EFFECT OF SELECTED SUPPLEMENTAL FEED ADDITIVES ON PRODUCTION PERFORMANCE AND LIVER HISTOLOGY OF LAYING CHICKENS FED MOULDY DIETS.

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Dr. T. O. Akande. Date

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DEDICATION

I dedicate this thesis to God Jehovah, the same yesterday, today and forever, my mother and my husband for their unwavering support during the course of my study and research.

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ABSTRACT

The studydetermined the effect of dietary supplemental retinol, tocopherol, a multi-vitamin and a commercial adsorbent on the production performance and liver histology of laying chickens fed mouldy diets and assessed the toxicity of mouldy diets on liver of birds with a view to ameliorate the toxic effects of mould contamination in feed for laying chickens.

The experimental birds were completely randomized into six treatments in three replicates of 10 birds each. The control group was fed Aflasafe maize diet while birds in treatments two were fed mouldy maize diets without supplement, treatment three were fed mouldy maize diet supplemented with retinol, treatment four were fed mouldy maize supplemented with tocopherol, treatment five were fed mouldy maize supplemented with a multi-vitamins (vitalyte) while treatment six were fed mouldy maize diet supplemented with a commercial adsorbent (activated charcoal) supplement respectively.

Results of chemical composition of test samples showed that crude protein and crude fat reduced by 11.54% and 12.72% respectively in the mouldy maize, crude fibre content was higher by 31.7% in the mouldy maize and there was a substantial depreciation in both retinol and tocopherol contents of the mouldy maize and diets compared to the control. Similarly the aflatoxin content rose from near zero in aflasafe maize grains to 267µg in mouldy maize and from 0.78µg in aflasafe diet to 118µg in the mouldy diet. In terms of performance, the control group had higher ($P<0.05$) body weight gains, hen day production, feed conversion efficiency and the highest feed cost/kg egg. The vitamins supplemented groups were similar ($P>0.05$) to the control in terms of performance except for their lower feed cost/kg egg with treatment three

having the lowest feed cost/kg egg among the additive supplemented groups. Egg quality differs ($P<0.05$) only in terms of shell thickness, shell weight and yolk colour. The commercial adsorbent had superior values ($P>0.05$) over vitamin supplements in term of shell thickness and compare more closely with the control while significantly low values ($P<0.05$) were obtained for birds on mouldy non- supplemented diet. Liver histology photomicrographs showed that supplementation with vitamins showed some ameliorative tendencies of aflatoxicosis as evidenced by essentially normal architecture of the hepatocytes particularly in the multi- vitamin supplemented group which was quite similar to the aflasafe control. Cellular infiltration, congestion, and hypercellularity or dilation of the sinuosoidal spaces characterized liver of bird fed on mouldy diets.

The study concluded that feeding mouldy diets supplemented with retinol, tocopherol and vitalyte is safe for poultry birds although mouldy feed should be prevented as much as possible in layer chicken production.

CHAPTER ONE

1.0 INTRODUCTION

1.1 BACKGROUND OF THE STUDY

Feed spoilage is caused by undesirable moulds and bacteria. Moulds are filamentous fungi ever-present in many feedstuffs including roughages and concentrates (Whitlow and Hagler, 2007). The rapid growth of moulds heats up feed which reduces the energy and vitamins available to the animal. Mouldy feeds also tend to be dusty making them less palatable (Morgan, 2012). The growth of moulds on grain can affect the nutritional quality of grain in several ways; firstly, moulds reduce the nutritional value of the commodity as they consume fats, protein and carbohydrates that are present in the grain this is because moulds utilize nutrients in grains for their own metabolism and propagation thus reducing its nutritional value and so these nutrients are no longer available to the animal (Whitlow and Hagler, 2005). Secondly, some species of moulds are able to produce highly toxic compounds called mycotoxins (Marquardt, 1996).

Mycotoxins are biologically active, toxic metabolites produced by toxigenic fungi mainly belonging to Aspergillus, Fusarium and Penicillium species, which invade crops in the field and may grow on food during storage under favourable conditions of temperature and humidity (Shamsudeen *et al.,* 2013). Mycotoxins as secondary metabolites are produced by filamentous fungi that cause a toxic response (mycotoxicosis) when ingested by higher animals. Mycotoxins are toxic to humans and animals and this explains the major concern of food and feed industries in preventing them from entering the food chain. Toxin-producing moulds may invade plant

material in the field before harvest, during post-harvest handling and storage and during processing into food and feed products (Pierre, 2007).

At the global level, it is considered that 25% of the world crop production are contaminated by mycotoxins, which may be a risk factor affecting human and animal health (Bouhet and Oswald, 2005; Weaver *et al.,* 2013). Like other environmental pollutants, mycotoxins also adversely affect the health and productivity in animals and poultry (Zain, 2011; Katole *et al.,* 2013). The economic impact of mycotoxins includes increased mortality, increased veterinary care costs, reduced livestock production and total disposal of contaminated foods and feeds (Goossens *et al.,* 2012). However, the major problem associated with animal feed contaminated with mycotoxins is not only acute disease episodes, but also the ingestion of low level of toxins which may cause an array of metabolic, physiologic, and immunologic disturbances (Greinier and Applegate, 2013).

It is observed that mycotoxins occur frequently in a variety of feedstuffs and these feeds are routinely fed to animals. Several efforts have been made to decontaminate or control them by the use of physical and chemical strategies but the success made so far is limited (Shetty and Jespersen, 2006, Zaki *et al*., 2012). Most of the current procedures were reported to be impracticable, cost ineffective and sometimes compromise nutritive value of feed (Patil *et a*l., 2006; Pfohl-Leszkowicz and Menderville, 2007; 2012). Chemical processes have also been used in transforming mycotoxins into other less toxic compounds in feed. However, these processes have been reported to only change the structures of selected mycotoxins but not necessarily reduce their inherent toxicity (He *et al.*, 2010).

Both the pathological and clinical signs of mycotoxicosis resemble the signs of vitamin deficiencies in chickens (Bains, 2007). The vitamin deficiency signs are the direct result of

incomplete or absence of specific vitamin participation in the biochemical and metabolic pathways necessary to utilize nutrients and maintain tissue integrity. Mycotoxins such as aflatoxins are known to interfere with absorption of dietary vitamins such as retinol, cholecalciferol, tocopherol and phylloquinone, Aflatoxins also decrease the production of vitamin A in the liver, and it has secondary effects such as decreased blood calcium levels, decreased bone strength, and a decreased tissue and serum tocopherol level (Bains, 2007). This decrease in absorption of dietary vitamins can lead to Vitamin A and E deficiencies (Bains, 2007). In broiler chickens, mouldy corn has been associated with deficiencies of vitamins D (rickets) and vitamin E (Encephalomalacia) despite the fact that these vitamins were supplemented at levels regarded as satisfactory (Mcdowell, 2004, Bains, 2007). The mechanism of mycotoxin induced tissue and organ damageis the result of complex biochemical processes that may react with other enzymes and cofactors. The absorption and bioconversion of vitamins may be adversely affected due to cell membrane damage caused by mycotoxins and their metabolites in organs such as gastrointestinal tract, liver or the kidneys (Bains, 2007).

Studies have indicated a positive response of vitamin supplementation to improve specific parameters that are of economic importance in poultry. Thus, under field conditions, supplementation of vitamins is frequently practiced to alleviate clinical signs caused by mycotoxins and to improve commercial parameters (McDowell *et al.,* 2007; Bains 2007). Nutritional approaches, such as supplementation of nutrients or additives with protective properties against mycotoxin toxicity, are also assuming increasing interest. These compounds exhibit their protective effects without actual interaction with mycotoxin molecules. Selenium, some vitamins (A, C and E) and their precursors have marked antioxidant properties that act as superoxide anion scavengers. Thus, these compounds have been demonstrated by *in vitro* and *in*

vivo studies to counteract some of the toxic effects of mycotoxins. Feed additives like antioxidants, some amino acids, vitamins, phenolic compounds, trace elements and plant extracts can be useful to reduce the toxic effects observed in animals (Dvorska *et al.*, 2007).

Vitamin E acts as an antioxidant and prevents peroxidation. Free radicals are formed primarily in the body during normal metabolism and also upon exposure to environmental factors. Fats are vulnerable to destruction through oxidation by free radicals and vitamin E, α-tocopherol, is uniquely suited to intercept free radicals and thus prevent a chain reaction of lipid destruction. Aside from maintaining the integrity of cell membranes throughout the body, α-tocopherol also protects the fats in low-density lipoproteins (LDLs) from oxidation (Traber *et al.*, 2006). Similarly, Vitamin A (Retinol) functions as a non-enzymatic anti-oxidizing agent that has capacity to protect cells from oxidative stress-induced toxicity and transformation (Kubena and McMurray, 1996; Cramer *et al.*, 2001; Nicolle *et al.*, 2003). Vitamin A can therefore be recommended as a supplement in poultry diets as it is readily available in many forms including inactive pro-vitamins like carotenes (Muzaffer *et al.*, 2003).