EFFECT OF METHANOL EXTRACT OF PARKIA BIGLOBOSA ROOT BARK ON ORGAN AND CARCASS WEIGHT AND HISTOPATHOLOGICAL CHANGES IN EIMERIA TENELLA INFECTED BROILER CHICKENS

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ABSTRACT

The study evaluated the effect of methanol extract of Parkia biglobosa root in Eimeria tenella infected broilers. One hundred and fifty broilers grouped into six treatment groups were used for the study as follows: uninfected treated with Sulfadoxinoxaline (A), uninfected treated with extract (B), uninfected untreated; (control) (C), infected untreated (D), infected treated with Sulfadoxinoxaline (E), infected treated with extract (F). Each broiler was infected at 28 days of age with 20,000 infective E. tenella oocysts and treated at day 7 post infection at a dose rate of 250 mg/ml in drinking water for 7 days. Carcass and organ weights were recorded at 35 days of age. There was significant difference ($p<0.05$) between the treated and untreated groups. In groups E and F, the gross findings showed a thickened and hemorrhagic caecae in 3 and 4 broilers each while in group D, 2 broilers had their caecae ballooned and filled with blood and tissue debris. Gross lesions were not observed in groups A, B and C. In group F histopathology showed that the lymphoid tissues of the Bursa of fabricius were highly reduced and the liver in groups D, E and F showed slight lymphocytic infiltration. No mortality was recorded. This study proved that the root bark of P. biglobosa has no harmful effects on organs of treated broilers, because P. biglobosa extract appeared to have improved carcass weight and significantly reduced clinical signs of coccidiosis such as blood stained droppings consistent with E. tenella infection in broilers.

Keywords: Broilers, Carcass, Coccidiosis, Eimeria tenella, Organs, Parkia biglobosa, Weight

INTRODUCTION

Coccidiosis is one of the most economically important prevalent parasitic diseases which has adversely affected the growth and fortune of poultry farmers’ worldwide (Gharekhani et al., 2014). Coccidiosis affects virtually all domestic and wild bird species, causing signs such as paleness, diarrhea with or without blood, poor feed conversion, weight loss, and in severe cases even death. Eimeria, a protozoan parasite of the Phylum Apicomplexa is considered the main risk to avian production since they are the causative agent of avian coccidiosis (Blake et al., 2015). Avian coccidiosis is caused by seven species of Eimeria, which parasitize chickens intestine. According to a recent estimate, the global cost of the protozoan to the poultry industry is estimated to be over USS 2.4 billion per annum (Ola-Fadunsin and Ademola, 2013).

In Nigeria, the disease is caused by Eimeria tenella, E. necatrix, E. bruneti, E. acervulins, E. mitis and E. praecox (Jatau et al., 2012). These species have predilection sites in
the different parts of gastrointestinal tract. *E. acervulina* occurs in the epithelial cells of the anterior portion of the small intestine mainly in duodenum. *E. bruneti* occurs in the mucosa of the lower portion of the small intestine, caecum, rectum and cloaca. *E. tenella* is present in caecum. *E. necatrix* occurs in the jejunum, mid gut, caecum and other parts of the large intestine (Kant *et al.*, 2013).

The management involves the use of ionophorous compounds (ionophores) and synthetic drugs (chemicals). Ionophores usually cause the death of parasite and are produced by the fermentation process. The chemicals inhibit several biochemical pathways of the parasite (Chapman and Jeffers, 2014). However, the increased occurrence of resistance against routine anticoccidial drugs, the toxic effects of these chemicals on poultry and the development of drug residues in poultry meat and egg (Goetting *et al.*, 2011) have left the poultry industry with a renewed challenge for coccidiosis prevention and control. These have propelled the search for alternative strategies among which plant products provide a good alternative. Also alternative feed additives and drugs to promote poultry production, carcass yield and quality as well as the health of birds is being sourced (Adegbola, 2004). In addition, the use of recombinant vaccines is limited mainly due to the low protection of antigens with the potential to induce potent protective immune response against *Eimeria* species (Quiroz-Castaneda, 2018).

The use of herbal remedies which are cheap and easy to procure becomes a desired alternative. Herbal remedies have the advantage of being of natural origin, could make the control of coccidian parasite cheaper and may also be a source of foreign revenue. The use of plants in the treatment and control of coccidiosis is not a new development. Recently, *Bidens pilosa* was used in diet of birds, and it significantly elevated body weight gain and lowered feed conversion ratio. Also, *B. pilosa* reduced cecal damage, villi destruction and decreased villus-to crypt ratio in chicken ceca (Chang *et al*. 2016). The stem bark of *P. biglobosa* has been reported to have some coccidiostatic activities (Maikai *et al.*, 2007) and antimalarial effects (Traore *et al.*, 2013). The stem bark is used for treating diabetes mellitus and hypertension (Karou *et al.*, 2011). It is listed as having wound healing properties in South-Western Nigeria (Adetutu *et al.*, 2013). A decoction of the root is used in the treatment of coccidiosis by natives of western Nigeria (Olanipekun, 2014). There is no data on the effect and anticoccidial activity of the root bark extract of *P. biglobosa* in broiler chickens. This study examined the beneficial and histopathological effects of methanol extract of *P. biglobosa* root bark in broiler chickens experimentally infected with *Eimeria tenella* oocysts.

**MATERIALS AND METHODS**

**Experimental Birds:** The study was carried out at the Farm Unit, Poultry section of the Department of Animal Health and Production, National Veterinary Research Institute, Vom, Nigeria. A total of 150 four-week commercial broilers (Cobb 500, Zartech Limited, Jos, Nigeria) were used for the experiment. The broilers were routinely vaccinated against Newcastle and Infectious bursal diseases at 2 weeks and 4 weeks concurrently.

**Housing and Management of Broilers:** The broilers were kept in standard battery cages. Each group was kept in a separate cage. The broilers were fed from day old to 28 days with a pelleted Vital Feed broiler starter feed and from 28th day to the end of the study with broiler finisher (Vital Feed) produced by Grand Cereals and Oil Mills Company Limited, Jos. Feed and clean water were provided *ad libitum*.

**Experimental Plant:** The root of *Parkia biglobosa* was obtained from Barkin Ladi, southern area of Jos, Plateau State, Nigeria. The specimen was identified in the herbarium section of the Botany Department, University of Jos, Nigeria, where a voucher specimen (Kujul 54, 2018) was deposited. The root barks were obtained by peeling them off the roots and dried under shade in the Laboratory until a constant weight was obtained at the National Veterinary Research Institute, Vom, Nigeria (NVRI). The
dried specimen was pulverized using hammer mill to a fine powder. The fine powder was macerated in absolute methanol at room temperature for 72 hours. The mixture was stirred daily for 3 days and filtered using Whatman Number 1 filter paper. The resultant liquid was concentrated by evaporating the solvent at 75°C using a rotatory evaporator to get the dark brown extract which was stored in a refrigerator until used.

Experimental Organism (Eimeria tenella Oocysts): The infective Eimeria oocysts suspension used in this study was obtained from the National Veterinary Research Institute (NVRI) Laboratory, Vom, Nigeria.

Ethical Standards: The experiments were conducted in accordance with the ethical guidelines contained in the guide for the care and use of laboratory animals (NRC, 2010). The experimental protocol was approved by the Institutional Animal Care and Use Committee (IACUC) Protocol, Faculty of Veterinary Medicine, University of Nigeria, Nsukka.

Experimental Design: The 150 broiler chickens (Cobb 500) were weighed and assigned by weight into six treatment groups A, B, C, D, E and F. Each treatment was replicated five times with five broilers in each replicate. Each replicate was placed in a separate pen. The groups were infected and treated as follows: Group A – Uninfected with E. tenella sporulated oocysts and treated with Sulfadiazine at the rate of 250 mg/ml in drinking water for 7 days (uninfected / sulfadiazine treated – Standard Control); B – Uninfected with E. tenella sporulated oocysts and treated with P. biglobosa extract at the rate of 250 mg/ml in drinking water for 7 days (uninfected / P. biglobosa treated); C – Uninfected with E. tenella sporulated oocysts and untreated with either P. biglobosa extract or sulfadiazine (uninfected / untreated – Negative Control); D – Infected with E. tenella sporulated oocysts at the rate of 20,000 sporulated oocysts/ml per broiler orally and treated with sulfadiazine at the rate of 250 mg/ml in drinking water for 7 days (infected / sulfadiazine treated – Standard reference); F – Infected with E. tenella sporulated oocysts at the rate of 20,000 sporulated oocysts/ml per broiler orally and treated with P. biglobosa extract at the rate of 250 mg/ml in drinking water for 7 days (infected / P. biglobosa treated). The broilers were fed broiler finisher (Vital Feed) and clean drinking water throughout the remaining period of the study (14 days). The total period of the study from day old broilers to the end of the study was 42 days.

Induction and Confirmation of Coccidiosis in Broilers: The broilers were screened for the presence of Eimeria oocysts two days before the experimental infection and were found to be free from natural infection. Groups A – C were not infected with E. tenella oocysts, while D – F were infected with 20,000 sporulated oocysts/ml per broiler orally. Broilers of each replicate group were housed in a separate pen. Establishment of infection was monitored by clinical signs, faecal oocyst counts, gross and microscopic lesions. Coccidiosis was confirmed on the 7th day post infection (P.I) in all the three infected groups of 25 broilers each, totaling 75 broilers.

Determination of P. biglobosa Effects on the Broilers: The average live body weight (ALBW), weight gain (WG), carcass/dressed weight (CDW), organ and live body weight ratio (OLBWR) and other portions were measured and recorded weekly and used to determine the growth and effect of the treatment on the broilers (Kiczorowska et al., 2016). The broilers were also observed for any changes in health by visually observing clinical signs, morbidities and mortalities.

Determination of Live Body Weights: The broilers were all weighed with a top loading balance (Camry Emperors, China) on day zero of the experiment, at 28 days of age and
subsequently every 3 days till the end of the study. Measurement of organs and carcass weights were carried out using a sensitive weighing balance (Mettler-Toledo, LLC Electronic Weighing Balance, Columbus). The variations in weights of organs were expressed as percentages of live weights so as to exclude any possible effect due to damage to tissue or organ as a result of *E. tenella* infection. The weekly average live body weights (ALBW) of the broilers were determined by subtracting respective bird initial weight ($W_1$) (kg) from the final broiler weight (kg) ($W_2$) divided by the number of weeks ($n$); ($W_2 - W_1/n$). The organ and live body weight ratio (%) was obtained by dividing the respective organ weight by ($W_2 - W_1/n \times 100$).

**Clinical Signs:** The birds were monitored strictly for development of clinical signs, such as dullness, diarrhea, clustering, anorexia, drooping of wings and mortality throughout the course of the experiment starting on the second day after infection.

**Faecal Oocysts Counts:** Faecal samples were collected daily from each group of the experimental birds, from the third day following infection of the broilers and analyzed for the presence of oocysts using the floatation technique. The modified McMaster technique was adopted when more than 100 oocysts were present (Majaro, 1993). The first appearance of oocysts in any group was scored as the prepatent period. The oocyst counts were conducted every day from day 3 of the infection till patency was established and thereafter every 3 days till the expiration of the experiment.

**Gross Pathological Findings:** At the end of the study, 10 broilers (randomly picked) from each of the experimentally infected groups were humanely sacrificed. On evisceration, presence of gross lesions especially haemorrhages, ballooned or thickened caeca and necrotic foci were looked for in the mucosa at various positions in the intestine (caeca, duodenum, jejunum, ileum and rectum) following a longitudinal incision of the intestine. Gross lesions were graded from 0 to 5 based on lesion score key (Dacie and Lewis, 1995). These include patchy haemorrhage of the intestinal mucosa graded as 1, ballooned caeca was scored 2 and caeca filled with tissue debris was scored 3.

**Histopathology:** The organs such as heart, liver, kidneys, brain, spleen, caeca and bursa of Fabricius from the 10 birds in each group were carefully collected at the end of the study, after the birds were feathered in hot water and dissected. These organs and pieces of the carcass were placed in plastic containers and fixed with 10 % neutral buffered formalin for 24 hours (Durry and Wallington, 1976).

The fixed organs and tissues were cut into 6 mm thickness. An automatic tissue processor with several partitions that contain graded concentrations of alcohol for a period of 24 hours was used in processing the tissues. The alcohol concentration percentages ranged between 70 to 100 %, while the tissues were soaked for two hours in each chamber. These tissues were infiltrated with paraffin wax at 60°C using an oven, allowed overnight to cool and mounted on blocks for cutting (6 micrometer thickness) using a rotary microtome. The tissues were later floated in a hot water bath maintained at 60°C, placed on albumenized microscopic slides, allowed to dry, then stained with Haematoxylin and Eosin stain (Durry and Wallington, 1976). Examinations of the stained slides in the light microscope were carried out using magnifications of x10, x20 and x40.

The following microscopic lesions observed in the tissues were scored from 0 to 5 based on the lesion score key (Dacie and Lewis, 1995) thus: few lymphocytes in the liver was scored 1, necrotic foci in the caecal mucosa was scored 2, slight lymphoid degeneration was scored 1, profound lymphoid degeneration of the bursa of fabrius was scored 3, fibrotic bursal follicle was scored 3, caecal tonsil highly congested was scored 4, congested bursal follicle in the liver was scored 4.

**Statistical Analysis:** The data generated were subjected to descriptive statistics for their central tendencies and Analysis of Variance.
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(ANOVA) and presented as means and percentages in Tables. The means and percentages were determined according to Olawuyi (1996). Variant means were separated using Duncan’s New Multiple Range Test and P<0.05 was accepted as significant.

RESULTS

Average Live Body Weight/Carcass Yield: The body weight generally increased in all the groups of broilers except in group D and mortality was not recorded in any of the groups. The mean live body weights (kg/broiler) for the infected groups (D, E and F) were 2.7 ± 0.04 kg, 3.2 ± 0.07 kg and 3.2 ± 0.07 kg respectively while those of the uninfected groups (A, B and C) were 3.3 ± 0.04 kg, 3.4 ± 0.29 kg and 3.1 ± 0.15 kg respectively (Table 1). Body weight gain was significantly higher (P<0.05) in the uninfected/*P. biglobosa* treated group B., The mean live body weight in infected/sulfaquinoxaline treated group E (3.2 kg/broiler) and infected/*P. biglobosa* treated group F (3.2 kg/broiler) were similar. Also the mean percentage organ and live body weight ratio was significantly higher (P<0.05) in the uninfected groups than the infected groups (Table 2).

Gross and Histopathological Findings: Studies of the gross and histopathological lesions were recorded in Table 3. These lesions were seen in various stages in the caecae, liver and bursa of fabricius. In all the groups there were few lymphocytes observed in the liver of the broilers. Gross lesion in Group F showed that the caecum was hemorrhagic in 4 broilers out of 10(40 %), 3(30 %) had thickened caecal mucosa and 2 other birds (20 %) had profound lymphoid degeneration of the Bursa of Fabricius. In Group E, 4 broilers out of 10(40 %) had haemorrhagic caecum and thickened caecal mucosa in 3(30 %) other broilers while in Group D, 5(50 %) haemorrhagic caecum were recorded, 3(30 %) with thickened caecal mucosa and 2 broilers (20 %) had ballooned caeca. In addition, 3 broilers (30 %) in Group D had fibrotic bursal follicles. Furthermore, in Group D, the caecal tonsils and mucosa of the caecum were highly congested in 3 out of 10(30 %) with varying degrees of lymphocytic infiltration in 6(60 %) broilers. The mean lesion counts for the infected groups (D, E and F) were 3.14 ± 0.74, 1.0 ± 0.65 and 1.3 ± 0.64 respectively while those of the uninfected groups (A, B and C) were 0.00 ± 0.00, 0.00 % ± 0.00 and 0.00 % ± 0.00 respectively. The mean percentage lesion counts was significantly higher (3.14 ± 0.74) in group D than other groups. No lesions were found in all the uninfected and untreated (Group C) and the uninfected and treated broilers (Groups A and B).

DISCUSSION

Significant differences in weights of organs were recorded in birds in Group E, where the liver, heart and the spleen weighed more than those in the other groups. The slight difference observed in the decrease in weight of the carcass parts (thigh, back and neck, wings) in Groups C and D may be due to the lack of treatment of the broilers with either *P. biglobosa* or sulfaquinoxaline. These findings were corroborated by Yvoré (1978) who observed that infection of chickens with *E. acervulina* or *E. tenella* results in depressed appetite, decreased feed efficiency and weight loss. On the contrary, the mechanisms of pathology are thought to differ between the two species, with the intestinal forms causing decreased nutrient absorption, whereas the bleeding associated with *E. tenella* causes morbidity through blood and serum protein loss (Fukata et al., 1997).

There were slight increases in the weight of the organs in Group E compared to the weight of organs in other groups. Similarly, Mustapha et al. (2015) in their study revealed that the increase in weight of internal organs such as the liver, kidney, gizzard, spleen and proventriculus may likely be because of increase in metabolic activities of these organs probably due to the presence of anti-nutritional factors or high fibre content in the diets. Carew et al. (2003) in their study reported that the consumption of unprocessed raw velvet beans (*Mucuna pruriens*) by broiler chickens reduced
### Table 1: Organ and carcass body weight (kg) of broiler chickens treated with *Parkia biglobosa* or Sulfaquinoxaline at 250 mg/ml after infection with *Eimeria tenella* sporulated oocysts

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
<th>Group D</th>
<th>Group E</th>
<th>Group F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Live body weight</td>
<td>3.3 ± 0.04ab</td>
<td>3.4 ± 0.29b</td>
<td>3.1 ± 0.15b</td>
<td>2.7 ± 0.04a</td>
<td>3.2 ± 0.07b</td>
<td>3.2 ± 0.07b</td>
</tr>
<tr>
<td>Carcass dressed weight</td>
<td>2.8 ± 0.07c</td>
<td>2.6 ± 0.08b</td>
<td>2.5 ± 0.08b</td>
<td>2.4 ± 0.04b</td>
<td>2.6 ± 0.07b</td>
<td>2.7 ± 0.08b</td>
</tr>
<tr>
<td>Breast + chest weight</td>
<td>23.2 ± 0.41b</td>
<td>22.2 ± 0.37c</td>
<td>23.1 ± 0.41b</td>
<td>23.2 ± 0.41b</td>
<td>21.2 ± 0.43b</td>
<td>22.3 ± 0.38b</td>
</tr>
<tr>
<td>Thigh + leg weight</td>
<td>23.2 ± 0.41b</td>
<td>22.7 ± 0.08c</td>
<td>20.8 ± 0.04b</td>
<td>22.5 ± 0.08c</td>
<td>21.7 ± 0.44b</td>
<td>22.4 ± 0.45c</td>
</tr>
<tr>
<td>Back + neck weight</td>
<td>23.2 ± 0.39c</td>
<td>22.1 ± 0.04c</td>
<td>20.8 ± 0.12a</td>
<td>21.1 ± 0.37c</td>
<td>21.8 ± 0.45b</td>
<td>21.8 ± 0.45b</td>
</tr>
<tr>
<td>Wing weight</td>
<td>9.6 ± 0.08b</td>
<td>10.0 ± 0.41b</td>
<td>9.5 ± 0.12b</td>
<td>9.6 ± 0.39b</td>
<td>11.3 ± 0.39b</td>
<td>11.1 ± 0.43b</td>
</tr>
<tr>
<td>Shank weight</td>
<td>6.0 ± 0.11b</td>
<td>5.7 ± 0.41b</td>
<td>5.2 ± 0.08b</td>
<td>6.1 ± 0.43b</td>
<td>6.3 ± 0.41b</td>
<td>6.4 ± 0.45b</td>
</tr>
</tbody>
</table>

Means within a row with different superscripts are significantly different (p<0.05), Group A = uninfected + Sulfaquinoxaline 250 mg/ml, Group B = uninfected + *P. biglobosa* 250 mg/ml, Group C = uninfected + untreated, Group D = infected + untreated, Group E = infected + Sulfaquinoxaline 250 mg/ml and Group F = infected + *P. biglobosa* 250 mg/ml

### Table 2: Organs weight (g) of broiler chickens treated with *Parkia biglobosa* or Sulfaquinoxaline at 250 mg/ml after infection with *Eimeria tenella* sporulated oocysts

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
<th>Group D</th>
<th>Group E</th>
<th>Group F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gizzard</td>
<td>2.4 ± 0.04c</td>
<td>2.2 ± 0.29c</td>
<td>2.0 ± 0.70a</td>
<td>2.0 ± 0.08a</td>
<td>2.1 ± 0.04b</td>
<td>2.0 ± 0.08a</td>
</tr>
<tr>
<td>Liver</td>
<td>1.6 ± 0.08a</td>
<td>1.8 ± 0.08b</td>
<td>1.7 ± 0.08b</td>
<td>1.7 ± 0.07b</td>
<td>2.0 ± 0.08c</td>
<td>1.7 ± 0.08b</td>
</tr>
<tr>
<td>Heart</td>
<td>0.6 ± 0.07a</td>
<td>0.5 ± 0.07a</td>
<td>0.6 ± 0.08b</td>
<td>0.6 ± 0.08b</td>
<td>0.8 ± 0.07b</td>
<td>0.6 ± 0.08b</td>
</tr>
<tr>
<td>Spleen</td>
<td>0.2 ± 0.04a</td>
<td>0.1 ± 0.02a</td>
<td>0.2 ± 0.04b</td>
<td>0.2 ± 0.04b</td>
<td>0.3 ± 0.04b</td>
<td>0.2 ± 0.04b</td>
</tr>
<tr>
<td>Intestine</td>
<td>9.2 ± 0.04b</td>
<td>10.7 ± 0.07c</td>
<td>11.3 ± 0.10c</td>
<td>12.1 ± 0.37c</td>
<td>10.2 ± 0.07c</td>
<td>8.3 ± 0.36a</td>
</tr>
</tbody>
</table>

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### Table 3: Postmortem and histopathologic findings in organs and tissues of broiler chickens treated with *Parkia biglobosa* or Sulfaquinoxaline after infection with *Eimeria tenella* oocysts

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group A</th>
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<th>Group D</th>
<th>Group E</th>
<th>Group F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Postmortem Findings</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Haemorrhagic caecum</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>5(50)</td>
<td>4(40)</td>
<td>4(40)</td>
</tr>
<tr>
<td>Thinned caecal mucosa</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>3(30)</td>
<td>3(30)</td>
<td>3(30)</td>
</tr>
<tr>
<td>Ballooned caeca</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>2(20)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
</tr>
<tr>
<td><strong>Histopathology Findings</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lymphoid degeneration of bursa of fabricius</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>2(20)</td>
</tr>
<tr>
<td>Bursal follicle fibrotic</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>3(30)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
</tr>
<tr>
<td>Congested caecal tonsil mucosa</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>3(30)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
</tr>
<tr>
<td>Lymphocytic infiltration</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>6(60)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
</tr>
<tr>
<td><strong>Mean</strong></td>
<td>0.00 ± 0.00a</td>
<td>0.00 ± 0.00a</td>
<td>0.00 ± 0.00a</td>
<td>3.14 ± 0.74c</td>
<td>1.0 ± 0.65b</td>
<td>1.3 ± 0.64b</td>
</tr>
</tbody>
</table>

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body weight gain, but weights of the pancreas, gizzard, proventriculus and the heart as well as length of the small and large intestine and caeca increased. Ogbe *et al.*, (2008) revealed that these variations in weight of organs may not be unconnected to a compensatory mechanism in relation to medication and the coccidian infection. It is a known fact that the weight of organs in broilers is indicative of the response of birds to the feed intake in relation to their age or growth. The weight of organs in broilers also reflects the anatomical response of broilers to the type of diets consumed such as the use of whole grains in feed or large high fibre particles (Atteh, 2004).

In group D the histopathological changes observed may be as a result of the effect of infection with *Eimeria* species since there was decrease in severity of lesions in the infected and treated broilers in Groups E and F. This finding is supported by McDougald and Reid (1997) in their study where they observed that *Eimeria tenella* is pathogenic chicken coccidia that parasitize caecum of young chickens. It is characterized by acute onset manifested by clinical signs of bloody diarrhea due to invasion of the mucosal lining of the caeca by the second generation merozoites.

There are associated microscopic changes due to coccidiosis which include congestion of the mucosa of the intestine, diffuse or patchy haemorrhagic enteritis, lymphocytic and mononuclear cells infiltration of the intestinal mucosa and sloughing of epithelial lining. It is possible that tissue damage and changes in the functional integrity of the intestine common with coccidian infection and other enteritis may allow colonization of the gut by various types of harmful bacteria, leading to inflammation of the intestine, followed by infiltration of leukocytes.

**Conclusion:** From this study, the improvement in clinical signs such as blood stained droppings which is consistent with *Eimeria tenella* infections in broiler chickens showed the anticoxidial effect of *P. biglobosa* extract. There were also increased weights of organs and the carcass, which indicated that the root bark of *P. biglobosa* had no harmful or adverse effect on the organs of treated broilers with or without *E. tenella* infection. It is suggested that in-depth research on the use of the root bark of *P. biglobosa* for the control of coccidiosis in broiler chickens be carried out.

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