4) were statistically similar and significantly ($P<0.05$) higher than non-aflatoxin treated birds irrespective of E-GM supplementation (T1 and T3). Treatments T1 and T3 were also statistically comparable.

The cumulative feed efficiency from zero to six weeks in non-aflatoxin treatment groups fed without or with E-GM (T1 and T3) had statistically similar but significantly ($P<0.05$) superior feed efficiency than their aflatoxin fed counterparts (T2 and T4). Birds fed both aflatoxin B1 and E-GM (T4) had significantly ($P<0.05$) superior feed efficiency than aflatoxin B1 alone (T2) fed birds.

4.6 SERUM PARAMETERS

The mean values of serum total protein, albumin, glucose and cholesterol levels in broiler chickens estimated at the end of sixth week of age as influenced by different dietary treatments are presented in Table 8 and graphically represented in fig.8 and 9. Statistical analysis of the data on serum albumin level at sixth week of

Table 8. Mean serum biochemical values of broiler chicken as influenced by aflatoxin B1 and Esterified Glucomannan supplementation in experimental diets.

Treatment	Serum total protein (g per dl)	Serum albumin (g per dl)	Serum glucose (mg per dl)	Serum total cholesterol (mg per dl)
T1 E-GM=0.0% Aflatoxin B1= 0 ppm	2.81^{a} ± 0.19	1.53^{a} ± 0.07	104.47^{b} ± 13.18	110.80^{a} ± 1.66
T2 E-GM=0.0% Aflatoxin B1=1 ppm	1.99^{b} ± 0.09	1.19^{b} ± 0.07	160.22^{a} ± 3.82	91.15^{b} ± 5.82
T3 E-GM=0.1% Aflatoxin B1=0 ppm	2.97^{a} ± 0.22	1.53^{a} ± 0.09	112.36^{b} ± 14.76	112.69^{a} ± 2.35
T4 E-GM=0.1% Aflatoxin B1= 1 ppm	3.26^{a} ± 0.24	1.55^{a} ± 0.09	109.85^{b} ± 3.66	106.64^{a} ± 2.37
Overall mean	2.76 ± 0.14	1.45 ± 0.05	121.73 ± 6.96	105.32 ± 2.51

Means bearing the different superscripts within the same column differ significantly ($P<0.05$)

age revealed significant (P<0.05) differences among treatments; whereas, total protein, glucose and cholesterol showed significant (P<0.05) differences among the treatments.

The serum protein level in aflatoxin B1 alone fed birds (T2) was significantly (P<0.05) lower than all the other treatments. The treatments T1 (control), T3 (E-GM alone) and T4 (aflatoxin B1 and E-GM) were statistically comparable and formed homogenous group. Similarly, the serum albumin level in aflatoxin B1 alone fed group (T2) was significantly (P<0.05) lower than other treatments. The treatments T1, T3 and T4 were statistically comparable and formed homogenous group.

On the other hand, glucose level in aflatoxin B1 alone fed group (T2) was significantly (P<0.05) elevated than other treatments. The birds fed control diet (T1), E-GM alone (T3) and aflatoxin B1 and E-GM combination (T4) had statistically comparable serum glucose level at sixth week of age.

The birds fed control diet (T1), E-GM (T3) and aflatoxin B1 plus E-GM (T4) had statistically similar serum cholesterol level. Whereas, significantly (P<0.05) lower serum cholesterol value was noted with the group T2 i.e. birds treated with aflatoxin B1 alone.

4.7 PROCESSING YIELDS AND LOSSES

The mean per cent dressed, eviscerated, giblet and ready-to-cook yields and blood, feather and total losses recorded in broilers slaughtered at six weeks of age as influenced by dietary inclusion of aflatoxin B1 and E-GM are presented in Table 9 and graphically represented in Figures. 10, 11 and 12. The statistical analysis of the data on processing yields and losses revealed that dressed yield, eviscerated

Table 9. Mean per cent processing yields and losses of broiler chicken as influenced by aflatoxin B1 and Esterified Glucomannan supplementation in experimental diets

Treatment	Dressed yield	Eviscerated yield	Giblet yield	Ready-to-cook yield	Blood loss	Feather loss	Total loss
T1 E-GM=0.0% Aflatoxin B1= 0 ppm	83.56^{a} ± 0.08	67.72^{a} ± 0.11	4.93^{bc} ± 0.34	72.65 ± 0.34	5.07 ± 0.08	12.25 ± 0.09	27.35 ± 0.34
T2 E-GM=0.0% Aflatoxin B1=1 ppm	82.64^{b} ± 0.09	65.17^{b} ± 0.50	6.62^{a} ± 0.07	71.78 ± 0.50	4.93 ± 0.21	12.08 ± 0.06	28.22 ± 0.50
T3 E-GM=0.1% Aflatoxin B1=0 ppm	83.57^{a} ± 0.07	67.71^{a} ± 0.21	4.87^{c} ± 0.18	72.57 ± 0.35	5.11 ± 0.08	12.16 ± 0.06	27.43 ± 0.35
T4 E-GM=0.1% Aflatoxin B1= 1 ppm	83.46^{a} ± 0.05	67.02^{a} ± 0.29	5.66^{b} ± 0.31	72.68 ± 0.23	4.84 ± 0.07	12.12 ± 0.19	27.32 ± 0.23
Overall mean	83.31 ± 0.10	66.90 ± 0.28	5.52 ± 0.20	72.42 ± 0.19	4.99 ± 0.06	12.15 ± 0.05	27.58 ± 0.19

Means bearing the different superscripts within the same column differ significantly ($P<0.05$)

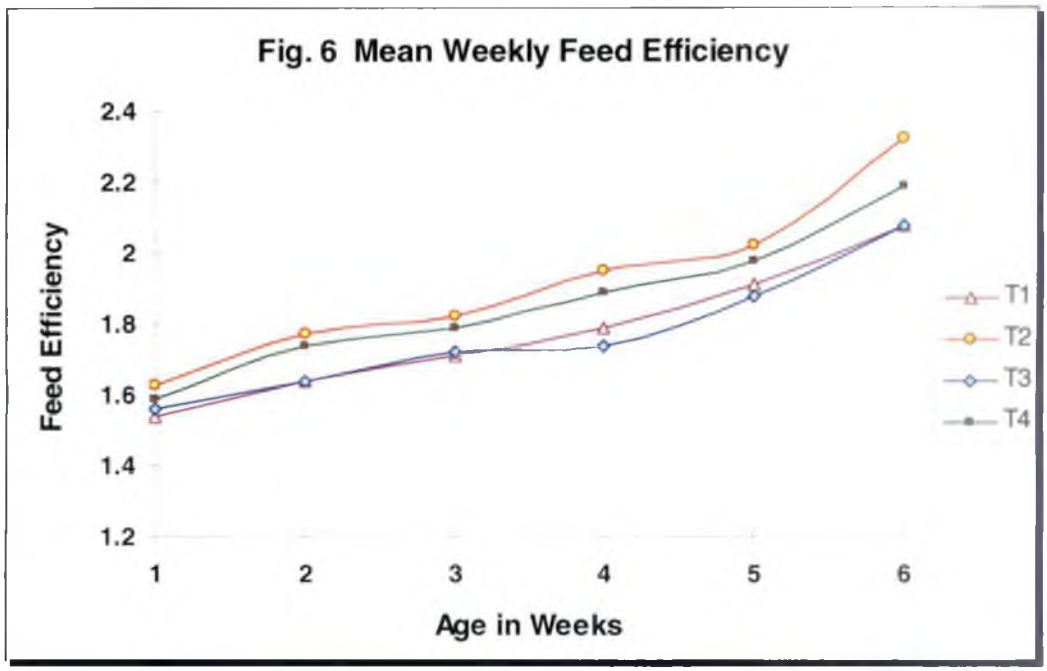


Fig. 6 Mean Weekly Feed Efficiency
Feed Efficiency
2.4
2.2
2
1.8
1.6
1.4
1.2
1
2
3
4
5
6
Age in Weeks
T1
T2
T3
T4

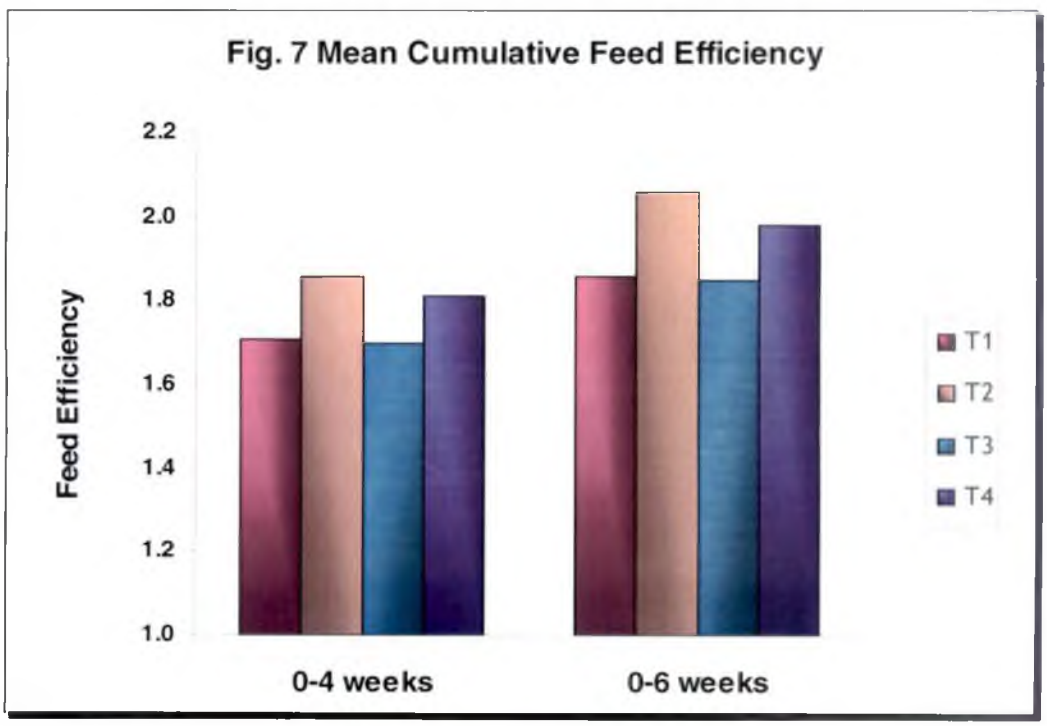


Fig. 7 Mean Cumulative Feed Efficiency
Feed Efficiency
2.2
2.0
1.8
1.6
1.4
1.2
1.0
0-4 weeks
0-6 weeks
T1
T2
T3
T4

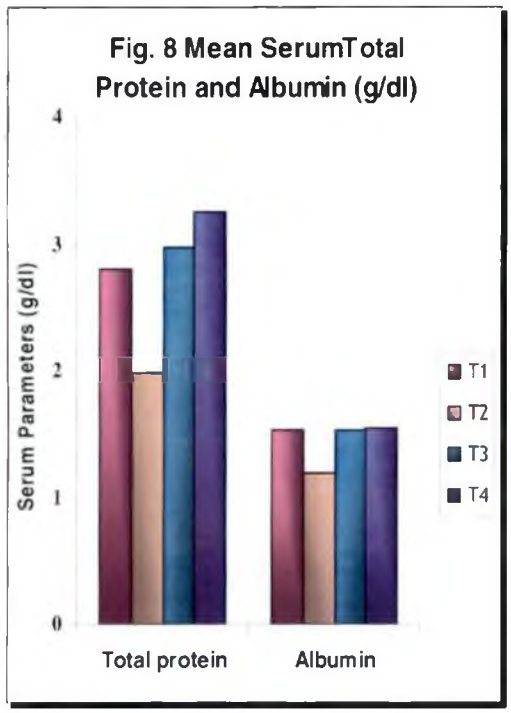


Fig. 8 Mean SerumTotal Protein and Albumin (g/dl)
Serum Parameters (g/dl)
0
1
2
3
4
Total protein
Albumin
T1
T2
T3
T4

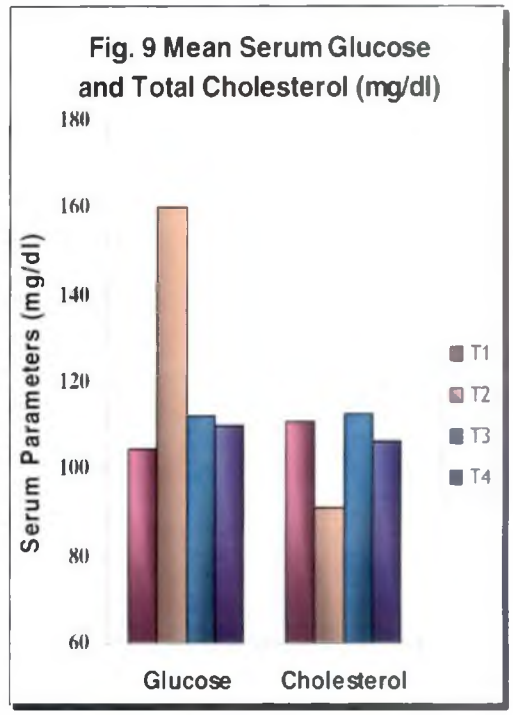


Fig. 9 Mean Serum Glucose and Total Cholesterol (mg/dl)
Serum Parameters (mg/dl)
60
80
100
120
140
160
180
Glucose
Cholesterol
T1
T2
T3
T4

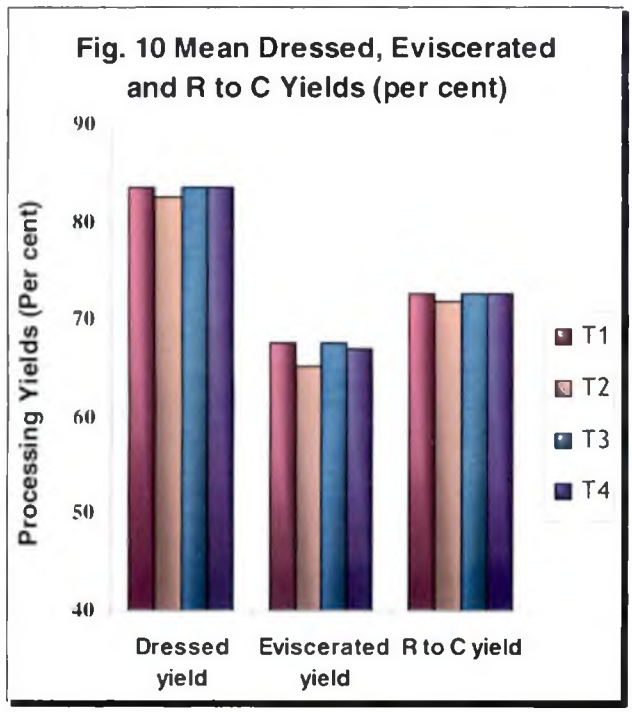


Fig. 10 Mean Dressed, Eviscerated and R to C Yields (per cent)
Processing Yields (Per cent)
40
50
60
70
80
90
Dressed yield
Eviscerated yield
R to C yield
T1
T2
T3
T4

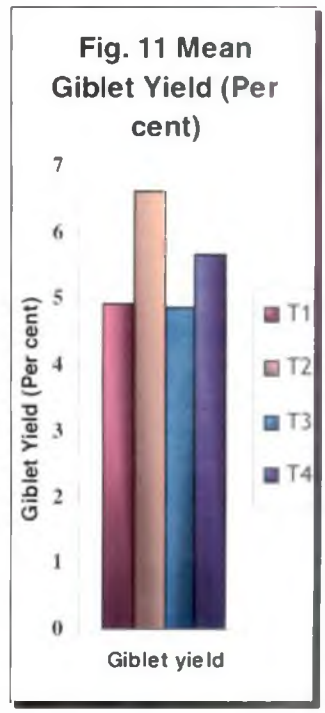


Fig. 11 Mean Giblet Yield (Per cent)
Giblet Yield (Per cent)
0
1
2
3
4
5
6
7
Giblet yield
T1
T2
T3
T4

yield and giblet yield had significant ($P<0.05$) effect among the treatments. However, feeding aflatoxin B1 and /or E-GM had no effect on the ready-to-cook yield, blood loss, feather loss and total loss.

The dressed yields of control (T1), E-GM alone (T3) and combination of aflatoxin B1 and E-GM (T4) fed birds were statistically comparable and significantly ($P<0.05$) higher than aflatoxin B1 alone (T2) fed group.

The eviscerated yields of birds fed control (T1), E-GM alone (T3) and combination of aflatoxin B1 and E-GM (T4) were statistically comparable and significantly higher than aflatoxin B1 alone (T2) fed group.

The birds fed aflatoxin B1 alone (T2) recorded significantly ($P<0.05$) higher giblet yield. On the other hand, significantly ($P<0.05$) lower giblet yield was recorded in E-GM alone fed group (T3). Treatments T1 (control) and T4 (aflatoxin B1 plus E-GM) showed intermediatary values and were statistically comparable.

The other carcass characteristics like ready-to-cook yield, blood loss, feather loss and total loss were not influenced by feeding aflatoxin B1 and/or E-GM.

4.8 RELATIVE WEIGHT OF LIVER, SPLEEN, KIDNEY AND BURSA OF FABRICIUS

The average relative weights of liver, spleen, kidney and bursa of Fabricius of broiler chicken at six weeks of age maintained on different dietary treatments are presented in Table 10 and graphically represented in Figures 13 and 14. Statistical analysis of data on relative organ weights showed significant ($P<0.05$) differences among the treatments for liver, spleen, and kidney but no difference for bursa of Fabricius.

Table 10. Mean relative organ weights (g per 100 g body weight) of broiler chicken as influenced by aflatoxin B1 and Esterified Glucomannan (E-GM) supplementation in experimental diets.

Treatment	Relative Weight			
	Liver (g)	Spleen (g)	Kidney (g)	Bursa of Fabricius (g)
T1 E-GM=0.0% Aflatoxin B1= 0 ppm	2.310^{c} ± 0.180	0.102^{c} ± 0.010	0.480^{c} ± 0.042	0.230 ± 0.020
T2 E-GM=0.0% Aflatoxin B1=1 ppm	4.020^{a} ± 0.160	0.240^{a} ± 0.020	0.930^{a} ± 0.040	0.230 ± 0.020
T3 E-GM=0.1% Aflatoxin B1=0 ppm	1.994^{c} ± 0.110	0.112^{c} ± 0.010	0.454^{c} ± 0.031	0.230 ± 0.010
T4 E-GM=0.1% Aflatoxin B1= 1 ppm	3.170^{b} ± 0.200	0.160^{b} ± 0.013	0.674^{b} ± 0.073	0.242 ± 0.005
Overall mean	2.871 ± 0.20	0.150 ± 0.013	0.634 ± 0.050	0.232 ± 0.006

Means bearing the different superscripts within the same column differ significantly ($P<0.05$)

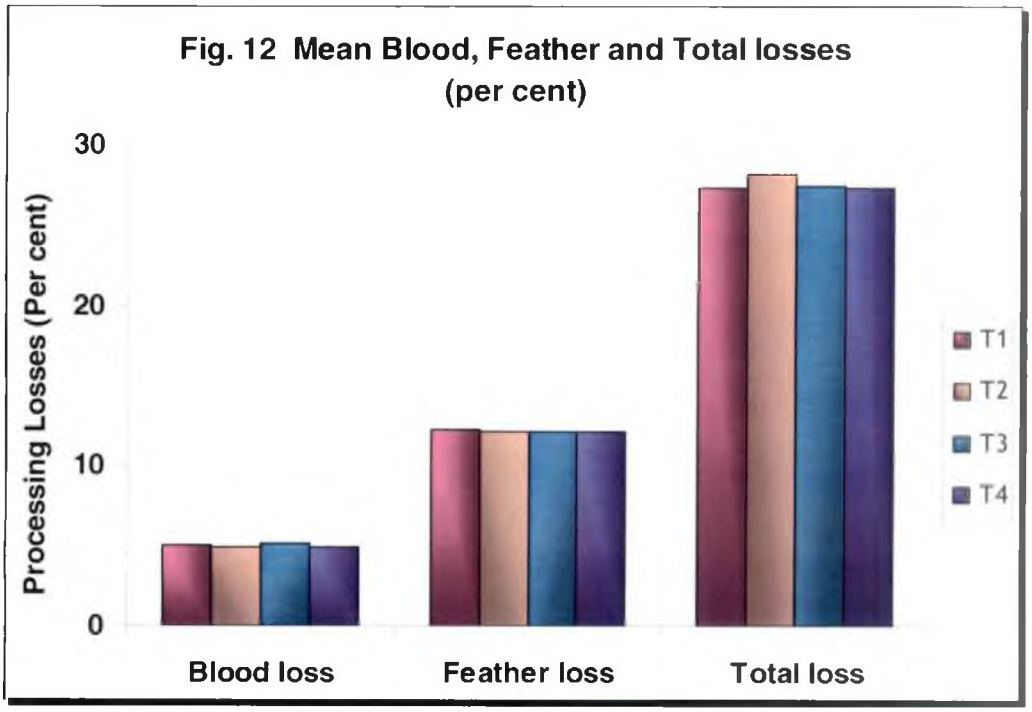


Fig. 12 Mean Blood, Feather and Total losses (per cent)
Processing Losses (Per cent)
30
20
10
0
Blood loss
Feather loss
Total loss
T1
T2
T3
T4

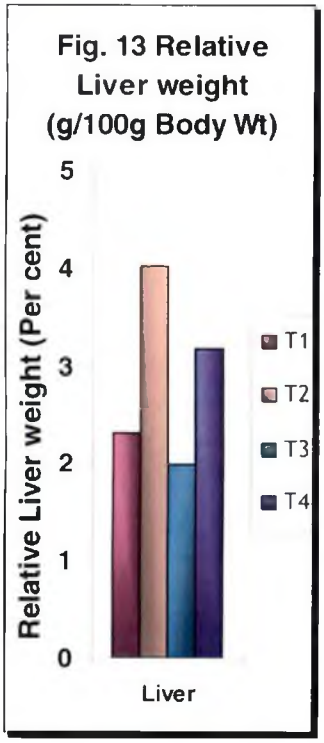


Fig. 13 Relative Liver weight (g/100g Body Wt)
Relative Liver weight (Per cent)
5
4
3
2
1
0
Liver
T1
T2
T3
T4

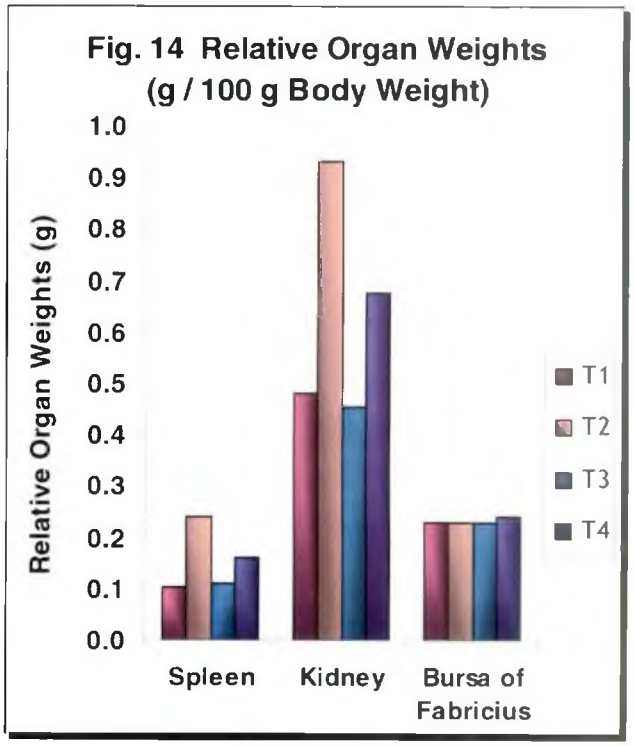


Fig. 14 Relative Organ Weights (g / 100 g Body Weight)
Relative Organ Weights (g)
1.0
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
Spleen
Kidney
Bursa of Fabricius
T1
T2
T3
T4

The relative weight of the liver in non-aflatoxin treatment groups (T1 and T3) were statistically similar and significantly lower than aflatoxin B1 treated groups irrespective of E-GM supplementation (T2 and T4). Between aflatoxin B1 fed groups (T2 and T4), the relative weight of liver was significantly ($P<0.05$) lower in E-GM supplemented group (T4) compared to their non-supplemented counterparts (T2). Among all the treatments, aflatoxin B1 alone fed group (T2) had significantly ($P<0.05$) higher relative weight of liver.

The birds fed with control diet (T1) and with 0.1 percent E-GM alone (T3) had statistically similar and significantly ($P<0.05$) low relative weight of spleen than their aflatoxin B1 fed counterparts with or without E-GM supplementation (T4 and T2). Between the birds fed aflatoxin B1 containing diets (T2 and T4), the E-GM supplemented group (T4) had significantly ($P<0.05$) low relative spleen weight than aflatoxin B1 alone fed group (T2).

The relative weights of the kidney in non-aflatoxin treatment groups (T1 and T3) were statistically comparable and significantly ($P<0.05$) lower than aflatoxin B1 treated groups irrespective of E-GM supplementation (T2 and T4).

The relative kidney weight in treatment T4 (aflatoxin B1 plus E-GM) was significantly lower than aflatoxin B1 alone fed group (T2).

The statistical analysis of the data on relative weight of bursa revealed no significant effect on feeding aflatoxin B1 and/or E-GM.

4.9 LIVABILITY

The mortality rate (per cent) as influenced by the feeding of aflatoxin B1 and/ or E-GM is presented in table 11.

Table 11. Weekly mortality rate (per cent) of broiler chicken as influenced by aflatoxin B1 and Esterified Glucomannan supplementation in experimental diets

Treatment	1st week	2nd week	3rd week	4th week	5th week	6th week	Total 0-6 weeks
T1 E-GM=0.0% Aflatoxin B1= 0 ppm	0.00	0.00	0.00	0.00	0.00	2.00	2.00
T2 E-GM=0.0% Aflatoxin B1=1 ppm	2.00	2.00	2.00	6.00	0.00	0.00	12.00
T3 E-GM=0.1% Aflatoxin B1=0 ppm	0.00	0.00	0.00	0.00	0.00	0.00	0.00
T4 E-GM=0.1% Aflatoxin B1= 1 ppm	0.00	2.00	4.00	2.00	2.00	0.00	10.00
Overall mean	0.50	1.00	1.50	2.00	0.50	0.50	10.50

There was only two per cent mortality in control group (T1) at sixth week of age; hence the livability in T1 was 98 per cent during the entire treatment period of six weeks.

There were two, two, two and six per cent mortalities during first, second, third and fourth weeks respectively and no mortality during the fifth and sixth week of age in aflatoxin B1 alone fed group (T2). The total mortality in T2 during entire experiment was 12 per cent. Therefore, overall livability in T2 was 88 per cent up to six weeks of age. There was no mortality in the E-GM alone (T3) treatment group during the entire experimental period of six weeks and hence the livability was 100 per cent. There were two, four, two and two per cent mortalities in the aflatoxin B1 plus E-GM fed group (T4) during second, third, fourth and fifth weeks of age respectively totaling to 10 per cent mortality for the entire trial period. Hence the livability in this group (T4) was 90 per cent for the total study period of six weeks.

4.10 GROSS LESION

The gross lesions observed in the liver of broiler chicken at six weeks of age maintained on different dietary treatments are shown in fig.15 On gross examination, the liver and kidneys of chicks fed 1 ppm aflatoxin B1 fed group (T2) were mostly swollen and pale yellow-red. The spleen was moderately enlarged while, no visible morphological changes were observed in bursa of Fabricius. The aflatoxin fed birds when supplemented with E-GM (T4) showed lesser degree of macroscopic lesions in liver, spleen and kidney.

The gross examination of organs from treatment T1 (control ration) and T3 (0.1 percent E-GM alone) revealed no gross pathological changes attributable to aflatoxicosis.

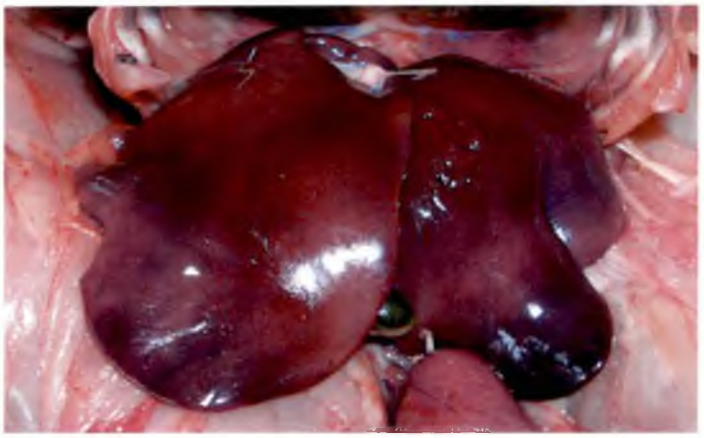


Fig 15a. Liver from Control diet fed group – Normal

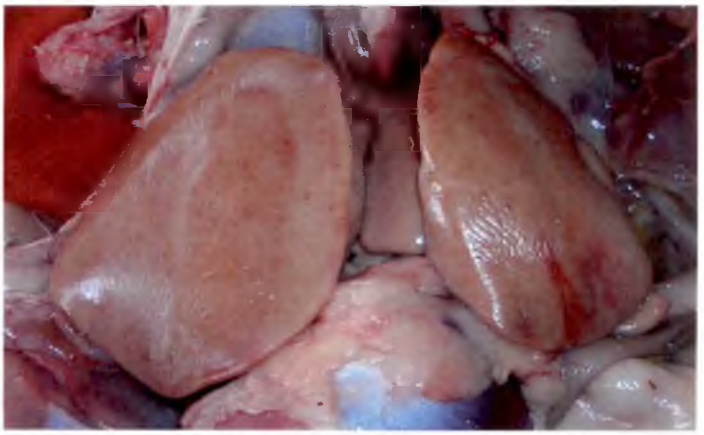


Fig 15b. Liver from Aflatoxin B1 alone fed group - Showing severe enlargement and paleness

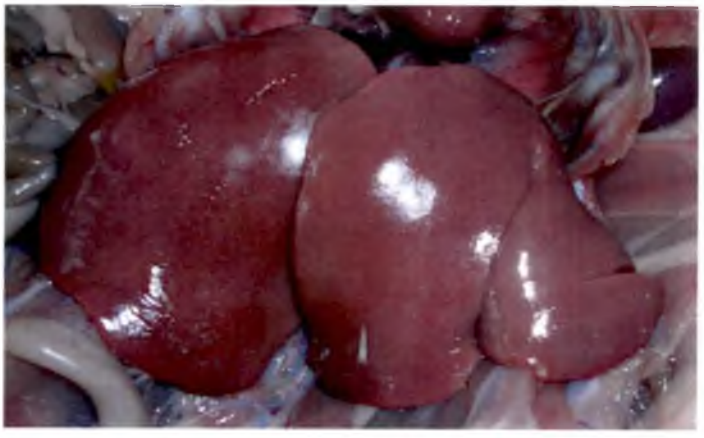


Fig 15c. Liver from Aflatoxin B1 plus E-GM fed group - Showing moderate enlargement and paleness

4.11 HISTOPATHOLOGY

Histopathological changes noticed in liver of broiler chicken at six weeks of age maintained on different dietary treatments are shown in fig.16. Microscopically, the liver of chicks fed 1 ppm aflatoxin B1 containing diet (T2) showed severe fatty changes, bile duct proliferation, periportal fibrosis in periportal areas, biliary hyperplasia and accumulation of lymphoid cell were seen within the hepatic lobules. While, in birds fed combination of 1 ppm aflatoxin B1 and 0.1 percent E-GM (T4), histopathological changes in the liver was reduced and no fatty changes were observed except the mild congestion. Histopathological examination of liver from treatment T1 (control) and T3 (E-GM alone fed) revealed no changes and were normal.

4.12 ECONOMICS

The economics of rearing broiler chicken by dietary inclusion of aflatoxin B1 and E-GM were worked out and given in Table 12. In order to calculate the cost of starter and finisher rations used in this experiment, the tender rate of feed ingredients for Kerala Agricultural University for the year 2006-07 was taken. The cost of basal starter and finisher rations for T1 and T2 used in this study were at Rs. 12.00. The cost of E-GM was added to the treatments T3 and T4 for arriving final feed cost for these experiments.

The cost of production per bird and total return from a bird at sixth week of age was calculated to assess the cost benefit. The cost of production included costs of starter and finisher rations and miscellaneous cost. The miscellaneous expenditure included vaccination, medication, electricity and litter cost. The birds were sold at the rate of Rs. 47 per kg live weight. Cost of poultry manure was also accounted for the total return. The net profit per kg body weight at sixth week of age was Rs. 18.13, 12.05, 17.82 and 14.37 for the treatments T1, T2, T3 and T4

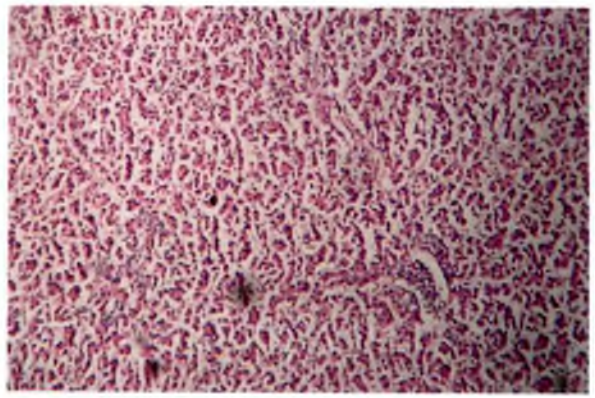


Fig 16a. Liver from Control group(T1)
Normal. H and E X 100

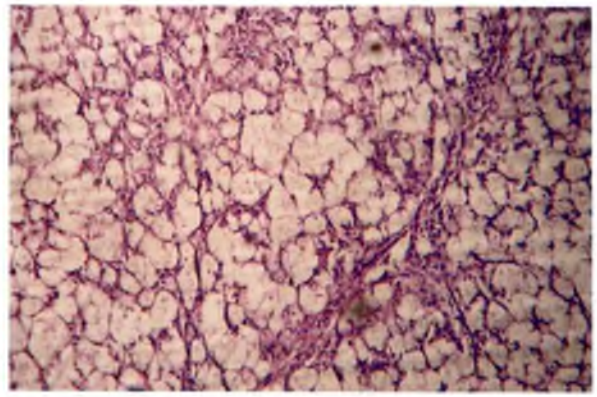


Fig 16b. Liver from aflatoxin B1 treated group (T2)
Severe fatty change and periportal fibrosis into lobules. H and E X 400

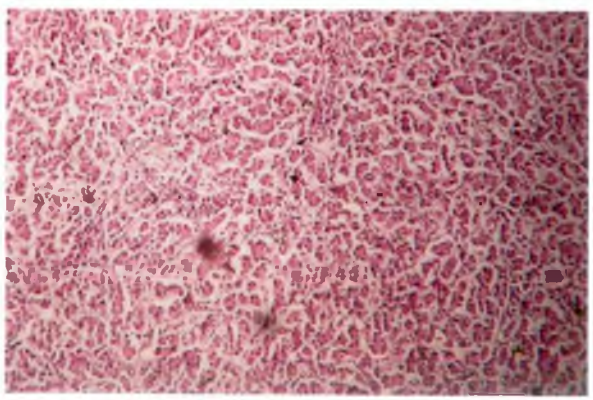


Fig 16c. Liver from E-GM alone fed group (T3)
Normal. H and E X 100

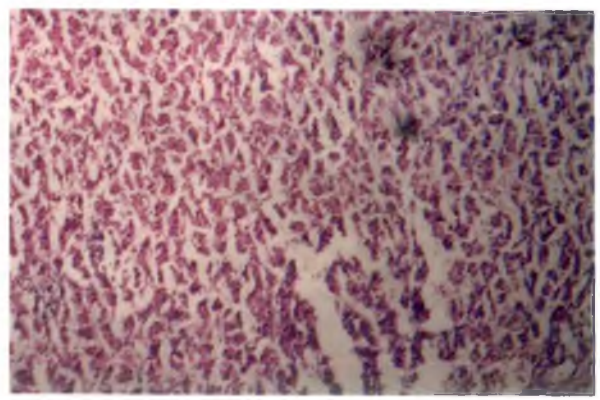


Fig 16d. Liver from aflatoxin B1 plus E-GM fed group (T4)
No fatty and fibrotic change. H and E X 100

Table 12. Cost benefit analysis as influenced by aflatoxin B1 and Esterified Glucomannan supplementation in experimental diets

Expenditure (per bird)	Unit price (Rs.)	Dietary groups							
		T1		T2		T3		T4	
		Quantity	Total Cost (Rs)	Quantity	Total Cost (Rs)	Quantity	Total Cost (Rs)	Quantity	Total Cost (Rs)
Chick cost	13.40	1.00	13.40	1.00	13.40	1.00	13.4	1	13.40
Starter feed	12.00	1773.20 g	21.28	965.79 g	11.59	1729.59 g	20.76	1174.44 g	14.09
E-GM for starter	250/ Kg	0.00%	0.00	0.00%	0.00	0.01%	0.43	0.01%	0.29
Finisher feed	12.00	2286.19 g	27.43	2006.40 g	24.08	2248.82 g	26.99	2235.23 g	26.82
E-GM for finisher	250/ Kg	0.00%	0.00	0.00%	0.00	0.01%	0.56	0.01%	0.56
Miscellaneous cost per bird	5.00	1.00	5.00	1.00	5.00	1.00	5.00	1.00	5.00
Total feed cost per bird			48.71		35.67		48.74		41.77
Total production cost per bird			67.11		54.07		67.14		60.17
RETURN									
Live birds	47.00	2218.94 g	104.29	1483.2 g	69.71	2198.29 g	103.32	1765.40 g	82.97
Manure	0.75	4059.39 g	3.04	2972.19 g	2.23	3978.41 g	2.98	3409.67 g	2.56
Total			107.33		71.94		106.30		85.53
Net profit per bird			40.22		17.87		39.17		25.36

	T1	T2	T3	T4
Net profit per kg body weight, (Rs.)	18.13	12.05	17.82	14.37
A. Margin of return over feed cost per bird (Rs.)	55.58	34.04	54.58	41.21
B. Chick cost + Miscellaneous cost (Rs.)	18.40	18.40	18.40	18.40
C=A-B (Rs.)	37.18	15.64	36.18	22.81
D=C+Manure cost (Rs.)	40.22	17.87	39.17	25.36

respectively. The net profit per kg body weight was more in the birds fed with the control diet. There was a reduction in net profit to a tune of Rs. 6.08, 0.31 and 3.76 per kg broiler chicken for the groups T2, T3 and T4 respectively from that of control. The net profit per kg body weight in group treated with aflatoxin free diets (T1 and T3) were more or less equal and markedly higher than the birds fed aflatoxin alone (T2); while the aflatoxin B1 and E-GM combination fed group (T4) had an intermediary value. The margin of return (Rs) over feed cost was highest in control (55.58) followed by T3 (54.58), T4 (41.21) and T2 (34.04) in that order.

Discussion

5. DISCUSSION

The results obtained in the study to find out the effect of supplementation of esterified glucomannan (E-GM) on alleviation of aflatoxicosis in broiler chicken are discussed in this chapter.

5.1 METEOROLOGICAL PARAMETERS

The data pertaining to microclimate inside the experimental house (Table 3) showed that the mean maximum temperature was lowest (31.14°C) during the third week of the biological trial and highest (32.57°C) during the fifth week. The mean maximum temperature ranged within a very narrow range of only 1.43°C during the trial period. Similarly, the mean minimum temperature was lowest (24.28°C) during the third week and highest (26.50°C) during the fourth week of the trial. Unlike mean maximum temperature, which showed a gradual decrease from first to third week and raised up to fifth week and again declined at sixth week of the experiment, the mean minimum temperature decreased from first week to third week and then raised during the fourth week and again declined at fifth week and then at sixth week increased to reach a temperature of 25.85°C.

The mean dry bulb temperature in forenoon (8 am) and afternoon (2 pm) were 27.1 and 31°C during the entire trial period. The dry bulb temperature at 8 am in the forenoon ranged from 24.86°C during third week to 28°C at fourth week of the experiment. In the afternoon (2 pm), the dry bulb temperature ranged from 29 (third week) to 31.14°C (fifth week). Likewise, the mean per cent relative humidity in the forenoon and afternoon during the experiment period were 63.14 and 48.00 respectively. The relative humidity (per cent) in forenoon was highest (68.71) at third week and lowest in first week (60). The relative humidity (per cent) in the afternoon was highest (55.5) at fourth week and lowest (42) at fifth week of

experiment. All these indicate that climatograph of this locality fall within the hot and humid climate.

5.2 BODY WEIGHT

In this study, broilers consuming 1mg per kg aflatoxin containing diet (T2) had significantly poor body weight than control (T1) from first week to sixth week of age. This was in agreement with the findings of Churchil (1996), who also observed a very similar result at same dietary concentration of aflatoxin. The growth depressing effect of aflatoxin in broilers has been confirmed by several earlier authors at different dose levels (Smith and Hamilton, 1970; Vasan *et al.*, 1998; Raju and Devegowda, 2000; Girish and Devegowda, 2006). Mani *et al.* (2001) reported significantly lower body weight in broilers at eight weeks of age even at a lower dose (200 ppb) of aflatoxin B1. In contrast to the above reports, Kratzer *et al.* (1969) observed that the growth of chicks fed 800 ppb aflatoxin exceeded control group. In the present study, one mg aflatoxin per kg feed depressed the fourth and sixth week body weights by 47.57 and 33.16 per cent respectively. At a very high level of 10 ppm aflatoxin, Smith *et al.* (1971) reported reduction of growth rate by about half in four weeks old commercial broilers. The most prevalent symptoms of aflatoxicosis in poultry and livestock are reduced growth rate and poor performance. The depression in body weight during aflatoxicosis might be due to hepatic cell damage and decreased pancreatic enzymes affecting digestion and to the inhibition of protein and nucleic acid synthesis.

In general, the supplementation of 0.1 per cent E-GM in aflatoxin free diet (T3) had no significant effect on body weight during all weeks except second week of age. At second week, the E-GM fed birds (T3) had significantly lower body weight than control (T1). Similar findings of no significant effect on body weight at

0.5 and 1 g/kg of yeast glucomannan have been reported earlier (Karaman *et al.*, 2005; Basmacioglu *et al.*, 2005). However, contrasting results of significant increase of 3.48 (Aravind *et al.*, 2003) and 6 per cent (Pavicic and Nemanic, 2001) in body weights of broilers at 35 and 42 days respectively are also available in the literature.

The depression in body weight caused by aflatoxin was significantly counteracted by the supplementation of 0.1 per cent E-GM from second to sixth week of age. This was in congruence with the findings Karaman *et al.* (2005), they found significant alleviation of growth depressive effect of aflatoxin (2mg/kg) by dietary inclusion of 1 g/kg esterified glucomannan at 21 days of age. Similarly, Raju and Devegowda (2000) and Girish and Devegowda (2006) reported that addition of yeast glucomannan (1 g/kg) to aflatoxin contaminated diet (0.3 and 2 mg/kg) improved the body weight of broilers at 35 days of age. At sixth week, the broilers receiving E-GM supplementation with aflatoxin contaminated diet (T4) in this study weighed 19.3 per cent more than their counterparts treated only with aflatoxin (T2). This was much higher than the earlier reports of 2.26 (Raju and Devegowda, 2000) and 6.6 per cent (Girish and Devegowda, 2006) in five weeks old broilers dosed with 0.3 and 2 ppm aflatoxin respectively. At a lower level of 0.05 per cent E-GM in 168 ppb aflatoxin contaminated diet, Aravind *et al.* (2003) reported 8.86 per cent improvement in body weight at 35 days of age. This effect of E-GM might be attributed to its mycotoxin adsorption property (Devegowda, 1997), ability to block the colonization of pathogens in the gastrointestinal tract (Olsen, 1995), immunomodulatory properties (Fernandez *et al.*, 2002) and provision of nutrients to beneficial gut flora (Newman, 1994). Though there was significant ($P<0.05$) improvement, the E-GM supplementation at 0.1 per cent level in the present study could not completely alleviate the toxic effects of aflatoxin and restore the body weight to normal throughout the experiment. The literature also

did not provide any evidence of complete alleviation of the toxic effects of aflatoxin and restoration of body weight by E-GM supplementation.

5.3 WEIGHT GAIN

Broilers fed with 1 ppm aflatoxin B1 (T2) recorded significantly poor weekly weight gain throughout the trial period. This adverse effect of aflatoxin was progressively seen from week one onwards. Similar reduction in weekly body weight gain was observed by Basmacioglu *et al.* (2005) at 21 days of age in 2 ppm level. The cumulative weight gain up to fourth as well as sixth week was also depressed significantly due to aflatoxin feeding in this study. The deleterious effects of aflatoxin on weight gain in broiler chicken have been well established by several researchers at various levels of aflatoxin (San-Gabriel, 1971; Lanza *et al.*, 1980; Huff *et al.* 1986 and Giroir *et al.*, 1991; Pimpukdee *et al.*, 2004; Bintvihok and Kositcharoenkul, 2006). In the present study, the cumulative weight gain from zero to sixth week showed a depression of 33.94 per cent at 1 ppm dietary aflatoxin. This was much higher in magnitude than the reduction of 19 and 11.71 per cent observed by Basmacioglu *et al.* (2005) and Pimpukdee *et al.* (2004) at 5 and 2 pm levels respectively. In this study, the depression from control in terms of weekly weight gain due to aflatoxin feeding was more severe in third week of age (64.10 per cent). The adverse effect of aflatoxin on body weight gain could be associated with anorexia, listlessness and inhibition of protein synthesis and lipogenesis (Oguz and Kurtoglu, 2000; Oguz *et al.*, 2000). Impaired liver functions and protein/lipid utilization mechanisms might also have affected the growth performance and general health.

Addition of E-GM to the aflatoxin free diet (T3) had no significant effect on body weight gain throughout the experimental period but for second week. Basmacioglu *et al.* (2005) also observed that addition of E-GM to aflatoxin free

diet did not produce any negative changes in 21 days old broilers compared to control. The general thought on E-GM of being an inert and nontoxic extract of *Saccharomyces cerevisiae* having no adverse impact on growth and performance of broilers has been strengthened by the results of this study.

The depression in weekly body weight gain caused by aflatoxin B1 was significantly improved by the supplementation of 0.1 percent E-GM throughout the experiment except first and third weeks. No significant change was noticed at first and third weeks of age. The depressed cumulative weight gain up to fourth as well as sixth weeks due to aflatoxin incorporation also showed significant improvement by E-GM supplementation. The beneficial effects of E-GM supplementation during aflatoxicosis on weight gain in this experiment agreed with the results of previous study performed by Basmacioglu *et al.* (2005) on 21 days old broilers at 2 ppm level. It was hypothesized that E-GM might trap the aflatoxin molecule in its glucomannan matrix and prevent toxin absorption from the gastrointestinal tract (Raju and Devegowda, 2000).

5.4 FEED INTAKE

The weekly feed intake of broilers receiving aflatoxin B1 (1 ppm) treated feed was significantly lower than that of control throughout the experimental period. This was in agreement with the findings of Raju and Devegowda (2000) they noticed significant decrease in feed intake from second week onwards up to fifth week at 0.3 ppm aflatoxin. The cumulative feed intake to fourth and sixth weeks also showed significant reduction in aflatoxin treated birds in the present study. Ghosh and Chauhan (1991) observed significant reduction of feed consumption in six weeks old broilers at the same dose of aflatoxin (1 ppm). Many researchers confirmed the deleterious effect of aflatoxin on feed intake in broiler chicken at various doses (Reddy *et al.*, 1982; Churchil, 1996; Mani *et al.*, 2001;

Bintvihok and Kositcharoenkul, 2006; Girish and Devegowda, 2006). However, Maurice *et al.* (1982) and Balachandran (1983) observed no significant difference in feed consumption in broilers at 0.65 and 0.3 ppm aflatoxin respectively. Similarly, Mani (1995) and Sudarshan *et al.* (1995) also observed no change in feed consumption in broilers at 0.5 ppm level. In the present study, the cumulative feed consumption up to six weeks showed a depression of 26.78 per cent from control. The magnitude of depression was much higher than the observations of Raju and Devegowda, (2000) (10 per cent) and Tedesco *et al.* (2004) (11 per cent) at 35 and 21 days at 0.3 and 2 ppm aflatoxin respectively. The loss of appetite in aflatoxicosis might be due to the impaired digestion by reduced digestive secretions from liver and pancreas.

The feed consumption of birds receiving E-GM in aflatoxin free diet (T3) remained unaltered in all the weeks except second week. The unchanged feed intake due to E-GM feeding was also observed by Rizzi *et al.* (2003) at 0.11 per cent in Warren hens.

The chicks supplemented with E-GM in aflatoxin containing diet (T4) showed significantly higher feed intake than the chicks receiving aflatoxin alone (T2) throughout the experimental period except at first and third weeks. The depressed cumulative feed intake up to fourth and sixth weeks due to aflatoxin was also improved significantly by E-GM supplementation in the present study. The beneficial effect of E-GM in improving the feed intake during aflatoxicosis has been reported by earlier researchers (Raju and Devegowda, 2000, Basmacioglu *et al.*, 2005; Girish and Devegowda, 2006).

The present study also revealed that the toxic effects of aflatoxin on weekly feed intake at first, fifth and sixth weeks of age was not only significantly ($P<0.05$)

counteracted but restored to normal. However, similar reports are not available in literature to support this finding. The beneficial effect on feed intake can be attributed to the selective irreversible binding ability of E-GM molecules with aflatoxin and thereby preventing its absorption (Devegowda *et al.*, 1996; Raju and Devegowda, 2000).

5.5 FEED EFFICIENCY

The results of the present study indicated that the weekly and cumulative feed efficiency in broilers which received aflatoxin was poorer than control group. This adverse effect of aflatoxin was progressively seen from second week onwards. Similar observations of poor feed efficiency with 1 ppm aflatoxin in broiler diet from zero to six weeks of age were recorded by Ghosh and Chauhan (1991) and from zero to eight weeks of age by Churchil (1996). However, Mani (1995) and Sudharshan *et al.* (1995) observed poor feed efficiency even at lower levels of 0.2 and 0.5 ppm respectively. Majority of the earlier reports agreed with the deleterious effect of aflatoxin on feed efficiency at different dose levels in broilers (Smith *et al.*, 1971; Reddy *et al.*, 1982; Swamy and Devegowda, 1998; Bintvihok and Kositcharoenkul, 2006; Girish and Devegowda, 2006). On the contrary, Shen *et al.* (1987) reported an improved feed efficiency at lower levels of 15 to 450 ppb aflatoxin. The poor feed efficiency in aflatoxin feeding could be attributed to hepatic cell damage and decreased pancreatic enzymes thereby affecting digestion and inhibition of protein synthesis.

The chicks received E-GM in aflatoxin free diet (T3) in this study had statistically similar weekly and cumulative feed efficiencies to that of control (T1) throughout the experimental period. However, on the contrary, Aravind *et al.* (2003) reported improved feed conversion ratio in broilers at 35 days of age due to esterified glucomannnan (0.05 per cent) supplementation.

The toxic effects of aflatoxin observed from second to sixth week was restored to normal by the supplementation of E-GM (1 g / kg) in the aflatoxin (1 ppm) containing feed. This was consistent with the results of Girish and Devegowda (2006), they observed that the toxic effect of 2 ppm aflatoxin on feed efficiency was reversed by supplementation of 0.01 percent E-GM. The cumulative feed efficiency from zero to four weeks in the present study showed no significant improvement. Similarly, Raju and Devegowda (2000) also reported that the feed efficiency was unaltered at 35 days of age due to E-GM supplementation in an aflatoxin containing feed. However, the cumulative feed efficiency at sixth week showed significant improvement in the present study. Similar results were reported earlier by Swamy and Devegowda (1998) in six week old broilers at 0.1 and 0.2 per cent E-GM supplementation even at lower aflatoxin concentrations of 200 and 400 ppb. At a higher dose of 2 ppm level, Basmacioglu *et al.* (2005) found partial improvement in feed efficiency by the supplementation of esterified glucomannan at 0.1 per cent level. The possible mechanism of E-GM in bringing about this beneficial effect could be attributed to the excretion of aflatoxin in an adsorbed form from the GI tract of the chicken, thereby minimizing its effects on liver and pancreas.

5.6 SERUM BIOCHEMISTRY

The results of this study revealed that the serum concentrations of total protein, albumin and cholesterol were significantly reduced and serum glucose level was increased due to feeding of 1 mg aflatoxin per kg feed. The depression in serum total protein in aflatoxicosis was well established by several earlier authors at varying levels from 0.2 to 2 ppm aflatoxin (Churchil, 1996; Vasan *et al.*, 1998; Raju and Devegowda, 2000; Mani *et al.*, 2000a; Basmacioglu *et al.*, 2005). The reduction in serum total protein might be due to the binding of aflatoxin to DNA

and thereby impairing messenger RNA synthesis and selective inhibition of the enzyme activity of RNA polymerase resulting in blockage of protein synthesis.

Similar to the present study, the reduction in serum albumin concentration at 1 ppm dietary aflatoxin was reported by Churchil (1996) in eight weeks old broilers. The depressing effect of aflatoxin on serum albumin has been proved by several earlier workers (Huff *et al.*, 1992; Smith *et al.*, 1992; Ali *et al.*, 1994; Rosa *et al.*, 2001; Basmacioglu *et al.*, 2005). In contrast, Chattopadhyay *et al.* (1985) observed no significant difference in serum albumin levels between control and treatment groups. Albumin, the aflatoxin sensitive constituent of total protein is affected much during aflatoxicosis and to lesser degree globulin level is affected.

Similar to the findings of this study, Churchil (1996) reported significant reduction in serum cholesterol level at 1 ppm aflatoxin. Vasan *et al.* (1998), Mani *et al.* (2000a) and Basmacioglu *et al.* (2005) also observed significant reduction in serum cholesterol level at 0.25, 0.2, and 2 ppm aflatoxin respectively. On the contrary, Maurice *et al.* (1982) observed elevated plasma cholesterol level in broiler chickens receiving 650 ppb aflatoxin. Reduced synthesis of cholesterol in liver due to hepatic damage might be attributed to the lower serum cholesterol level observed in this study.

The increased serum glucose level in aflatoxin treated broilers found in the present study was congruent with the observations of Mani (1995) and Mani *et al.* (2000a). The finding was however contrary to the results of Chaturvedi and Singh (2002) and Basmacioglu *et al.* (2005). The increase in the blood glucose level might be due to the impaired glucose utilization since aflatoxin has been shown to reduce the activities of enzymes involved in glucose metabolism.

In the present study, all the serum biochemical parameters studied (total protein, albumin, glucose and cholesterol) remained unaltered due to the inclusion of E-GM at 0.1 percent level in the aflatoxin free diet. The results were in accordance with the findings of Basmacioglu *et al.* (2005) for serum albumin, cholesterol and glucose but in case of serum protein they obtained significantly lower value due to E-GM supplementation.

The reduced concentrations of serum total protein, albumin and cholesterol and increased glucose level observed in this study due to aflatoxin feeding were significantly counteracted and restored to normal with the supplementation of E-GM at 0.1 per cent level. Basmacioglu *et al.* (2005) also observed that the serum parameters affected by aflatoxin (2 ppm) feeding was significantly improved by 0.1 per cent E-GM supplementation in 21 days old broilers. However, the E-GM supplementation in this study not only improved the altered serum parameters but restored them to normal. The available literature on E-GM–aflatoxin interactions in restoration of serum parameters is scanty to make any meaningful interpretations.

5.7 PROCESSING YIELDS AND LOSSES

The birds receiving aflatoxin treated feed had significantly lower dressed and eviscerated yields but significantly higher giblet yield than control. Similar reduction in eviscerated carcass yield has been reported in eight week old broilers by Churchil (1996) and Mani *et al.* (2001) at 1 and 0.2 ppm levels respectively. The reduction of 2.55 per cent of eviscerated yield noticed in this study due to aflatoxin feeding was similar to the findings of Mani *et al,* (2001), where the carcass yield was reduced from 63.58 - 64.51 to 61.09 - 62.40 per cent by 200 ppb aflatoxin feeding in eight week old broilers. The reduced carcass yield might be a sequel of general reduction in growth rate due to wasting of musculature during aflatoxicosis.

The increased giblet yield observed in this study could be attributed to the hypertropic effect of aflatoxin on liver (Smith and Hamilton, 1970), gizzard (Devegowda *et al.*, 1998; Shivachandra *et al.*, 2003a) and heart (Huff *et al.*, 1992; Stanley *et al.*, 1993). The aflatoxin feeding in this experiment had no significant effects on ready-to-cook yield, blood loss, feather loss and total loss. The reports on the effect of aflatoxin on these parameters are scanty in the literature for effective corroboration.

E-GM supplementation in the aflatoxin free diet did not show any significant changes in the processing yields and losses. However, the literature citation about the effect of E-GM alone on processing yields and losses are scanty.

Supplementation of 0.1 per cent E-GM in the aflatoxin treated feed counteracted the toxic effect of aflatoxin significantly and restored the dressed, eviscerated and giblet yields. The statistical analysis of data revealed that per cent ready-to-cook yield, blood loss, feather loss and total loss were not influenced by dietary treatments. Earlier reports on these parameters are also scanty in the literature.

5.8 RELATIVE ORGAN WEIGHT

Chicks fed aflatoxin treated feed had significantly higher relative liver, spleen and kidney weights than control. No significant difference was observed on the relative weight of bursa of Fabricious between control and aflatoxin treated group. Several workers observed increased liver weight at different levels of aflatoxin (Carnaghan *et al.*, 1966; Smith and Hamilton, 1970; Chen *et al.* 1985; Huff *et al.* 1992, Churchil, 1996, Raju and Devegowda, 2000; Girish and Devegowda, 2006). However, Shen *et al.* (1987) observed no change in liver-body weight ratio in broilers up to a dietary level of 400 ppb aflatoxin. Effect of aflatoxin

in increasing the relative weight of spleen was well established (Smith and Hamilton, 1970; Churchil, 1996; Rosa *et al.* 2001; Girish and Devegowda, 2006). The increased relative weight of kidney during aflatoxicosis was also confirmed by several workers (Chen *et al.* 1985; Huff *et al.*, 1992). In contrast to the non-significant effect of aflatoxin on bursa observed in this study, majority of the earlier workers reported reduction in bursal weight during aflatoxicosis (Smith and Hamilton, 1970; Ghosh and Chauhan, 1991; Ortatatli and Oguz, 2001; Girish and Devegowda, 2006). The enlargement of liver and kidney, the major organs in the excretion of xenobiotics indicates the degenerative damage caused to them by the aflatoxin. Enlargement of liver might also be due to the deposition of lipids in hepatocytes. The increased spleen weight might be due to lymphoid hyperplasia.

The addition of E-GM to aflatoxin free diet did not show any significant change in the mean relative weights of liver, spleen, kidney and bursa in this study. Perusal of the available literature on E-GM does not endow enough information to explain its effect on these organs in an aflatoxin free diet.

The increased relative weights of liver, spleen and kidney due to aflatoxin treatment were significantly reduced with the inclusion of E-GM in this study. The effect of E-GM in diminishing the toxic effects of aflatoxin (Girish and Devegowda, 2006) and combined mycotoxins (Raju and Devegowda, 2000; Aravind *et al.*, 2003) on these organs were well investigated. The mode of action of E-GM in decreasing the organ weights is not clear. It is thought to trap the aflatoxin molecule in its glucomannan matrix, which prevents its absorption from gastrointestinal tract and subsequently reducing the induced tissue changes. Though the relative organ yields of aflatoxin treated birds were significantly improved, the same was not restored to normal in this study.

5.9 LIVABILITY

In the present study, the aflatoxin treated birds showed higher percentage of mortality than control group from zero to six weeks of age. This was in agreement with the findings of Smith *et al.* (1971), who observed death of about one fourth of the stock when the diet contained 10 ppm aflatoxin was fed to them for four weeks. Reddy *et al.* (1982), Churchil (1996) and Shivachandra *et al.* (2003a) also reported higher mortality rate at 1 ppm aflatoxin. Mani *et al.* (1992) observed a dose related mortality in broilers fed 0 to 2 ppm aflatoxin B1. On the contrary, Giambrone *et al.* (1985) and Chattopadhyay *et al.* (1985) recorded no mortality attributable to aflatoxin feeding. Poor feeding status, impaired liver functions and protein/lipid utilization mechanisms might have affected the performance and general health. In addition, aflatoxin, a potent immunosuppressant could have affected the immunity of the birds adversely and made them susceptible to infection. All these factors might have contributed to the increased rate of mortality in aflatoxin feeding.

There was no mortality recorded in 0.1 per cent E-GM alone supplemented group and thereby the overall livability in E-GM alone (T3) supplemented group was 100 per cent throughout the experiment. However, the literature citations regarding the influence of E-GM alone on livability are scanty.

The livability of aflatoxin treated birds was slightly improved when supplemented with 0.1 per ent E-GM. Basmacioglu *et al.* (2005) also reported statistically non-significant improvement in livability of broilers at 21 days of age.

5.10 GROSS LESION

In the present study livers and kidneys of chicks fed with aflatoxin (1 ppm) treated feed were mostly swollen and pale yellow-red. The spleens were moderately enlarged while, no visible morphological changes were observed in bursa of

Fabricious. Similar enlargement and yellowish discoloration of liver and enlargement of kidneys at 1 ppm aflatoxin level was observed by Kumar (1995) and Churchil (1996). Rosa *et al.* (2001) observed friable and pale colored liver in broilers fed with 5 mg/kg aflatoxin for 30 to 52 days. Karaman *et al.* (2005) observed similar swollen and pale yellowish red livers and kidneys with enlarged spleen while, no visible morphological changes in bursa of Fabricious were observed by them in broilers at 2 mg/kg aflatoxin level. The enlargement of liver and kidney indicates the degenerative damage caused to them by the aflatoxin. Enlargement and discolouration of liver might also be due to the deposition of lipids in hepatocytes. The spleenomegaly might be due to lymphoid hyperplasia.

No gross lesions in visceral organs attributable to aflatoxicosis was recorded in 0.1 percent E-GM alone (T3) supplemented group during the entire experimental period.

Addition 0.1 percent E-GM to 1 ppm aflatoxin B1 treated group (T4) reduced the severity of macroscopic lesions in organs (liver, spleen and kidney). This agreed with the finding of Karaman *et al.* (2005). E-GM is thought to trap the aflatoxin molecule in its glucomannan matrix and prevents its absorption from gastrointestinal tract and thereby, subsequent toxin induced gross tissue changes.

5.11 HISTOPATHOLOGY

Microscopically, the liver of chicks fed with 1 ppm aflatoxin B1 containing diet (T2) shown severe fatty changes, bile duct proliferation, periportal fibrosis in periportal areas, biliary hyperplasia and accumulation of lymphoid cells within the hepatic lobules. This was in accordance with the finding of Karaman *et al.* (2005). Similar degenerative and fatty changes in hepatocytes of liver in

aflatoxin treated broilers were observed by Shivachandra *et al.* (2003a) and Rosa *et al.* (2001).

No histopathological change in liver attributable to aflatoxicosis was recorded in 0.1 percent E-GM alone (T3) supplemented birds during the entire experimental period.

Addition of 0.1 percent E-GM to 1 ppm aflatoxin B1 treated group (T4) ameliorated the effect of toxin on liver. This agreed with the finding of Karaman *et al.* (2005). The beneficial effect can be attributed to the selective irreversible binding ability of E-GM molecules with aflatoxin and thereby preventing its absorption (Devegowda *et al.*, 1996; Raju and Devegowda, 2000) and subsequent toxin-induced microscopic changes.

5.12 ECONOMICS

The net profit per kg body weight at the end of six weeks of age was Rs. 18.08, 11.98, 17.82 and 14.37 for the treatments T1, T2, T3 and T4 respectively. The net profit per kg body weight was more with the control group followed by T3 (E-GM alone), T4 (aflatoxin B1 plus E-GM) and T2 (aflatoxin B1 alone). Higher body weight at the end of six weeks of age for control group (T1) had contributed to this. The birds consumed aflatoxin contaminated diet had 33.53 per cent less net profit per kg body weight than the birds in control group. This reduction in net profit in T2 is due to significantly ($P<0.05$) low body weight compared to control at the end of six weeks. This might be the sequel to poor feeding status, altered nutrient digestion and metabolism and disturbances in protein synthesis in birds under aflatoxicosis. Arulmozhi (1999) reported minimum economic loss at 20 ppb aflatoxin level compared to higher levels (0, 20, 40, 60, 80, 100 ppb levels).

Net profit per kg body weight in E-GM alone supplemented group (T3) was marginally lower (Rs.0.31) than control (T1), which could be due to insignificant difference in body weight. From this result, the inert and nontoxic nature of E-GM having no impact on growth and economics has been confirmed.

Among the aflatoxin treated groups (T2 and T4), the net profit per kg body weight for the birds supplemented with E-GM (T4) was Rs. 2.32 more as compared to birds supplemented with aflatoxin B1 alone (T2). The reduction in net profit per kg live weight due to aflatoxin was improved from 33.53 per cent in aflatoxin B1 alone treated group to 20.74 per cent in Aflatoxin B1 plus E-GM supplemented birds.

In terms of the cost benefit analysis between the aflatoxin treated birds (T2 and T4), birds supplemented with E-GM (T4) were more economical than the birds fed with aflatoxin B1 alone (T2). The merit of adding E-GM as a toxin binder is much worthy when compared to its high profit margin from that of aflatoxin alone fed birds.

Summary

6. SUMMARY

An experiment was conducted at the Department of Poultry Science, College of Veterinary and Animal Sciences, Mannuthy to study the effect of esterified glucomannan (E-GM) on alleviation of aflatoxicosis in broiler chicken. The study was conducted for a period of six weeks with two hundred day-old straight run commercial broiler chicks. The chicks were wing banded, weighed individually and randomly divided into four dietary treatment groups viz., T1, T2, T3 and T4 with five replicates of ten chicks each. The T1 was control and the ration for which was formulated as per the BIS specifications (1992). Aflatoxin B1 (1 ppm), E-GM alone (0.1 percent) and combination of aflatoxin B1 (1 ppm) and E-GM (0.1 percent) were supplemented in the basal diet to prepare diets for T2, T3 and T4 respectively. Standard management practices were followed throughout the experimental period. Feed and water were provided *ad libitum*. The birds were provided with broiler starter ration from zero to four weeks and broiler finisher ration from five to six weeks of age.

Performance parameters such as body weight, weight gain, feed intake and feed efficiency were evaluated. The serum total protein, albumin, glucose and cholesterol were determined at the end of sixth week of age. The processing yields and losses and the weight of liver, spleen, kidney and bursa of Fabricius were also recorded at the end of sixth week of age. Livability and cost benefit were also ascertained.

Based on the results obtained during the course of the study, the following conclusions could be made.

1. The sixth week mean body weight of broilers fed toxin free basal diet (T1), diet treated with 1 ppm aflatoxin (T2), diet supplemented with 0.1 percent E-GM alone (T3) and diet supplemented with 1 ppm aflatoxin B1 plus 0.1 percent E-GM (T4) were 2218.94, 1483.20, 2198.29 and 1765.40g respectively. The aflatoxin B1 treated birds (T2) weighed significantly ($P<0.05$) lower than the control birds (T1) at the end of the experiment. Supplementation of E-GM with aflatoxin free diet had no significant effect on body weight. The toxic effect of aflatoxin on final body weight of broilers was significantly ($P<0.05$) alleviated by dietary supplementation of E-GM (T4).

2. The cumulative body weight gain of broilers up to sixth week of age was 2182.86, 1441.95, 2155.87 and 1720.62 g for treatments T1, T2, T3 and T4 respectively. Dietary inclusion of aflatoxin B1 alone (T2) significantly ($P<0.05$) depressed the weight gain in broilers. Supplementation of E-GM in the control diet (T3) had no significant ($P<0.05$) effect on body weight gain. The toxic effect of aflatoxin on final body weight gain of broilers was significantly ($P<0.05$) alleviated by dietary supplementation of E-GM (T4).

3. The mean cumulative feed intake of birds up to sixth week of age was 4059.41, 2972.19, 3978.41 and 3409.67 g for treatments T1, T2, T3 and T4 respectively. The cumulative feed intake in aflatoxin treated birds (T2) was significantly ($P<0.05$) lower than control. Dietary supplementation of E-GM alone (T3) did not reveal any change in feed intake compared with the control. Supplementation of E-GM in aflatoxin treated feed significantly improved the feed intake.
4. The mean cumulative feed efficiency of birds up to six weeks of age recorded for the treatments T1, T2, T3 and T4 were 1.86, 2.06, 1.85 and 1.98

respectively. The group fed with aflatoxin B1 alone (T2) recorded significantly ($P<0.05$) inferior feed efficiency. Addition of E-GM alone (T3) had no influence on the sixth week feed efficiency. However, addition of E-GM in aflatoxin B1 treated feed (T4) significantly ($P<0.05$) improved feed efficiency of broilers.

5. Supplementation of aflatoxin B1 in the basal diet (T2) caused significant ($P<0.05$) reductions in per cent dressed, eviscerated and giblet yields. Addition of E-GM to the basal diet (T3) had no influence on these carcass characteristics. However, addition of E-GM to aflatoxin B1 treated feed (T4) significantly alleviated the toxic effects of aflatoxin on dressed, eviscerated and giblet yields and restored them to normal. Dietary supplementation of aflatoxin B1 and E-GM either alone or in combination did not influence the per cent ready-to-cook yield, blood loss, feather loss and total loss compared to control.

6. Dietary supplementation of aflatoxin B1 (T2) increased the relative weights of liver, spleen and kidney. Feeding E-GM alone (T3) in toxin free diet had no effect on relative organ weights. Addition of E-GM to aflatoxin B1 treated feed (T4) significantly improved relative organ (liver, spleen and kidney) weights. However, no significant difference due to feeding of aflatoxin B1 and /or E-GM was observed in relative weight of bursa of Fabricius.

7. The supplementation of aflatoxin B1 alone (T2) caused significant ($P<0.05$) reduction in serum total protein, albumin and cholesterol but significant ($P<0.05$) increase in glucose level. Feeding E-GM alone (T3) in toxin free diet showed no change in serum parameters. Supplementation of E-GM to

aflatoxin B1 treated feed (T4) restored the normal values of serum parameters.

8. The dietary inclusion of aflatoxin B1 at 1 ppm level showed higher mortality rate (12 per cent) than control (2 per cent). Supplementation of E-GM alone in this study had no deleterious effect on livability. Addition of E-GM to aflatoxin B1 treated feed (T4) had improved livability up to six weeks of age.

9. The profit margin was very less in birds reared on aflatoxin B1 contaminated feed. However, addition of E-GM to aflatoxin B1 treated feed increased the profit substantially compared to aflatoxin alone treated group.

It could be concluded that the inclusion E-GM at 0.1 per cent to the aflatoxin B1 (1 ppm) contaminated feed could significantly counteract the toxic effects of aflatoxin B1 on the production parameters and relative organ weights of six week old broilers. E-GM supplementation was also found beneficial in restoring altered serum biochemical values and processing yields.

References

REFERENCES

Afzal, M. and Saleem, Z. 2004. Effects of addition of a mycotoxin detoxifier in poultry feed containing different levels of aflatoxin on the perfrmance of broilers. *Asian - Aust. J. Anim. Sci.*, 17(7): 990-994.

Ali, M.V., Mohiuddin, S.M. and Reddy, V.K. 1994. Effect of dietary aflatoxin on cell mediated immunity and serum proteins in broiler chickens. *Indian Vet. J.*, 71:760-762.

AOAC. 1990. Official Methods of Analysis. 15th edn. Association of Official Analytical Chemists, Washington, D.C.

Aravind, K.L., Patil, V.S., Devegowda, G., Umakantha, B. and Ganpule, S.P. 2003. Efficacy of esterified glucomannan to counteract mycotoxicosis in naturally contaminated feed on performance and serum biochemical and hematological parameters in broilers. *Poult. Sci.*, 82:571-576.

Arulmozhi, A., Varghese, K., Ismail, P.K. and Peethambaran, P.A. 2000. Haemato-biochemical changes in experimental aflatoxicosis in broiler chicken. *Indian J. Vet. Pathol.*, 24:143-144.

Arulmozhi, A. 1999. Determination of permissible level of aflatoxin in broiler chicken feed. M.V.Sc. Thesis, Kerala Agricultural University, Thrissur, pp. 100

Bakshi, C.S., Sikdar, A. and Chattopadhyay, S.K. 1995. Experimental aflatoxicosis in commercial broilers: Pathomorphological studies. *Indian J. Vet. Path.*, 19:112-115.

Balachandran, C. 1983. Aflatoxicosis in broiler chicken. M.V.Sc. Dissertation submitted to the Tamil Nadu Agricultural University, Coimbatore.

Basmacioglu, H., Oguz, H., Ergul, M., Col, R. and Birdane, Y.O. 2005. Effect of dietary esterified glucomannan on performance, serum biochemistry and haematology in broilers exposed to aflatoxin. *Czech J. Anim. Sci.*, 50(1): 31-39.

Bintvihok, A. and Kositcharoenkul, S. 2006. Effect of dietary calcium propionate on performance, hepatic enzyme activities and aflatoxin residues in broilers fed a diet containing low levels of aflatoxin B1. *Toxicon.*, 47(1): 41-46.

Blount, W.P. 1961. Turkey 'X' disease. *Turkeys.*, 9:52-57.

Bureau of Indian Standards (BIS). 1992. Poultry feeds-specification. Fourth revision. Bureau of Indian Standards, New Delhi. pp. 4.

Carnaghan, R.B.A., Lewis, G., Patterson, D.S.P. and Allcroft, R. 1966. Biochemical and pathological aspects of groundnut poisoning in chickens. *Path. Vet.*, 3:601-605.

Chattopadhyay, S.K., Taskar, P.K., Schwabe, O., Das, Y.T. and Brown, H.D. 1985. Clinical and Biochemical effects of aflatoxin in feed ration of chicks. *Cancer Biochem. Biophys.*, 8:67-71.

Chaturvedi, V.B. and Singh, K.S. 2002. Detoxication of aflatoxin by adsorbents. *Indian J. Anim. Sci.*,72 (10): 924-927.

Chen, C., Pearson, A.M., Coleman, T.H., Gray, J.I., Wolzak, A.M. 1985. Broiler aflatoxicosis with recovery after replacement of the contaminated diet. *Br. Poult. Sci.*, 26: 65-71.

Churchil R.R. 1996. A study on counteraction of aflatoxicosis by yeast culture in broiler chickens. M.V.Sc. Thesis, Tamil Nadu Veterinary And Animal Sciences University, Madras, 69 pp.

Council for Agricultural Science and Technology. 1989. Pages 1-91 in Mycotoxins:Economic and Health Risks. K.A. Nisi, ed. Council for Agricultural Science and Technology, Ames, IA.

Devegowda, G., Aravind B.I.R. and Morton. A. 1996. *Saccharomyces cerevisiae* and mannan oligosaccharides to counteract aflatoxicosis in broilers [abstract]. In: *Proc. Aust. Poult. Sci. Symposium,* 8:103-106.

Devegowda, G. 1997. Mycotoxins: Hidden killers in animal feeds, the search for biological solutions. In: Mulrennan, F. (ed.), Feeding Times. Dublin, Republic of Ireland.

Devegowda, G., Raju, M. V. L. N. and Swamy, H. V. L. N. 1998. Mycotoxins: Novel solutions for their counteraction. *Feedstuffs*, 70:12-16.

Devurkar, U., Devegowda, G. and Prabhudas, K. 1995. Immuno-suppression during aflatoxicosis in broiler chicken. *Indian J. Poult., Sci.*, 30:255-256.

Doerr, J.A. and Huff, W. E. 1981. Effect on young broiler chickens of combining low levels of dietary aflatoxin and ochratoxin A. *Poult. Sci.* 60(7): 1648.

Edwards, H.M. Jr. 1988. Effect of dietary calcium, phosphorous, chloride and zeolite on the development of tibial dyschondroplasia. *Poult. Sci.*, 67:1436-1446.

Espada, Y., Domingo, M., Gomez, J., and Calvo, M.A. 1992. Pathological lesions following an experimental intoxication with aflatoxin B1 in broiler chickens. *Res. Vet. Sci.*, 53:275-279.

F.A.O. 2005. www.faostat.fao.org

Fernandez, F., Hinton, M. and Gils, B.V. 2002. Dietary mannan-oligosaccharide and their effect on chicken caecal microflora in relation to *Salmonella enteritidis* colonization. *Avian Pathol.*, 31:49-58.

Gabal, M.A. and Azzam, A.H. 1998. Interaction of aflatoxin in the feed and immunization against selected infectious diseases in poultry. II. Effect on one-day-old layer chicks simultaneously vaccinated against Newcastle disease, infectious bronchitis and infectious bursal disease. *Avian pathol.*, 27:290-295.

Ghosh, R.C. and Chauhan, H.V.S. 1991. Effect of purified aflatoxin B1 on the performance of broiler chicks. *Indian J. Anim. Hlth.* 30:59-62.

Giroir, L.E., Huff, W.E., Kubena, L.F., Yersin, A.G. and Ivie, G.W. 1991. The individual and combined toxicity of Kojic acid and aflatoxin in broiler chicken. *Poult. Sci.* 70(6): 1351-1356.

Giambrone, J.J., Diener, U.L., Davis, N.D., Panangala, V.S. and Hoerr, F.J. 1985. Effects of aflatoxin on young turkeys and broiler chickens. *Poult. Sci.*, 64:1678-1684.

Girish, C.K. and Devegowda, G. 2006. Efficacy of glucomannan containing yeast product (Mycosorb®) and hydrated sodium calcium aluminosilicate in preventing the individual and combined toxicity of aflatoxin and T-2 toxin in commercial broilers. *Asian-Aust. J. Anim. Sci.*, 19(6): 877-883.

Gupta, K. and Singh, A. 2003. Experimental studies on aflatoxicosis, infectious bursal disease and their interaction in broiler chicks. *Indian J. Vet. Pathol.*, 27(1):5-7.

Huff, W.E., Kubena, D. E., Harvey, R.B., Corrier, D.E. and Mollenhauer, H.H. 1986. Progression of aflatoxicosis in broiler chicken. *Poult. Sci.* 65(10): 1891-1899.

Huff, W.E., Kubena, L.F., Harvey, R.B. and Phillips, T.D. 1992. Efficacy of hydrated sodium calcium aluminosilicate to reduce the individual and combined toxicity of aflatoxin and ochratoxin. *Poult. Sci.*, 71:64-69.

Johri, T.S., Sadagopan, V.R., Shrivastava, H.P. and Majumdar, S. 1990. Effect of dietary aflatoxin on the performance of purebred broiler chicks. *Indian J. Anim. Sci.*, 60:1246-1248.

Karaman, M., Basmacioglu, H., Ortatatli, M. and Oguz, H. 2005. Evaluation of the detoxifying effect of yeast glucomannan on aflatoxicosis in broilers as assessed by gross examination and histopathology. *Br. Poult. Sci.,* 46:394-400.

Khajarern, J. and Khajarern, S. 1999. Positive effects of Mycosorb against aflatoxicosis in ducklings and broilers. In: Poster presentation at Alltech's 15th Annual Symposium on Biotechnology in the Feed Industry, Lexington, K.Y.

Kratzer, F.H., Bandy, D., Wiley, M. and Booth, A.N. 1969. Aflatoxin effects in poultry. *Proc. Soc. Exptl. Biol. Med.*, 31: 1281-1284.

Kumar, A. A., Chanda, A.T., Rao, A.T., Bisoi, P.C. and Mishra, P.K. 1993. Clinicopathological changes in experimental aflatoxicosis in quail. *Indian J. Poult. Sci.,* 28(2): 150-153.

Kumar, R. 1995. Combined effect of aflatoxin and cyclopiazonic acid in broiler chicken. M.V.Sc., Dissertation submitted to the Tamil Nadu Veterinary and Animal Sciences University, Madras.

Lanza, G.M., Washburn, K.W., and Wyatt, R.D. 1980. Variation with age in response of broilers to aflatoxin. *Poult. Sci.,* 59: 282-288.

Ledoux, D.R., Rottinghaus, G.E., Bermudez, A.J. and Alanso-Debolt, M. 1999. Efficacy of hydrated sodium calcium aluminosilicate to ameliorate the toxic effects of aflatoxin in broiler chicks. *Poult. Sci.,* 78:204-210.

Mani, K., Narahari, D., Kumarraj, R. and Ramamurthy, N. 1992. Effect of graded levels of aflatoxin B1 in broiler chicks. *Cheiron*, 21:23-26.

Mani, K., Narhari, D., Kumararaj, R. and Ramamoorthy, N. 1993. Influence of dietary aflatoxin B1 on certain haematological and biochemical characters of broiler chicken. *Indian Vet. J.*, 70:801-804.

Mani, K. 1995. Studies on the effect of aflatoxin B1 on the immune status of commercial broilers. Ph.D. Thesis, Tamil Nadu Veterinary And Animal Sciences University, Madras, pp. 96

Mani, K., Sundaresan, K. and Viswanathan, K. 2000a. Performance of commercial broiler strains under experimental aflatoxicosis. *Indian J. Poult. Sci.*, 35 (2): 176-180.

Mani, K., Sundaresan, K. and Viswanathan, K. 2000b. Effect of dietary aflatoxin B1 on the blood constituents in commercial broilers. *Indian Vet. J.*, 77:993-995.

Mani, K., Sundaresan, K. and Viswanathan, K. 2001. Effect of immunomodulators on the performance of broilers in aflatoxicosis. . *Indian Vet. J.*, 78:1126-1129.

Mashaly, R.D., Salem, M.H., Mahoud, Z.H., Leeb, S.A., Sharani, G. and Ismail, A.A. 1986. Effects of aflatoxin on body weight gain and on protein and RNA synthesis in chicken. *Indian J. Anim. Sci.* 56(6): 698-702.

Maurice, D.V., Rehrer, N.J. and Bodine, A.B. 1982. Metabolic effects of low levels of aflatoxin B1 in broiler chicks. *Poult. Sci.*, 61:1387.

Mohiuddin, S.M., Vikram Reddy, M., Madhava Reddy, M. and Ramakrishna, K. 1986. Studies on phagocytic activity and haematological changes in aflatoxicosis in poultry. *Indian Vet. J.* 63:442-445.

Mundas, S. and Rao, D.G.K. 2001. Histological changes in liver by aflatoxin and fenvalerate treated broiler birds. *Indian Vet. J.*, 78: 1152-1153.

Murthy, T.N.K. and Devegowda, G. 2004. Efficacy of modified glucomannan (Mycosorb) to adsorb aflatoxin B1 in gut conditions of broiler chickens. [abstract] In: Poster presentation at XXII World's Poultry Congress, Istanbul, 8th to 13th June.

Nath, R., Bisoi, P.C., Mohapatra, M. and Mishra, S.C. 1995. Effect of aflatoxin on serum constituents and enzymes. 17'th Annual Conference of Indian Poultry Science Association abstracts, 0403.

Newman, K.E. 1994. Mannan oligosaccharide, natural polymers with significant impact on the gastrointestinal microflora and immune system. In: Lyons, T. P. and Jacques, K.A. (eds.), Biotechnology in the feed industry. Alltech Inc., Kentucky. pp. 167-174.

Oguz, H. and Kurtoglu, V. 2000. Effect of clinoptilolite on fattening performance of broiler chickens during experimental aflatoxicosis. *Br. Poult. Sci.*, 41:512-517.

Oguz, H., Kececi, T., Birdane, Y.O., Onder, F. and Kurtoglu, V. 2000. Effect of clinoptilolite on serum biochemical and haematological characters of broiler chickens during experimental aflatoxicosis. *Res. Vet. Sci.*, 69:89-93.

Olsen, R. 1995. Mannan oligosaccharides: Experience in commercial turkey production. In: Lyons, T. P. and Jacques, K.A. (eds.), Biotechnology in the feed industry. Nottingham University Press, Loughborough, Leics, UK. pp. 389-392.

Ortatatli, M. and Oguz, H. 2001. Ameliorative effects of dietary clinoptilolite on pathological changes in broiler chickens during aflatoxicosis. *Res. Vet. Sci.*, 71: 59-66.

Pavicic, P. and Nemanic, A. 2001. Influence of esterified glucomannnan (MycosorbReg.) on performance parameters of broiler chickens [abstract]. In: Proceedings of the 4th symposium of poultry-days-porec, 16 to 19 May, pp.151-154.

Pimpukdee, K., Kubena, L.F., Baily, C.A., Huebner, H.J., Afriyie-Gyawu, E. and Phillips, T.D. 2004. Aflatoxin induced toxicity and depletion of hepatic vitamin A in young broiler chicks: Protection of chicks in the presence of low levels of Novasil Plus in the diet. *Poult. Sci.*, 83:737-744.

Prabaharan, S. and George, V.T. 1999. Effect of aflatoxin and *Eimeria tenella* infection on mortality and lesion score in broiler chicken. *Indian Vet. J.*, 76:693-695.

Raju, M.V.L.N., and Devegowda, G. 2000. Influence of esterified-glucomannan on performance and organ morphology, serum biochemistry and haematology in broilers exposed to individual and combined mycotoxicosis (aflatoxin, ochratoxin and T-2 toxin). *Br. Poult. Sci.*, 41: 640-650.

Raju, M.V.L.N. and Devegowda, G. 2002. Esterified glucomannan in broiler chicken diets-contaminated with aflatoxin, ochratoxin and T-2 toxin: evaluation of its binding ability (in vitro) and efficacy as immunomodulatory. *Asian - Aust. J. Anim. Sci.*, 15:1051-1056.

Raina, J.S., Roy, K.S. and Singh. 1991. Biochemical and histochemical studies in experimental mycotoxicosis in chicks. *Indian J. Anim. Sci.*, 62:1276-1281.

Reddy, A. R., Reddy, V.R., Rao, P.V. and Yadagiri, B. 1982. Effect of experimentally induced aflatoxicosis on the performance of commercial broiler chicks. *Indian J. Anim. Sci.*, 52:405-410.

Rizzi, L., Zaghini, A. and Roncoda, P. 1998. Aflatoxin B1 oral administrations to laying hens: Efficacy of modified mannanoligosaccharide (Mycosorb) to prevent mycotoxicosis. In: Lyons, T. P. and Jacques, K.A. (eds.), Biotechnology in the feed industry. Nottingham University Press, Loughborough, leics, UK.

Rizzi, L., Simioli, M., Altafini, A and Zaghini, A. 2003. Effect of esterified glucomannan on laying hens exposed to combined mycotoxins (aflatoxin B1, zearalenone and fumonisin B1). *Italian J. Anim. Sci.*, 2 (suppl. 1): 465-467.

Romer. 1975. Qualitative/quantitative analysis for detection and estimation of aflatoxin (Romer mini column method). *J. Assoc. Off. Anal. Chem.* 58:500-506.

Rosa, C.A.R., Miazzo, R., Magnoli, C., Salvano, M., Chiacchiera, S.M., Ferrero, S., Saenz, M., Carvalho, E.C.Q. and Dalcero, A. 2001. Evaluation of the efficacy of bentonite from the South Argentina to ameliorate the toxic effects of aflatoxin in broilers. *Poult. Sci.*, 80:139-144.

San-Gabriel, C.A. 1971. Pathology of broiler production aflatoxins: Histological lesions they produce. *Revista-del-patronate-de-Biologia-Animal.* 4:369-386.

Shen, T.F., Yen, C.C. and Kuo, C.C. 1987. The effect of low levels of aflatoxin in the diet on the performance of broilers. *J. Chin. Soc. Anim. Sci.*, 16:63-71.

Shivachandra, S.B., Sah, R.L., Singh, S.D., Johri, T.S. and Manimaran, K. 2003a. Patho biochemical and Immunological changes in experimental aflatoxicosis. *Indian J. Poult. Sci.*, 38 (2): 137-141.

Shivachandra, S.B., Sah, R.L., Singh, S.D., Kataria, J.M., Johri, T.S. and Manimaran, K. 2003b. Haemato-biochemical changes in aflatoxin fed broiler chicks infected with Hydropericardium syndrome. *Indian J. Anim. Sci.*, 73(4): 390-391.

Shotwell, O.L., Hesseltine, C.W., Stubblefield, R.D. and Sorenson, G.W. 1966. Production of aflatoxin on rice. *Appl. Microbiol.*, 14:425-428.

Smith, J.W. and Hamilton, P.B. 1970. Aflatoxicosis in broiler chicken. *Poult. Sci.*, 49:207-215.

Smith, J.W., Hill, C.H. and Hamilton, P.B. 1971. The effect of dietary modifications on aflatoxicosis in the broiler chicken. *Poult. Sci.*, 50:768-774.

Smith, E.E., Kubena, L.F., Braithwaite, C.E., Harvey, R.B., Phillips, T.D. and Reine, A.H. 1992. Toxicological evaluation of aflatoxin and cyclopiazonic acid in broilers. *Poult Sci.*, 71:1136-1144.

Snedecor, G.W. and Cochran, W.G. 1994. *Statistical Methods* Eighth Edition, Afiliated East-West Press Pvt. Ltd, New Delhi, India. 313 pp.

Stanley, V.G., Ojo, R., Woldesenbet, S., Hutchinson, D.H. and Kubena, L.F. 1993. The use of *Saccharomyces Cerevisiae* to suppress the effect of aflatoxicosis to suppress the effect of aflatoxicosis in broiler chicks. *Poult. Sci.*, 72:1867-1872.

Sudarshan, C., Gowdh, C.V., Devegowda, G. and Aravind B.I.R. 1995. Efficacy of live yeast and *Lactobacillus* on performance of broilers fed aflatoxin. 17th Annual Conference of Indian Poultry Science Association Abstracts. 0608.

Swamy, H.V.L.N. and Devegowda, G. 1998. Ability of mycosorb to counteract aflatoxicosis in commercial broilers. *Indian J. Poult. Sci.*, 33 (3): 273-278.

Tedesco, D., Steidler, S., Galletti, S., Tameni, M., Sonzogni, O. and Ravarotto, L. 2004. Efficacy of silymarin-phospholipid complex in

reducing the toxicity of aflatoxin B1 in broiler chicks. *Poult. Sci.*, 83: 1839-1843.

Vasan, P., Ravi, R. and Purushothaman, M.R. 1998. Effect of graded levels of aflatoxin (AFB1) on performance of broiler chicks. *Indian J. Poult. Sci.*, 33 (2): 214-216.

EFFECT OF ESTERIFIED GLUCOMANNAN ON ALLEVIATION OF AFLATOXICOSIS IN BROILER CHICKEN

MALDHURE NITEEN ARVIND

Abstract of the thesis submitted in partial fulfilment of the requirement for the degree of

Master of Veterinary Science

Faculty of Veterinary and Animal Sciences
Kerala Agricultural University, Thrissur

2007

Department of Poultry Science
COLLEGE OF VETERINARY AND ANIMAL SCIENCES
MANNUTHY, THRISSUR-680651
KERALA, INDIA

ABSTRACT

An experiment was conducted at the Department of Poultry Science to investigate the effect of esterified glucomannan (E-GM) on alleviation of aflatoxicosis in broiler chicken. Day old broiler chicks numbering two hundred were reared under four different treatments with five replicates of ten chicks each. The four dietary treatments were T1 (control), T2 (1 ppm aflatoxin B1), T3 (0.1 per cent E-GM) and T4 (1 ppm aflatoxin B1 plus 0.1 per cent E-GM). Chicks were reared under standard managemental conditions up to six weeks of age. The broiler starter ration was fed from zero to four weeks and finisher ration from five to six weeks of age.

Results of the study revealed that, inclusion of aflatoxin B1 (1 ppm) adversely affected the weekly body weight, weight gain, feed consumption, feed efficiency and overall livability during the experimental period. Serum concentrations of total protein, albumin and cholesterol in broilers fed 1 ppm aflatoxin B1 were significantly ($P<0.05$) depressed whereas; serum glucose level was significantly ($P<0.05$) elevated than control. Aflatoxin B1 caused significant increase in relative weights of liver, spleen and kidney. No significant difference existed between treatments for relative weights of bursa of Fabricius. The per cent dressed and eviscerated yields were significantly ($P<0.05$) decreased whereas giblet yield was significantly ($P<0.05$) increased in aflatoxin treated group. The per cent ready-to-cook yield, blood loss, feather loss and total loss were not influenced by dietary supplementation of aflatoxin B1.

Supplementation of E-GM to toxin free diet caused no significant difference in the weekly body weight, weight gain, feed consumption, feed efficiency, relative

organ weights, serum parameters and processing yields compared to control. Livability was better in T3 than control during the experimental period.

Inclusion of E-GM in the aflatoxin B1 treated diet significantly counteracted the toxic effects of aflatoxin B1 on final body weight, cumulative weight gain and feed consumption and feed efficiency up to sixth week of age. Decreased level of serum total protein, albumin and cholesterol and increased glucose level due to aflatoxin feeding was restored to normal level. The altered relative weights of liver, spleen and kidney due to aflatoxin feeding were significantly improved by E-GM supplementation. The per cent eviscerated, dressed yield and giblet yield were restored by supplementation of E-GM to aflatoxin B1 treated feed. However no effect was observed on ready to cook yield, blood loss, feather loss and total loss compared to other treatments. The reduction in net profit per kg body weight caused by dietary aflatoxin was increased substantially by supplementation of E-GM to the contaminated feed.