



Research Article

# Carcass, Organ Weights and Histo-morphology of Internal Organs of Sows Fed Fermented and Enzyme-Supplemented Cassava Peels Meal (CPM) Based Diets

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Fresh cassava peels were subjected to submerged fermentation, sundried for 3-5 days and also subjected to proximate analysis. Fermentation reduced cyanide and improved crude protein. A group of 27 weaner gilts (Largewhite x Duroc), aged 8-9 weeks and weighed  $10.61 \pm 0.27$ kg were fed fermented cassava-peels-based-diets. They were allotted to three treatments comprising T<sub>1</sub> (control), T<sub>2</sub> (fermented CPM) and T<sub>3</sub> (fermented CPM + enzyme) in a completely randomized design and fed for 22 weeks. Data on carcass and some visceral organs weights were subjected to analysis of variance and means separated using Duncan's Multiple Range Test. Histo-morphology on the organs was conducted. The dressing percentages were 66.53, 60.25 and 64.11% for T<sub>1</sub>, T<sub>2</sub> and T<sub>3</sub> respectively whereas the head, heart, lungs and kidney were the weightiest for T<sub>1</sub>, the stomach/intestine for T<sub>2</sub> and the liver and spleen for T<sub>3</sub> while the histo-morphology of T<sub>1</sub> sows were all normal except for mild architectural deviation in the duodenum and ileum. Histo-morphological changes were observed in the ileum and duodenum of T<sub>2</sub> and T<sub>3</sub>. It is therefore recommended that fermented peels be supplemented with enzyme for improvement in dressing percentage and watch-out for pathological lesions in the visceral organs.

**Keywords:** Sows, carcass, visceral organs, histo-morphology, fermented cassava peels, maxigrain<sup>R</sup> enzyme

## INTRODUCTION

Cassava (*Manihot esculenta* Crantz) is a staple food in tropical Africa and central and South-America. Nigeria with an annual production of 34-40 million tonnes is the world largest producer of the crop (CBN, 2003). Chief among the wastes obtained from cassava processing is the cassava peels which account for 5-20% of the root and it is estimated that about 4 million tonnes of cassava peels are generated from cassava processing in Nigeria annually (Hahn and Keyster, 1985; Nwokoro et al., 2005; Aro et al., 2010). Omole and Sonaiya (1981) reported that CPM can substitute up to 40 per cent of the diets of rabbits. Obioha et al. (1984) observed that laying hens performance was not retarded when the diets consisted of 40 percent CPM. Also, Obioha et al. (1985) working on growing-finishing pigs reported that there was a progressive decline in average daily gain, feed efficiency and protein efficiency ratio from the zero CPM diet to the zero maize diet, but

these comparisons were not significant. Liver weight and spleen weight (expressed as percentage of body weight) were slightly higher in the CPM diets than the control. Ikurior and Onuh (1996) observed that daily gains of growing pigs fed cassava peel declined significantly ( $P < 0.05$ ) as level of inclusion increased.

The greatest limitation to the use of cassava for livestock feeds is its content of cynogenic glucosides, linamarin and lotaustralin.

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Toxicity of cassava is caused by hydrocyanic acid (HCN) which is liberated when the glucoside is hydrolyzed by the action of linamarase enzyme. The degree of toxicity depends upon the variety, ecological conditions for growth of the plant, the form of the product being fed and its processing technology (Coursey, 1973). The normal range of HCN in fresh cassava root is 15-400ppm (Rogers, 1963). It has long been established that the Peel contained 5-10 times the Prussic acid content of the pulp (Oyenuga and Amazigo, 1957). Maner (1974) observed that pigs can tolerate 150- 200ppm HCN on a fresh basis or 102ppm on a dry matter basis. Utilization of CPM in animal feed is dependent on effective detoxification mechanisms for the cyanide.

Cyanides are components of electroplating solutions, fertilizers, fumigant mixtures, metal polishes and rodenticides. It is an environmental factor has been associated with many intoxication episodes in humans and animals resulting from the ingestion of foods, environmental pollution, chemical war, suicide, homicide, occupational factors and use in some drugs such as nitroprusside and laetrile (Watts, 1998). In plants, cyanide can be found mainly as cyanogenic glycosides, as found in *Manihot* spp (cassava), *Linum* spp, *Lotus* spp, *Phaseolus lunatus*, *Sorghum* spp (Conn, 1978) and the content of this substance can be as high as 100-800 mg/kg of the plant material (Poulton, 1983). Acute cyanide toxicity is lethal, resulting in death due to respiratory failure (Greer and Jo, 1995), while chronic cyanide exposure has been implicated in the etiology of goitre (Cliff et al., 1986), tropical ataxic neuropathy (Osuntokun, 1981). Regardless the route of exposure, cyanide is rapidly absorbed into the blood stream and distributed throughout the body. Cyanide concentrates in erythrocytes through binding to methemoglobin (Towill et al., 1978) and cause hypothyroidism that leads to goiter (Kamalu and Agharanya, 1991; Elsaid and Elkomy, 2006). Subacute oral administration of cyanide in rats produced changes in several biochemical indices and pathology in various organs (Tulswani et al., 2005).

In this study, fermentation was used to reduce the cyanide to a safe level and increased the crude protein content to help assuage the effect of protein deficiency. What is the effect on the carcass, visceral organs and pathomorphological characteristics of sows fed diets compounded with fermented cassava peels? This forms the basic research question which this study intends to answer. This assessment is expected to x-ray the suitability of use of fermented CPM as an ingredient in pig's diet with respect to weights of carcass and visceral organs as well as its impact on the micro-structures of visceral organs.

## MATERIALS AND METHODS

### Experimental Site

The experiment was carried out at the piggery unit (Bora Farm) of The Research Farm of Federal College of Animal

Health and Production Technology, Moor Plantation, Ibadan, Oyo State, Nigeria. Ibadan is geographically located at latitude 7° 22' 39" N and longitude 3° 54' 21" E. Ibadan has a tropical wet and dry climate, with a lengthy wet season and relatively constant temperature throughout the course of the year. It has mean total rainfall of 1420.06 mm, mean maximum temperature of 26.46° C with 21.42° C as the minimum and relative humidity of 74.55%.

### Fermentation of Cassava Peels

The washed fresh cassava peels which had not stayed for longer than 4-6 hours since peeled off were immersed in clean bore-hole water in a plastic container (vat) and left at an ambient temperature of 26-30 °C for four days. Sign of fermentation which include foaming was looked out for. After four days, the cassava peels were separated from the broth and spread on a clean polythene sheet under the full glare of the sunlight for 3-5 days during which it dried to constant weight (Okpako et al., 2008; Naa et al., 2010).

### Experimental Animals, Management, Design and Duration

A group of 27 female weaner pigs (Largewhite x Duroc), aged 8-9 weeks and weighed 10.61±0.27kg each, with good body conformation were randomly selected from many and kept under one big compartment for stabilization for 2 weeks during which they were placed on conventional diet (16.5% crude protein and 2800kcal/kg metabolizable energy) *ad-libitum* with ample supply of clean bore-hole water. During the same period, close observation for deformity and other aberrations that could render them unfit for the experiment were looked out for and replacements made. The pigs were also prophylactically treated against endo- and ectoparasites using ivermectin<sup>R</sup> (ivermectin) injection at the dose of 1ml/33kg body weight, subcutaneously. There was also administration of long acting oxytetracycline injection at the dose rate of 1ml/10kg body weight (im) which was repeated after 48 hours to help eliminate possible pathogenic microbes that had not manifested as disease(s). Thereafter, they were randomly allotted to their respective pens according to treatments comprising T<sub>1</sub> (control), T<sub>2</sub> (fermented CPM) and T<sub>3</sub> (fermented CPM + Maxigrain<sup>R</sup> enzyme) in a completely randomized design (Obi, 2002). The above treatments were replicated thrice with each replicate containing three weaned pigs. At this point, the grower treatment diets were introduced at 4% of their body weight daily (Santiago and Tegbe, 1987; Onyimonyi, 2002). The grower diets were given for the first eleven weeks after which they were replaced with the finisher diets for another eleven weeks. Similarly, clean drinking water sourced from the borehole in the farm was supplied *ad-libitum* to the pigs.

### Feed Formulation

The diets were compounded thus:-

T<sub>1</sub> = Conventional maize-based diet (control).

**Table 1:- Dietary composition of pig's grower diets**

Ingredient	T <sub>1</sub>	T <sub>2</sub>	T <sub>3</sub>
Maize	40.00	-	-
CPM	-	40.00	40.00
PKC	20.00	29.50	29.50
BDG	14.00	10.00	10.00
GNC	12.50	11.00	11.00
BLM	5.00	5.00	5.00
Palm oil	4.00	4.00	4.00
Bone meal	2.00	2.00	2.00
Oyster shell	1.00	1.00	1.00
Methionine	0.20	0.20	0.20
Lysine	0.75	0.75	0.75
Premix	0.40	0.40	0.40
Salt	0.15	0.15	0.15
Maxigrain <sup>R</sup>	-	-	+
Total	100	100	100
C.P. (%)	20.82	20.47	20.47
DE (kcal/kg)	3368.61	3118.39	3118.39

**Table 2:- Dietary composition of pig's finisher diets**

Ingredient	T <sub>1</sub>	T <sub>2</sub>	T <sub>3</sub>
Maize	40.00	-	-
CPM	-	40.00	40.00
PKC	22.50	23.50	23.50
BDG	10.00	10.00	10.00
W/O	14	9.00	9.00
SBM	-	4.00	4.00
BLM	5.00	5.00	5.00
Palm oil	4.00	4.00	4.00
Bone meal	2.00	2.00	2.00
Oyster shell	1.00	1.00	1.00
Methionine	0.20	0.20	0.20
Lysine	0.75	0.75	0.75
Premix	0.40	0.40	0.40
Salt	0.15	0.15	0.15
Maxigrain <sup>R</sup>	-	-	+
Total	100	100	100
C.P. (%)	17.02	17.36	17.36
DE (kcal/kg)	3239.20	3068.58	3068.58

Provided the following/kg diet: Vitamin A-8,000 IU, Vitamins D3 -3,000 IU, Vitamins E-8 IU, Vitamin K -2mg, Vitamin B1- 1 mg, Vitamin B2-0.2 mg, Vitamin B12-5 mg, Nicotinamide -10 mg, Selenium- 0.1 mg, Ca Pantothenate - 5 mg, Folic acid -0.5 mg, Choline Chloride -150 mg, Iron -20 mg, Manganese -80 mg, Copper -8 mg, Zinc -50 mg, Cobalt -0.225mg, Iodine -2 mg Antioxidant - 0.1ppm plus Maxigrain<sup>R</sup> enzyme.

Key:- CPM = Cassava peels meal, PKC = Palm kernel cake, W/O = Wheat ofal, BDG = Brewer's dried grain, SBM = Soybean meal, BLM = Blood meal, C.P. = Crude protein, ME = Metabolizable energy.

The above diets were analyzed for proximate chemical composition using procedures of Association of Official Analytic Chemistry (AOAC, 1990).

T<sub>2</sub>= Diet with 40% maize-replaced fermented cassava peels.

T<sub>3</sub> =Diet with 40% maize-replaced fermented cassava peels supplemented with maxigrain<sup>R</sup> enzyme.

Concerted efforts were made to ensure that the feeds as shown in Tables 1 and 2 were isocaloric and isonitrogenic by closing up the gaps amongst treatment groups as regards energies and crude proteins in the feeds. The constituents of maxigrain<sup>R</sup> enzyme is as stated in Table 3.

### Data Collection

Three pigs each from every treatment were weighed, slaughtered by stunning with captive bolt at the frontal position, bled by severing the jugular vein while still

unconscious and eviscerated through careful dissection with scalpel to separate various parts and organs for carcass and organ measurements. The measurement and assessment were carried out on the carcass, liver, spleen, kidney, lungs and the large/small intestine. With the aid of a digital sensitive scale, the carcass and visceral organs were measured. They were grossly checked for pathological defects. After which histopathological examinations were done using the method of Humason (1962).

### Statistical Analysis

All data were subjected to one way analysis of variance (ANOVA) and where statistical difference in means was

**Table 3:- Constituent of maxigrain<sup>R</sup> enzyme**

Enzyme	Quantity (IU)
Cellulase	10,000
Beta-glucanase	200
Xylanase	10,000
Phytase	2500

Maxigrain<sup>R</sup> enzyme is used at the rate of 1g/10kg of feed.

**Table 4: Carcass traits of sows fed fermented CPM-based diets**

Variables	T <sub>1</sub> (control)	T <sub>2</sub> (CPM only)	T <sub>3</sub> (CPM + Enzyme)
Live wt (kg)	67.82±0.14 <sup>a</sup>	58.19±0.19 <sup>b</sup>	65.71±0.16 <sup>a</sup>
Hot carcass wt (kg)	45.12±0.09 <sup>a</sup>	35.06±0.06 <sup>b</sup>	42.13±0.08 <sup>a</sup>
Dressing % (%)	66.53±0.14 <sup>a</sup>	60.25±0.11 <sup>b</sup>	64.11±0.18 <sup>a</sup>
Head (kg)	2.72±0.06 (6.03%)	2.23±0.03 (6.36%)	2.68±0.05 (6.36%)
Heart (g)	216.32±0.15 (0.48%) <sup>a</sup>	203.20±0.23 (0.58%) <sup>b</sup>	191.73±0.20 (0.46%) <sup>c</sup>
Liver (g)	1128.62±0.31 (2.5%) <sup>b</sup>	977.50±0.26 (2.79%) <sup>c</sup>	1261.02±0.13 (2.99%) <sup>a</sup>
Spleen (g)	93.14±0.07 (0.21%)	101.28±0.16 (0.29%)	114.39±0.18 (0.27%)
Lungs (g)	483.67±0.22 (1.07%)	444.61±0.14 (1.27%)	428.82±0.28 (1.02%)
Kidney (g)	81.64±0.14 (0.18%) <sup>a</sup>	62.83±0.23 (0.18%) <sup>b</sup>	68.69±0.21(0.16%) <sup>b</sup>
Stomach/intest (kg)	4.16±0.37 (9.22%) <sup>b</sup>	4.69±0.41(13.38%) <sup>a</sup>	3.92±0.25 (9.30%) <sup>b</sup>

abc:- means on the same row with different superscripts are statistically different (P<0.05)

found, it was separated using Duncan's New Multiple Range Test (Steel and Torrie, 1980) whereas the histo-morphology was based on expertise comparison of normal histology with the prepared slides as stated above.

## RESULTS AND DISCUSSIONS

Table 4 shows the carcass traits of sows fed fermented and enzyme-supplemented CPM-based diets. The live weights, hot carcass weight, dressing percentage, weights of head and kidney recorded the highest by T<sub>1</sub> and closely followed by T<sub>3</sub> and T<sub>2</sub>. All the parameters showed numerical superiority of the control diet (T<sub>1</sub>) followed closely by the enzyme supplemented diet (T<sub>3</sub>) and lastly the CPM-only based diet (T<sub>2</sub>). This trend was however, with the exclusion of lungs and stomach/intestine where T<sub>2</sub> was superior to T<sub>3</sub> but not T<sub>1</sub>.

The histo-morphology of the visceral organs/tissues that showed some pathological features is represented in plates 1 to 6. All the organs/tissues in T<sub>1</sub> (control) showed normal histo-architecture. The lungs (Plate 2) from T<sub>2</sub> (CPM only) sows showed a severe congestion and haemorrhage of the pulmonary parenchyma. The alveolar interstitia were also severely congested and some of the alveoli contained clusters of red blood cells. Similarly, the duodenum (Plate 5) of T<sub>2</sub> showed severe degeneration and necrosis of the villi with fewer mucosal glands. As shown by plate 6, duodenum from T<sub>3</sub> (CPM + Maxigrain<sup>R</sup> enzyme) sows had loss of enterocytes in the villi (i.e the villi are still evident but the columnar epithelia which should normally line the villi are gone) as well as a decrease in the number of mucosal glands.

### Carcass Traits

The carcass traits are in favor of T<sub>1</sub> (control) possibly because of higher energy and protein in the diet but generally, it was not significantly different (P>0.05) from T<sub>3</sub>

probably because of the effect of enzyme on the feed which could have aided availability of locked up nutrients as well as helped in digesting the cellulose and other lignins in the fibrous cassava peels. Garcia-Valverde *et al.* (2008) reported that pigs on high level of nutrition deposited both lean and fat at a faster rate than those fed moderate level of nutrition on both age and weight constant bases. Pigs on higher nutritional plane obtain adequate intake of nutrients required to sustain rapid growth and development (Njoku *et al.*, 2013). The higher live weight, hot carcass weight and better dressing percentage of T<sub>3</sub> (CPM + enzyme) over T<sub>2</sub> (CPM only) was probably because of the superiority conferred by the maxigrain enzyme on the T<sub>3</sub> diet. These parameters are similar to those of T<sub>1</sub> (control). Generally, the dressing percentages of the pigs in T<sub>1</sub> (66.53±0.14%), T<sub>2</sub> (60.25±0.11%) and T<sub>3</sub> (64.11±0.18%) were similar to 67-68.2% obtained in Luthuanian indigenous pigs (Razmaite *et al.*, 2009) but differed from 71.68-80.65% of commercial pig crossbreeds in China (Jiang *et al.*, 2012) as well as 72.09%, 74.43% and 81.86% got from growing pigs fed graded levels of feed (1.5kg, 2.0kg and 2.5kg daily) respectively (Njoku *et al.*, 2015) and 77-85% reported by Onyimonyi and Okeke (2005) on pigs placed on graded cassava peel meal. Adequate quantity of energy intake is critical to optimize lean growth rate and efficiency (Augenstein *et al.*, 1997). The feeding level, pattern and protein-energy ratio of the diet, together with the genetic growth potential of pigs determine the growth rate and composition of weight gain at both whole-body and muscle level (Lebret, 2008a; Merck manual, 2008). The amount of feed offered per day played vital role in the growth performance which therefore had direct bearing on the quality of carcass produced. Limited-feeding leads to depletion of apparent rate of glycogen as measured by muscle acidity (McPhee and Trout, 1995), resulting to reduction in back fat depth and rate of lean growth (McPhee *et al.*, 1988). This study contradicts Nguyen and

Cam (2001) who raised pigs on restricted feeding and reported high growth rate, low back fat and high lean percentage in the carcass of their descendants.

### Organ Weights

The head weight of T<sub>1</sub>(2.72kg) and T<sub>3</sub>(2.68kg) were very similar unlike in T<sub>2</sub>(2.23kg), corroborating Njoku *et al.* (2015) who reported that pigs with larger body weights had higher head, ham, shoulder and feet weights. Lo-Fiego *et al.* (2005) and Latorre *et al.* (2008) also observed that the weights of ham, shoulder and loin increased with increase in weight at slaughter. However, the observation contradicted the findings of Virgili *et al.* (2003) who suggested that primal cut proportion decreases with increasing body weight because the growth rate of primal cuts was lower with age than the growth rate of the whole body. Similarly, the weights of other visceral organs were in favour of T<sub>1</sub> and T<sub>3</sub>. Barca *et al.* (2006) reported that feed intake stimulates visceral organ growth and it also alters the distribution of whole-body protein. Some factors known to influence visceral organ size are body weight, feeding level, diet composition and pig genotype (Nyachoti, 1998). The relationship between visceral organ mass and body weight appears to reflect both changes in feed intake and maintenance energy requirements with increasing body weight (Van-Milgen and Noblet, 2003). Lebret (2008b) reported that feeding level and pattern of feeding are tools used to manipulate growth rate, composition of weight gain and intramuscular fat deposition. The superiority of T<sub>1</sub> in this research was in contradiction with the finding of Onyimonyi and Okeke (2005) that carcass and organ weights increased with increasing cassava peel meal in the diets of pigs. It is also possible that the HCN content of the CPM could have affected the organs and carcass negatively. It has been established that cyanide and thiocyanate are selective poisons. In sublethal concentrations, they cause cell death by interfering with oxidative production of energy from glucose, fatty acids and amino acids (Kamalu, 1993). Cyanide inhibits the enzyme; cytochrome oxidase, thereby preventing the use of oxygen, while thiocyanate inhibits the enzyme fumarate hydratase in the Krebb citric acid cycle (Massey and Alberty, 1954). Intact linamarin in sublethal concentrations inhibits the activity of Na<sup>+</sup>-K<sup>+</sup> ATPase (Hill, 1977) and decreases the intracellular K<sup>+</sup> concentration (Philbrick *et al.*, 1977) in cardiac tissues. The combined effects of all these were the retarded growth observed with CPM inclusion in the diet and hence the resultant decline in carcass weight. Meanwhile, the observed highest numerical weight of stomach/intestine in T<sub>2</sub> could be due to pressure on the GIT to digest the relatively high fibrous feed. Regrettably, recognition of cyanide poisoning may be delayed because the majority of clinical and laboratory findings are non-specific (Graham *et al.*, 1977; Gonzales and Sabatini, 1989; Kulig and Ballantyne, 1993). Cyanide exerts its toxic effects by binding to the ferric ion in the a-a<sup>3</sup> complex of cytochrome oxidase resulting in the

inhibition of aerobic metabolism (Vogel *et al.*, 1981; Holland and Kozlowski, 1986; Baskin *et al.*, 1992; Beasley and Glass, 1998). This may account for cellular damage seen in most of the tissues as metabolism shifts from aerobic to anaerobic, with consequent production of lactic acid (Nnoli *et al.*, 2013). Acute cyanide toxicity is lethal, resulting in death due to respiratory failure (Greer and Jo, 1995).

### Histo-morphology

Subacute oral administration of cyanide in rats produced changes in several biochemical indices and pathology in various organs (Tulswani *et al.*, 2005). The sows in T<sub>1</sub> had no deleterious effects associated with HCN on the gastrointestinal tract (GIT) and visceral organs although the duodenum had numerous tightly packed submucosal glands while the ileum had increased crypt of Lieberkuhn and goblet cells. This trend suggested increased secretion of mucus by the goblet cells and other underlying cells of the crypts. This could aid digestion and improved innate immunity by the secretions of the Paneth cells. The lungs of sows fed T<sub>2</sub> diet showed severe congestion, haemorrhage of pulmonary parenchyma and severely congested interstitia. This is in corroboration with Pathak *et al.* (2015) who reported pulmonary edema, pulmonary hemorrhages and marked pulmonary vessels' congestion in human patient that was poisoned with cyanide. The duodenum of sows on T<sub>2</sub> and T<sub>3</sub> diets revealed severe degeneration and necrosis of the villi which was suggestive of the effect of HCN since the two diets were compounded with CPM. Such histopathological features were not seen in the sows fed with maize-based feed (T<sub>1</sub>). This attests to the submission of Tulswani *et al.* (2005) who opined that subacute oral administration of cyanide in rats produced changes in several biochemical indices and pathology in various organs. However, the visceral organs in all the treatment groups showed little or no histopathological changes. This could be attributed to the absence of HCN in T<sub>1</sub> and tolerable quantities of HCN in T<sub>2</sub> and T<sub>3</sub> due to fermentation and enzyme supplementation. This result was partly in consonance with the report of Nnoli *et al.* (2013) that other organs and small intestine showed no pathological changes in acute cyanide poisoning of a man but also supports the report of Pathak *et al.* (2015) who reported congested liver, kidney and spleen as well as cloudy changes in the renal tubules. Meanwhile, they reported normal myocardial fibers in the heart.

### CONCLUSIONS

Generally, pigs that were fed with conventional feed (T<sub>1</sub>) and mixture of fermented cassava peels and cocktail enzyme (T<sub>3</sub>) diets performed similarly and better than

fermented cassava peels (T<sub>2</sub>) with respect to carcass and visceral organs weight. Therefore, if fermented cassava peels have to be used as ingredient in pig's diet, supplementation with cocktail enzyme is advised for better carcass performance and reduction in pathological consequences on the visceral organs.

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Plate 1: Section of the lungs from T<sub>1</sub> showed the normal histo-architecture of the mammalian lung. It showed normal bronchi (Br) and bronchiole (B), normal alveoli (A) and alveolar interstitium (arrow). Cartilage (C). H&E x40

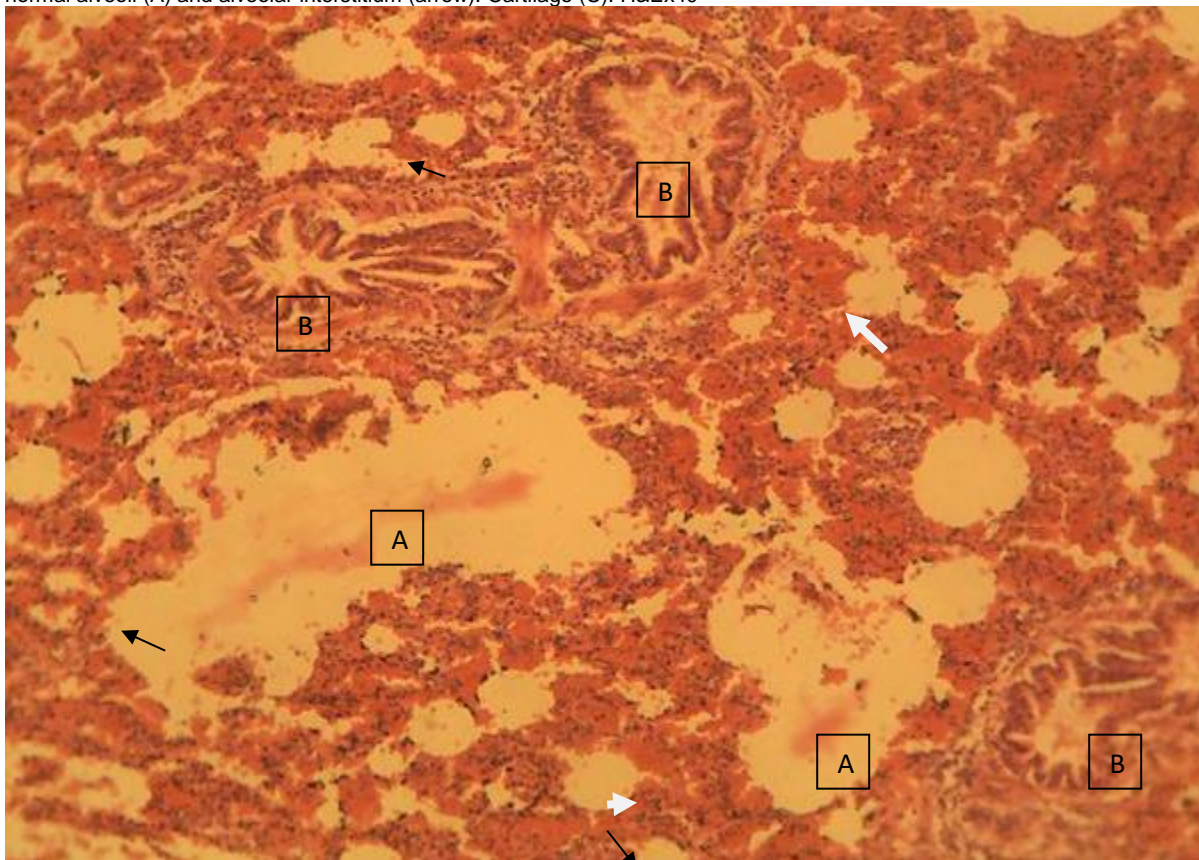


Plate 2: Section of the lungs from T<sub>2</sub> (CPM only) group showing a severe congestion and haemorrhaging of the pulmonary parenchyma. The alveolar interstitia were severely congested (black arrow) and some of the alveoli contain clusters of red blood cells (white arrow). Bronchiole (B), Alveoli (A). H&E (x40).



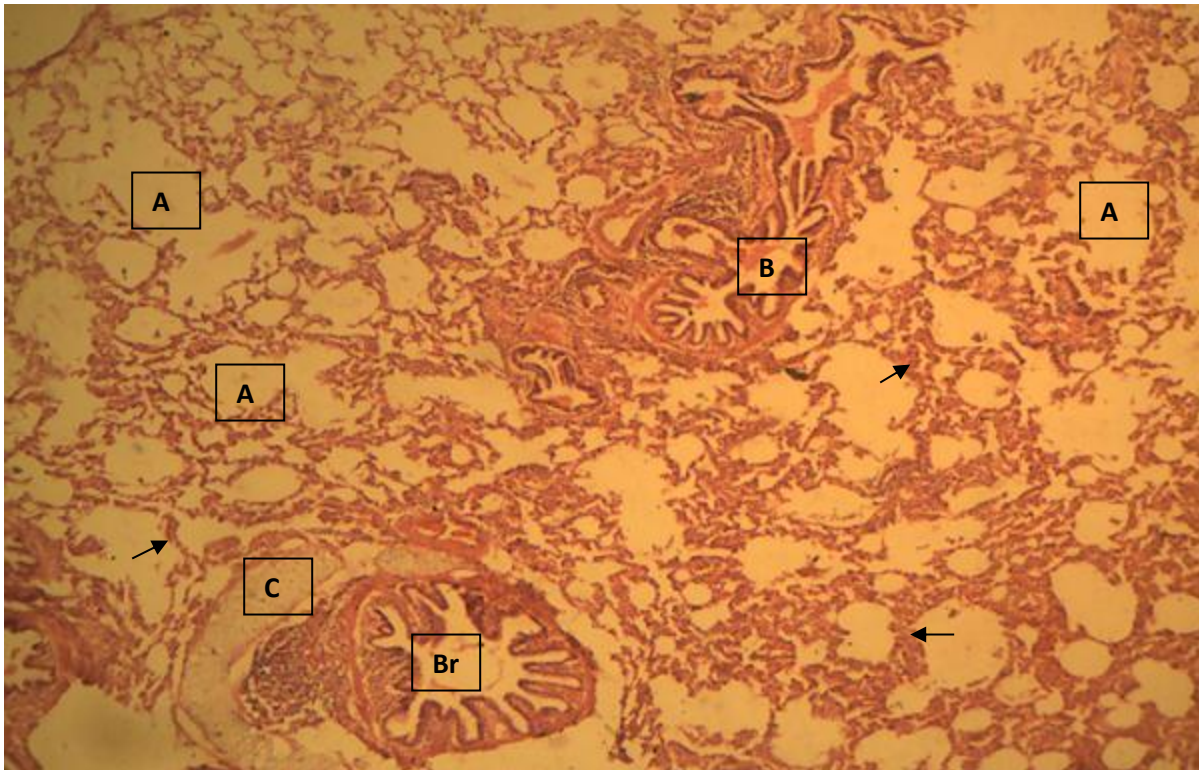


Plate 3: Sections of the lungs from T<sub>3</sub> showed the normal histo-architecture of the mammalian lung. It showed normal bronchi (Br) and bronchiole (B), normal alveoli (A) and alveolar interstitium (arrow). Cartilage (C). H&Ex40

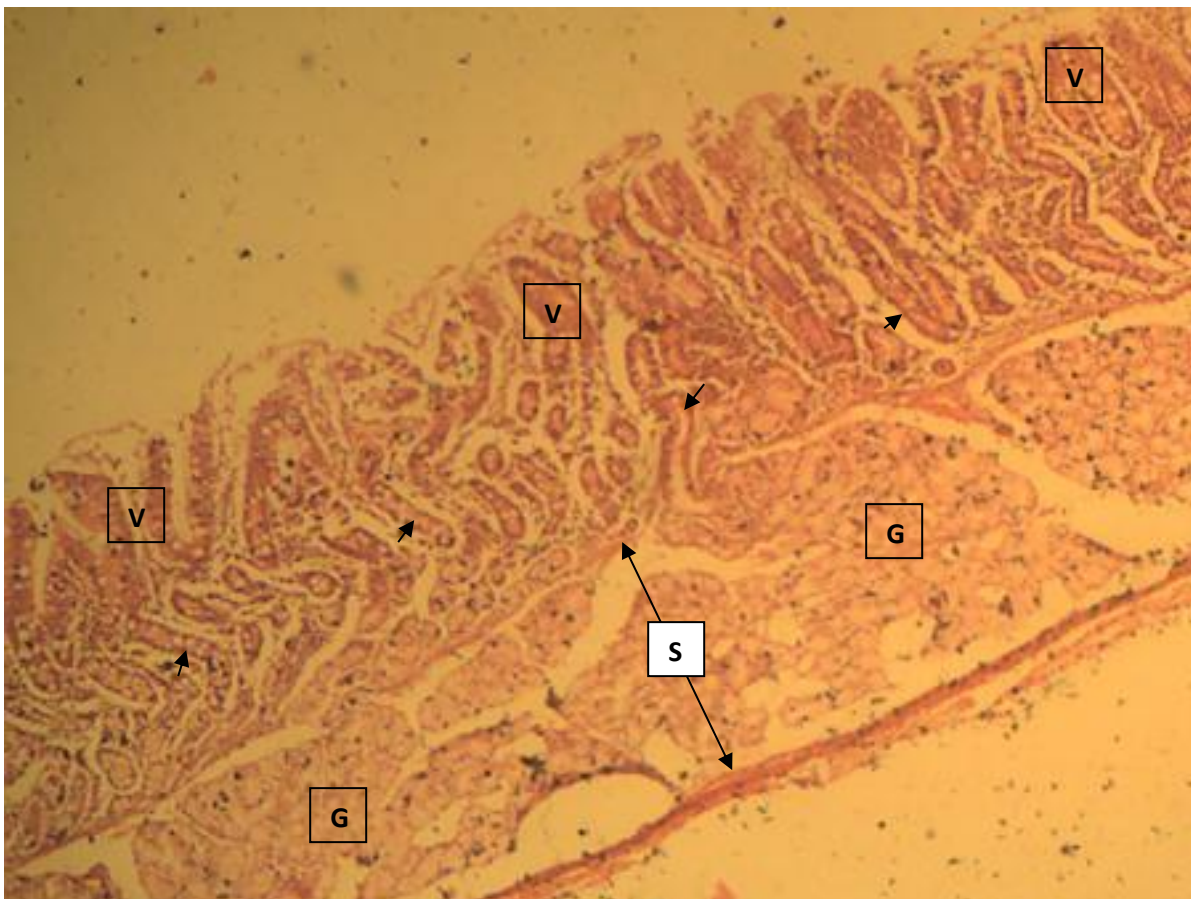


Plate 4: Sections of the duodenal mucosa from T<sub>1</sub> group showed normal villi (V) and crypts of Lieberkuhn (arrow). The submucosa (S) showed numerous tightly packed submucosal glands (G).

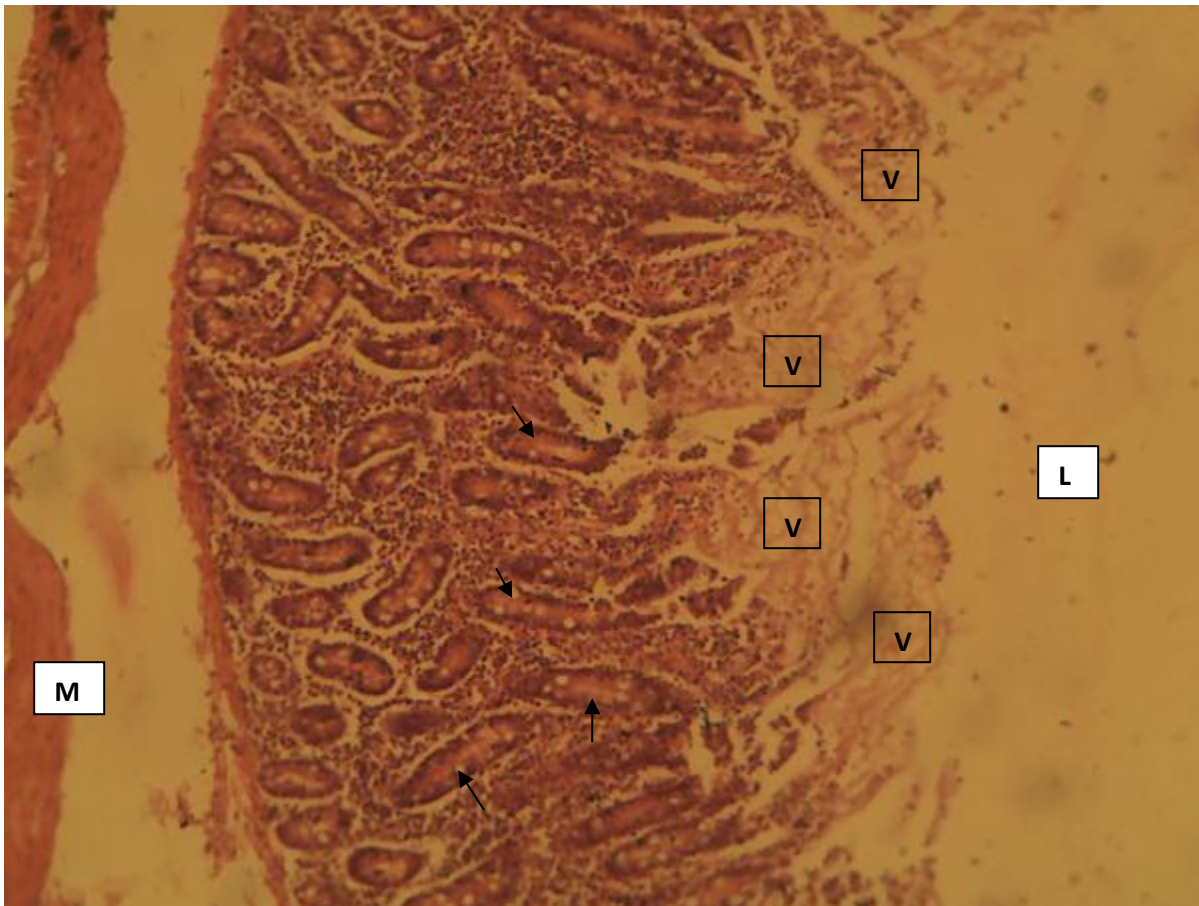


Plate 5: Section of the duodenum from the animals in  $T_2$  (CPM only) group showing severe degeneration and necrosis of the villi (V). Fewer mucosal glands (arrow) were also observed. Muscularis mucosa (M), Intestinal lumen (L). H&E (x40).

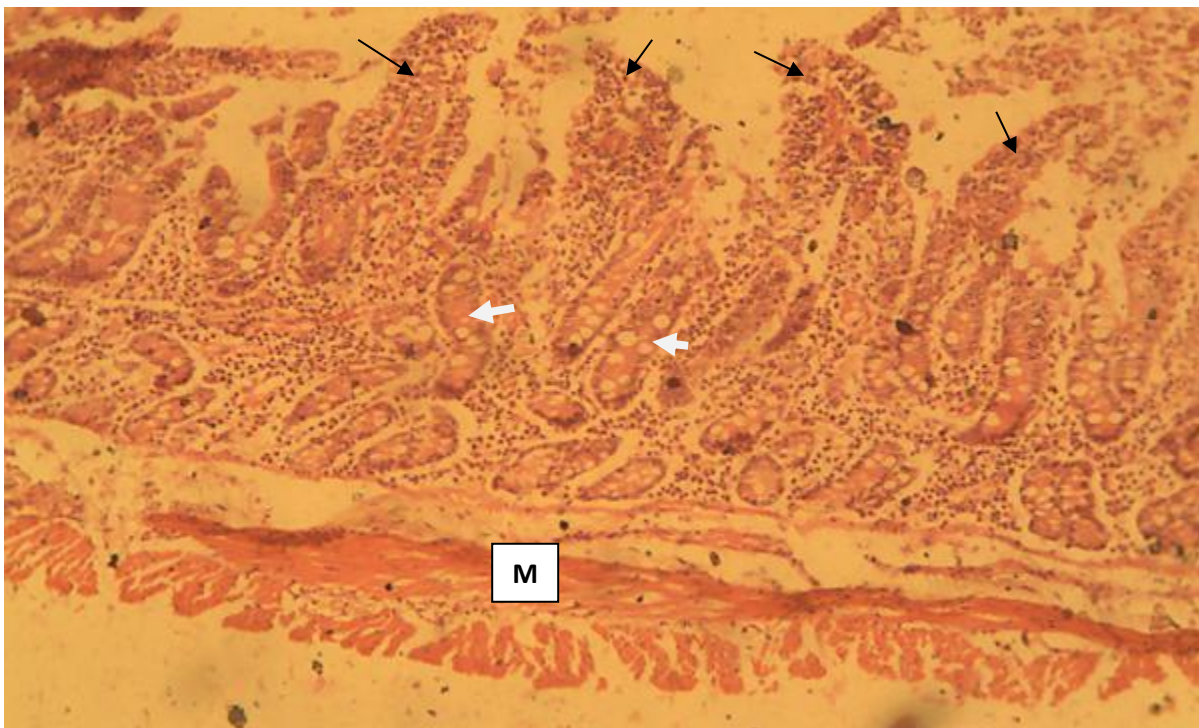


Plate 6: Section of the duodenum from  $T_3$  (CPM + Maxigrain<sup>®</sup> enzyme) sows showing loss of enterocytes in the villi (black arrow) (i.e the villi are still evident but the columnar epithelia which should normally line the villi are gone) as well as a decrease in the number of mucosal glands (white arrow). Muscularis mucosa (M). H&E (x40).

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